Toxicological profile for

Petroleum resin

This ingredient has been assessed to determine potential human health effects for the consumer. It was considered not to increase the inherent toxicity of the product and thus is acceptable under conditions of intended use.

1. Name of substance and physico-chemical properties

1.1. IUPAC systematic name

Not applicable.

1.2. Synonyms

Caswell No. 647; EPA Pesticide Chemical Code 011401; EINECS 265-116-8; Arien; Petroleum resins (ChemIDplus)

1.3. Molecular formula

Unspecified. A complex combination of organic compounds, predominantly hydrocarbons, obtained as a fraction of the extract from solvent extraction of residuum. It consists predominantly of high molecular weight compounds with high carbon-to-hydrogen ratios (ChemIDplus).

1.4. Structural Formula

Not applicable.

1.5. Molecular weight (g/mol)

Its components have molecular weights in the range of 500 to 2000 Daltons.(Health Canada and Environment and Climate Change Canada, 2022)*1.6. CAS registration number*

1.6. CAS registration number

64742-16-1

1.7. Properties

1.7.1. Melting point

(°C): 30.00-60.00 (estimated by anology) (US EPA, 2021)

1.7.2. Boiling point

(°C): >482 (US EPA, 2021)

1.7.3. Solubility

<1.0×10^{-4} mg/L, based upon representative structures. (estimated) (US EPA, 2021); less than 10^{-5} mg/L; The water solubility range of components of petroleum resins is between 3.5 x 10-4 and 8.1 x 10-4 mg/L at 20°C and a pH between 6.22 and 7.42 (ECHA 2019a). (Health Canada and Environment and Climate Change Canada, 2022)

1.7.4. pKa

No data available to us at this time.

1.7.5. Flashpoint

(°C): No data available to us at this time.

1.7.6. Flammability limits (vol/vol%)

No data available to us at this time.

1.7.7. (Auto)ignition temperature

(°C): No data available to us at this time.

1.7.8. Decomposition temperature

(°C): No data available to us at this time.

1.7.9. Stability

No data available to us at this time.

1.7.10. Vapor pressure

<$1.0\text{x}10^{-10}$ to $2.6\text{x}10^{-7}$ mmHg at 25°C, range based upon representative structures. (estimated) (US EPA, 2021); 3×10^{-7} mm Hg ($4\text{x}10^{-5}$ Pa) or less (Health Canada and Environment and Climate Change Canada, 2022)

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> 10 (estimated) (US EPA, 2021)

2. General information

2.1. Exposure

Petroleum resins (CAS RN 64742-16-1) are listed as ingredients in a home maintenance (at 5-10%) and an “old” inside the home (at 33%) product by the CPID.

The following Australian industrial uses were reported under previous mandatory and/or voluntary calls for information.

Petroleum resins (CAS No. 64742-16-1) have reported commercial use including as: additives in construction materials; and lubricants and additives.

Petroleum resins (CAS No. 64742-16-1) has reported potential domestic use in adhesives and binding agents.

Petroleum resins (CAS No. 64742-16-1) has reported site-limited use in stabilisers.

The following chemicals are listed on the 2006 High Volume Industrial Chemicals List (HVICL): petroleum resins (CAS No. 64742-16-1) with a total reported volume of 1000–9999 tonnes.

The following international uses have been identified through the European Union (EU) Registration, Evaluation and Authorisation of Chemicals (REACH) dossiers (REACH a; REACH b); the Substances and Preparations in the Nordic countries (SPIN) database; the OECD High Production Volume chemical program (OECD HPV); and various international assessments/documents (IPCS 2004; Asphalt Institute 2011, EPA 2006; IARC 2013):

the chemicals with CAS Nos 8052-42-4, 64742-16-1, 64742-85-4 and 64742-93-4 have reported commercial use including:

as absorbents;

as fuel additives;

as impregnation materials;

as lubricants and additives;

in processing for rubber production; and

as reprographic agents.

Domestic uses have been identified for chemicals with CAS Nos 8052-42-4, 64742-16-1, 64742-85-4 and 64742-93-4.

The chemicals with CAS Nos 64742-16-1 and 64742-85-4 have reported domestic uses in the Substances and Preparations in the Nordic countries (SPIN) database including:

as adhesives, binding agents;

as corrosion inhibitors;

as fillers;

in insulating materials; and

in paints, lacquers and varnishes.

However, it should be noted that SPIN does not distinguish between direct use of the chemicals, or use of the materials that are produced from chemical reactions involving the chemical.

As taken from AICIS, 2018

Petroleum resins (CAS RN 64742-16-1) are used for adhesive purposes in non-medicinal natural health products (Health Canada, 2021).

“Petroleum resins is used in asphalt, adhesives and sealants, lubricants and greases, and polishes and waxes. Petroleum resins appears in some cosmetics and natural health products as an adhesive.”

“According to information submitted in response to a CEPA section 71 survey, and a voluntary data gathering initiative, uses of petroleum resins in Canada (ECCC 2015, 2016) include in enamels, primers, and automotive protective paint materials and sealants.”

“Petroleum resins is either sold in its original form, or is blended with, or processed with other asphaltic materials to produce end-use asphalt products, including speciality products such as pipe coatings, roofing adhesives, some special-use paints, and lubricants (US EPA 2011a). Searches of material safety data sheet (MSDS) databases identified additional potential uses, including use as adhesives and coatings (MSDS 2015a), as an ingredient in asphalt modifiers (MSDS 2016a), and in lubricants (MSDS 2015b).

With regards to non-asphalt related uses, petroleum resins is one of the components of hot melt glues (including glue sticks used by crafters) and tackifiers, in which it is present up to concentrations of 10-60%. Petroleum resins is used in solvent-based construction adhesives in mass fractions of 1-5% (MSDS 2004, 2011, 2016b).

There is no definitive information on the use of petroleum resins in food packaging or as an incidental additive in Canada (personal communication, email from the Foods Directorate, Health Canada (HC) to the Existing Substances Risk Assessment Bureau, HC, dated June 21, 2019; unreferenced). It does not appear in Therapeutic Products as a medicinal or non-medicinal ingredient (personal communication, email from the Therapeutic Products Directorate, HC to the Existing Substances Risk Assessment Bureau, HC, dated March 7, 2019; unreferenced), however, it appears in one licensed natural health product (LNHPD 2018), and in a limited number of cosmetic products as an adhesive in patch products (personal communication, email from the Consumer and Hazardous Products Safety Directorate, HC to the Existing Substances Risk Assessment Bureau, HC, dated March 14, 2019; unreferenced)."

As taken from Health Canada and Environment and Climate Change Canada, 2022

2.2. Combustion products

No data available to us at this time.

2.3. Ingredient(s) from which it originates

All petroleum products are derived from crude oil whose major constituents are hydrocarbons. Petroleum components can be separated into four fractions, the saturated, aromatic, resin and asphaltene fractions, by absorption chromatography. Each of these fractions contains a large number of compounds (Karlsen and Larter,1991).

As taken from Harayama et al., Petroleum Biodegradation in Marine Environments, J. Molec. Microbiol. Biotechnol. (1999) 1(1): 63-70, available at http://www.horizonpress.com/jmmb/v1/v1n1/10.pdf

"TSCA Definition 2019: A complex combination of organic compounds, predominantly hydrocarbons, obtained as a fraction of the extract from solvent extraction of residuum. It consists predominantly of high molecular weight compounds with high carbon-to-hydrogen ratios".

As taken from ChemIDplus.

Petroleum resins (CAS RN 64742-16-1) is a complex combination of organic compounds, predominantly hydrocarbons, obtained as a fraction of the solvent extract of residuum, which is derived from the vacuum distillation of petroleum (API 2017).

As taken from Health Canada and Environment and Climate Change Canada, 2022

3. Status in legislation and other official guidance

Petroleum resins are included on the US FDA's Inventory of Food Contact Substances Listed in 21 CFR and covered under 21 CFR sections 175.105 (adhesives), 176.180 (components of paper and paperboard in contact with dry food), 177.2600 (rubber articles intended for repeat use) and 178.3800 (preservatives for wood).

As taken from FDA, 2022a,b

Petroleum resins (no CAS RN given) are permitted for use as inert ingredients in non-food pesticide products by the US EPA (US EPA InertFinder Database, 2022).

There is a REACH dossier on petroleum resins (CAS RN 64742-16-1) (ECHA, 2021).

Petroleum resins (CAS RN 64742-16-1) are not classified for packaging and labelling under Regulation (EC) No. 1272/2008 (ECHA, 2022).

Petroleum resins (CAS RN 64742-16-1) are listed in the US EPA Toxic Substances Control Act (TSCA) inventory, and also in the US EPA 2012 CDR and 2020 CDR Partial Exempt lists (Chemical Data Reporting Rule).

Petroleum resins (CAS RN 64742-16-1) are listed by the US EPA Office of Pesticide Programs (2021) and were first registered as antimicrobial and "conventional chemical" pesticides on 13 November 1984.

Petroleum resins is considered within the United States Environmental Protection Agency (US EPA) asphalt category and is described as predominantly comprised of high molecular weight compounds with high carbon-to-hydrogen ratios and a carbon number greater than 25 (US EPA 2011a, 2011b).

As taken from Health Canada and Environment and Climate Change Canada, 2022

4. Metabolism/Pharmacokinetics

4.1. Metabolism/metabolites

"They are expected to be oxidatively metabolised and slowly eliminated in the urine and faeces (IARC, 2013). PACs [polycyclic aromatic compounds] are also a component of the chemicals (at ppm levels), or may be present at higher concentrations in fumes generated during use. Following inhalation, ingestion, or skin contact, they are expected to be metabolised and subsequently eliminated by urinary or biliary excretion."

As taken from AICIS, 2018

4.2. Absorption, distribution and excretion

"The chemicals are not expected to be absorbed dermally, due to their high molecular weight and large molecular size, low water solubility and negligible vapour pressure"

"Long chain aliphatic hydrocarbons are a major component of asphalt. Following inhalation of such chemicals, hydrocarbons of 9–16 carbon atoms were found in the blood, brain, liver, kidneys and fat of rats. Aerosols of hydrocarbons with >16 carbon atoms were found in the lung and liver in mice. They are expected to be oxidatively metabolised and slowly eliminated in the urine and faeces (IARC, 2013). PACs [polycyclic aromatic compounds] are also a component of the chemicals (at ppm levels), or may be present at higher concentrations in fumes generated during use. Following inhalation, ingestion, or skin contact, they are expected to be metabolised and subsequently eliminated by urinary or biliary excretion. Whole body studies in rodents have also demonstrated detectable levels of polycyclic aromatic hydrocarbons in the majority of internal organs (IARC, 2013)."

As taken from AICIS, 2018

4.3. Interactions

No data available to us at this time.

5. Toxicity

5.1. Single dose toxicity

Type of Test	Route of Exposure or Administration	Species/Test System	Dose Data	Toxic Effects	Reference
LD50 - Lethal	Oral	Mammal - species	7 gm/kg	Behavioral - somnolence	GTPZAB Gigiena Truda i Professional'nye Zabolevaniya.

dose, 50 percent kill		unspecified		(general depressed activity) Behavioral - excitement Behavioral - muscle contraction or spasticity	Labor Hygiene and Occupational Diseases. (V/O Mezhdunarodnaya Kniga, 113095 Moscow, USSR) V.1-36, 1957-1992. For publisher information, see MTPEEI Volume(issue)/page/year: 32(4),55,1988

As taken from ChemIDplus; RTECS, 1997

“Based on the data available, the chemicals in this group have low acute toxicity based on results from animal tests following oral exposure to residues, petroleum, vacuum (CAS No. 64741-56-6).”

“Based on the data available, the chemicals in this group have low acute toxicity based on results from animal tests following dermal exposure to residues, petroleum, vacuum (CAS No. 64741-56-6).”

“Based on the data available, the chemicals in this group have low acute toxicity following inhalation exposure.”

As taken from AICIS, 2018

In an acute oral toxicity study (OECD test guideline 423), single-dose exposure to 2000 mg/kg-bw (gavage) of petroleum resin (CAS 64742-16-1) did not cause any mortality or signs of toxicity in female rats that were examined daily for up to 14 days postexposure. A necropsy examination performed at the end of the study did not reveal any gross abnormalities. The study authors determined the LD50 value to be >2000 mg/kg bw (ECHA 2019a; 2018c).

As taken from Health Canada and Environment and Climate Change Canada, 2022

5.2. Repeated dose toxicity

Petroleum resins (CAS 64742-16-1)

Repeated dose toxicity

Study type	Summary	Length/duration
Estimated by analogy	In a combined repeated-dose/reproductive/developmental toxicity study [OECD TG 422] Wistar rats (12/sex/dose) were exposed via inhalation (nose-only) to roofing asphalt fume condensate to target concentrations of 0, 0.03, 0.10 and 0.30 mg/L total hydrocarbons (actual concentrations were 0.03, 0.10 and 0.297 mg/L). Animals were acclimated for three weeks prior to treatment to acquaint them with the nose-only apparatus. Also, male animals were appropriately randomized to test groups but females were not (due to an error in the randomization program, females were assigned by body weight so that at study start there was a statistically significant difference in body weight between control	males for six hours/day, seven days/week for 28 days, and females were exposed for 35-48 days

	and treated animals (controls were lower than treated groups)).Males were exposed to control and test atmospheres for six hours/day, seven days/week for 28 days, and females were exposed for 35-48 days (14 days pre-mating, up to 14 days during mating and for 20 days during gestation; with some animals receiving less based on the date of conception). There were no exposures to females after gestation day 20 or during the four-day post-partum lactation period. Females for which there was no evidence of copulation were dosed for 54 days. The robust summary is ambiguous about the number of females treated since there appear to be two groups – one pregnant and one not pregnant (termed the "breeding" and "subchronic" female groups, respectively).There were no deaths and no adverse clinical signs observed during the study. Body weight gain and food consumption were significantly reduced in high concentration males. There was no effects on female body weight; however as stated above, females were inappropriately randomized by body weight. There were no treatment-related changes in clinical chemistry or hematology parameters (details not provided in robust summary). The following statistically significant organ weight changes were observed: increases in absolute and relative lung weights(high concentration males; mid and high concentration "subchronic females5" and high concentration "breeding females"); increases in absolute lung weights only (low concentration "subchronic females" and mid concentration "breeding females"); and increases in absolute liver weight (high concentration "subchronic females"; although non-significant increases were noted in low and mid concentration "subchronic females"). There were no pathological lesions in the liver or any other organs except for the lung and nasal cavity. Lung effects observed included a slight increase in alveolar macrophage accumulation in conjunction with minimal mononuclear/inflammatory cell infiltration. Also, there was a minimal to slight increase in alveolar hyperplasia in the bronchioles (considered adaptive). These lung effects were seen only in high concentration animals. There was a statistically significant decrease of inflammatory cell infiltration in high concentration "subchronic females". Also, an increase in inflammatory cell infiltration was seen in the nasal cavity of high concentration males. There were no adverse effects noted for any group in the neurobehavioral tests that were performed. LOAEC (systemic, males) = 0.3 mg/L/day (based on decreased body weight gain, increased absolute and relative lung weight, and histopathology in the lungs)NOAEC (systemic, males) = 0.1 mg/L/day LOAEC (systemic, females) = 0.1 mg/L/day (based on increased absolute and relative lung weight)NOAEC (systemic, females) = 0.03 mg/L/day. Roofing Asphalt fume condensate (supporting chemical; derived from CASRN 64742-93-4)	
Estimated by analogy	New Zealand White rabbits (5/sex/dose) were administered residues (petroleum), vacuum(API sample 81-13) via the dermal route at 0, 200, 1000 or 2000 mg/kg-bw/day to clipped dorsal skin under occluded conditions for 6 hours/day, 3 days/week for 4 weeks. Two animals died and two were sacrificed moribund during the study – the identity of the dose groups for these mortalities were not reported in the robust summary; however the full report is available in TSCATS (OTS 0000186-1) and shows that one control female and one high dose male were found dead, and one	6 hours/day, 3 days/week for 4 weeks

	control male and a mid-dose female were sacrificed during the study. This supports the conclusion that these deaths were not likely treatment-related. Treatment-related clinical signs observed in survivors included thin appearance, decreased food intake, flaking skin and wheezing (doses not stated). All animals treated with residues (petroleum), vacuum exhibited slight edema. Decreased body weight gain was observed in males at 2000mg/kg-bw/day. There were no treatment-related changes in the hematology parameters. Alkaline phosphatase was reduced by 50% in males at 2000 mg/kg-bw/day. Changes in absolute and/or relative organ weights were observed at 2000 mg/kg-bw/day (adrenal, kidney, pituitary and spleen), but were not considered to be treatment-related. Treatment-related gross necropsy and microscopic findings were confined to the skin. The skin of females appeared to be more severely affected. Effects in females were limited to the point of contact with the test substance. Incidental findings were observed and were consistent with Encephalitozoon infection. LOAEL (systemic) = 2000 mg/kg-bw/day (based on decreased body weight gain and reduced alkaline phosphatase in males)NOAEL (systemic) = 1000 mg/kg-bw/day LOAEL (local) = 200 mg/kg-bw/day (lowest tested dose) (based on irritation)NOAEL (local) = Not established. Residues (petroleum), vacuum (CASRN 64741-56-6)	
Estimated by analogy	New Zealand White rabbits (5/sex/dose) were administered residues (petroleum), vacuum(API sample 81-14) via the dermal route at 0, 200, 1000 or 2000 mg/kg-bw/day using the same methodology described above for API sample 81-3. Details of this study were not presented in the robust summary, but the full report is available in TSCATS (OTS 0000186-1). Two animals died during the study (low dose male and high dose female), and the deaths were considered treatment-related due to clinical signs observed prior to death being consistent with clinical signs observed in survivors. Treatment-related clinical signs included thin appearance, decreased food intake, flaking skin and wheezing at >/= 200 mg/kg-bw/day. Reduced body weight was observed in females at 2000 mg/kg-bw/day and considered treatment-related. Changes in hematological, clinical chemistry, and organ weights were observed, but not in a dose-related manner and so were not considered treatment-related (limited information supporting this was presented in the TSCATS report, but associated tables and appendices with the raw data were not provided).Edema was observed at >/= 200 mg/kg-bw/day and erythema was observed at >/= 1000 mg/kg bw/day. Histopathological observations were confined to the skin and consisted of subacute acanthotic dermatitis and hyperkeratosis at 2000 mg/kg-bw/day. Incidental findings associated with Encephalitozoon infection included meningoencephalitis, nephritis and periportal lymphoid infiltrates. LOAEL (systemic) = 2000 mg/kg-bw/day (based on reduced body weight in females and mortality)NOAEL (systemic) = 1000 mg/kg-bw/day LOAEL (local) = 200 mg/kg/day (lowest tested dose) (based on irritation)NOAEL (local) = Not established. Residues (petroleum), vacuum (CASRN 64741-56-6)	6 hours/day, 3 days/week for 4 weeks

As taken from US EPA, 2022.

“Based on the data available, the chemicals in this group are not considered to cause serious damage to health from repeated dermal exposure.”

“Based on the data available, the chemicals in this group are not considered to cause serious damage to health from repeated inhalation exposure.”

As taken from AICIS, 2018.

5.3. Reproduction toxicity

Petroleum resins
64742-16-1
Developmental toxicity:

Study type	Summary	Length/duration
Combined Repeated Dose with Reproduction/ Developmental Toxicity Screen	In a combined repeated-dose/reproductive/developmental toxicity study [OECD TG 422] Wistar rats (12/sex/dose) were exposed via inhalation (nose-only) to roofing asphalt fume condensate to target concentrations of 0, 0.03, 0.10 and 0.30 mg/L total hydrocarbons (actual concentrations were 0.03, 0.10 and 0.297 mg/L). Animals were acclimated for three weeks prior to treatment to acquaint them with the nose-only apparatus. Also, male animals were appropriately randomized to test groups but females were not (due to an error in the randomization program, females were assigned by body weight so that at study start there was a statistically significant difference in body weight between control and treated animals (controls were lower than treated groups)).Males were exposed to control and test atmospheres for six hours/day, seven days/week for 28 days, and females were exposed for 35-48 days (14 days pre-mating, up to 14 days during mating and for 20 days during gestation; with some animals receiving less based on the date of conception). There were no exposures to females after gestation day 20 or during the four-day post-partum lactation period. Females for which there was no evidence of copulation were dosed for 54 days. The robust summary is ambiguous about the number of females treated since there appear to be two groups – one pregnant and one not pregnant (termed the “breeding” and “subchronic” female groups, respectively).Wistar rats (12/sex/dose) were exposed via inhalation (nose-only) to roofing asphalt fume condensate to target concentrations of 0, 0.03, 0.10 and 0.30 mg/L total hydrocarbons (actual concentrations were 0.03, 0.10 and 0.297 mg/L) in a combined repeated dose/reproductive/developmental toxicity study following the OECD 422 protocol that has already been summarized above. On post-partum day 4, all lactating females and offspring were sacrificed. In addition to reproductive organs evaluated for the repeated-dose portion of the study (along with sperm evaluations), the following reproductive parameters were evaluated: pregnancy incidence, number of pregnant animals delivering, mating index, fertility index, gestation length,	males for six hours/day, seven days/week for 28 days, and females were exposed for 35-48 days

	number of implantation sites, number of corpora lutea. There were no treatment-related affects on any reproductive toxicity parameter. Although there were no statistically significant differences observed in any of the sperm parameters assessed, there was a dose-dependent trend (not statistically significant) in a decrease in sperm number (38.125,32.250, 27.125, and 24.542 million sperm/epididymis for the control, low, mid and high concentrations, respectively). There were no effects observed in developing animals (no differences in pup sex ratio, pup body weight, pup body weight gain, and no apparent, gross malformations).NOAEC (reproductive) > 0.3 mg/L/day (highest tested concentration)LOAEC (maternal) = 0.1 mg/L/day (based on lung effects in females; see Repeated-Dose Toxicity section for description)NOAEC (maternal) = 0.03 mg/L/day NOAEC (developmental) > 0.3 mg/L/day (highest tested concentration).Roofing Asphalt fume condensate (supporting chemical; derived from CASRN 64742-93-4)	

As taken from US EPA, 2022

“Certain petroleum stream chemicals have been shown to be developmentally toxic by the dermal route of exposure. Effects include increased incidence of early and total resorptions and decrease in foetal body weight (IPIECA 2010; Murray et al. 2013).”

As taken from AICIS, 2018

Petroleum resins, hydrocarbon resin, and polymerized C5-12 distillates were not identified as posing a hazard to human health based on classifications by other national or international agencies for carcinogenicity, genotoxicity, developmental toxicity, or reproductive toxicity.

As taken from Health Canada and Environment and Climate Change Canada, 2022

5.4. Mutagenicity

Petroleum resins

CAS 64742-16-1

Mutagenicity/Genetic toxicity

Study type	Summary
Estimated by Analogy	Some rats from the two year cancer bioassay described below (Fuhst et al., 2007) were evaluated for chromosome damage via a micronucleus assay (the results of which are reported separately in Halter et al., 2007). Wistar rats (6/sex/dose/exposure period for this portion of the study) were exposed via inhalation (nose-only) to roofing asphalt fume condensate to target concentrations of0, 0.004, 0.020 and 0.100 mg/L total hydrocarbon concentration (THC). Peripheral blood was taken from the tail vein at 5 days, 20 days and 12 months of exposure and analyzed for micronuclei in erythrocytes. These animals were

	sacrificed at 12 months and the bone marrow was also extracted and analyzed for micronuclei formation. There were no positive controls. Results showed no increases in micronuclei formation in peripheral blood after 5 days, 20 days, or 12 months of exposure; and no increases in micronuclei formation in bone marrow after 12months.Paving asphalt fume condensate did not induce micronuclei formation in peripheral blood or bone marrow erythrocytes in this assay. Paving Asphalt fume condensate (supporting chemical; derived from CASRN 64742-93-4)
Estimated by Analogy	Wistar rats (12/sex/dose) were exposed via inhalation (nose-only) to roofing asphalt fume condensate to target concentrations of 0, 0.03, 0.10 and 0.30 mg/L total hydrocarbons (actual concentrations were 0.03, 0.10 and 0.297 mg/L) in a combined repeated dose/reproductive/developmental toxicity study following the OECD 422 protocol. Five rats/sex/group were used to evaluate the induction of micronuclei in bone marrow erthryocytes. A positive control group (cyclophosphamide) was also used. One femur from each rat was collected 24 hours after the last day of 28 days of exposure to the test material. Results showed the positive control responded appropriately and there was no induction of micronuclei in any treated group (all data presented in robust summary).Roofing asphalt fume condensate did not induce micronuclei in rat bone marrow erythrocytes. Roofing Asphalt fume condensate (supporting chemical; derived from CASRN 64742-93-4)
Estimated by Analogy	In two bone marrow cytogenetic studies, Sprague-Dawley rats (10/sex/dose) were administered residues (petroleum), vacuum (API samples 81-13 and 81-14) as solutions in corn oil via gavage for 5 days at 300, 1000 or 3000 mg/kg-bw/day for API 81-13 or 400, 1300 or 4000mg/kg-bw/day for API 81-14. Positive and negative controls were tested concurrently and yielded expected results. No increase in the number of chromosomal aberrations was found in rats treated with either test substance, compared to the negative controls. Sialodacryoadenitis(SDAV) infections were observed in several rats upon necropsy, but SDAV is common among rats and is not believed to have influenced the results. Residues (petroleum), vacuum did not induce chromosomal aberrations in this assay. Residues (petroleum), vacuum (CASRN 64741-56-6)
Estimated by Analogy	Some rats from the two year cancer bioassay described below (Fuhst et al., 2007) were evaluated for DNA adduct formation (results of which are reported separately in Halter et al., 2007).Wistar rats (8/sex/dose/exposure period for this portion of the study) were exposed via inhalation(nose-only) to roofing asphalt fume condensate to target concentrations of 0, 0.004, 0.020 and0.100 mg/L THC. DNA from the lung, nasal and alveolar epithelium was collected from rats at5 days, 30 days and 12 months. Results showed an increase in 3-4 stable DNA adducts in these tissues over the endogenous adducts seen in the clean air control animals. Paving asphalt fume condensate did induce DNA adduct formation in lung, nasal and alveolar epithelium in this assay.. Paving Asphalt fume condensate (supporting chemical; derived from CASRN 64742-93-4)
Estimated by Analogy	In two studies, mouse lymphoma cells (L5178Y TK+/-) were exposed to residues (petroleum),vacuum (API samples 81-13 or 81-14) at concentrations ranging from 62.5 to 1000 nL/mL with and without metabolic activation. Positive and negative controls were tested concurrently and yielded expected results (data provided in robust summary). In both studies, residues(petroleum), vacuum was not mutagenic without activation, but was weakly mutagenic with metabolic activation (TSCATS: OTS0000175-1).Residues (petroleum), vacuum was mutagenic in this assay (only under metabolic activation conditions)..Residues (petroleum), vacuum (CASRN 64741-56-6)

As taken from US EPA, 2022

Based on the weight of evidence, the chemicals in this group (as whole materials) are not considered to be mutagenic.

As taken from AICIS, 2018

Similarly, in an OECD test guideline 471 study, petroleum resin was found to be non-mutagenic in a bacterial reverse mutation assay in the presence and absence of metabolic activation in various Salmonella typhimurium strains.

As taken from Health Canada and Environment and Climate Change Canada, 2022

5.5. Cytotoxicity

No data available to us at this time.

5.6. Carcinogenicity

“Based on the available data, the chemicals in this group as whole materials are not considered carcinogenic, although dilution in organic solvents may produce some carcinogenic effects following prolonged dermal exposure.”

As taken from AICIS, 2018

Petroleum resins, hydrocarbon resin, and polymerized C5-12 distillates were not identified as posing a hazard to human health based on classifications by other national or international agencies for carcinogenicity, genotoxicity, developmental toxicity, or reproductive toxicity.

As taken from Health Canada and Environment and Climate Change Canada, 2022

5.7. Irritation/immunotoxicity

“Based on the available data, the chemicals in this group may slightly irritate skin in animal studies, particularly following repeated exposure.”

“Based on the available data, the chemicals in this group may be, at most, slightly irritating to the eye in animal studies.”

“The negative results observed for residues, petroleum, vacuum (CAS No. 64741-56-6), in several skin sensitisation animal studies conducted in accordance with OECD TG 406 (Buehler test), support a conclusion that the chemicals in this group are not skin sensitisers.

As taken from AICIS, 2018

This substance did not show potential to cause skin sensitization in standard in vitro assays (ECHA 2018c).

As taken from Health Canada and Environment and Climate Change Canada, 2022

5.8. All other relevant types of toxicity

No data available to us at this time.

6. Functional effects on

6.1. Broncho/pulmonary system

No data available to us at this time.

6.2. Cardiovascular system

No data available to us at this time.

6.3. Nervous system

No data available to us at this time.

6.4. Other organ systems, dependent on the properties of the substance

No data available to us at this time.

7. Addiction

JTI is not aware of any information that demonstrates that this ingredient has any addictive effect.

8. Burnt ingredient toxicity

No data available to us at this time.

9. Heated/vapor emissions toxicity

No data available to us at this time.

10. Ecotoxicity

10.1. Environmental fate

More information can be found in Geraci and & St. Aubin, SYNTHESIS OF EFFECTS OF OIL ON MARINE MAMMALS, Department of Interior Minerals Management Service Atlantic OCS Region, Contract No. 14-1 2-0001 -30293, September 1988 (no weblink available because of secured access)

Persistence is reported to be high (US EPA, 2011)

Fugacity

(Level III Model)

Air (%) <0.1–0.8

Water (%) 3.9–37.2

Soil (%) 62.0–93.8

Sediment (%) <0.1–25.1

(US EPA, 2011)

The Ecological Categorization Results from the Canadian Domestic Substances List simply state that petroleum resins are persistent in the environment.

Data accessed December 2014 on the OECD website: http://webnet.oecd.org/CCRWeb/Search.aspx

10.2. Aquatic toxicity

“The state of the Ob ecosystem has sharply deteriorated for recent 25 years because of high rates and scales of hydrocarbon raw material mining in the Tyumen region. The external level of pollution is characteristic of the flood bed of the Middle Ob and lower current of the Irtysh. The grounds of both flood plain water bodies and the Ob and Irtysh beds, a migration way of fluvial anadromous fish are greatly polluted with oil. Destruction of biotopes, sharp intoxication during the emergency situations, sublethal effect via deterioration of nutrition under chronic action followed by a decrease in resistivity of organisms to the changes in environmental parameters and bioaccumulation of toxic and cancerogenic hydrocarbons induce a decrease in reproduction of biological production at all the levels of trophic chain."

As taken from Mikhailova LV, Current hydrochemical regimen and the effect of pollution on an aquatic ecosystem and pisciculture in the Ob River basin, GIDROBIOL ZH; 27 (5). 1991. 80-90. Toxline powered by Toxnet, 2010 available at http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?TOXLINE

"When petroleum is spilled into the sea, it spreads over the surface of the water. It is subjected to many modifications, and the composition of the petroleum changes with time. This process is called weathering, and is mainly due to evaporation of the low-molecular-weight fractions, dissolution of the water-soluble components, mixing of the oil droplets with seawater, photochemical oxidation, and biodegradation.

Those petroleum components with a boiling point below 250 °C are subjected to evaporation. Therefore, the content of n-alkanes, whose chain length is shorter than C14, is reduced by weathering. The content of aromatic hydrocarbons within the same boiling point range is also reduced as they are subjected to both evaporation and dissolution. The mixing of oil with seawater occurs in several forms. Dispersion of the oil droplets into a water column is induced by the action of waves, while water-inoil emulsification occurs when the petroleum contains polar components that act as emulsifiers. A water-in-oil emulsion containing more than 70% of seawater becomes quite viscous; it is called chocolate mousse from its appearance. After the light fractions have evaporated, heavy residues of petroleum can aggregate to form tar balls whose diameter ranges from microscopic size to several tenths of a centimeter.

After a large oil spill, the oil slick is sometimes treated with a dispersant. Dispersants emulsify petroleum by reducing the interfacial tension between petroleum and water. The small droplets that are formed are dispersed into a water column to a depth of several meters, preventing wind-induced drift of the oil slick. It is claimed that treatment by a dispersant enhances the biodegradation of petroleum. However, the results of such tests are controversial (Tjessem et al., 1984). The original dispersants used were highly toxic; however, less toxic dispersants have subsequently been developed.

Under sunlight, petroleum discharged at sea is subjected to photochemical modification. Some reports have suggested the light-induced polymerization of petroleum components, while others have suggested their photodegradation. An increase in the polar fraction and a decrease in the aromatic fraction have also been observed.

Aliphatic components do not significantly absorb solar light, and are by themselves photochemically inert. However, they can be degraded by photosensitized oxidation. The aromatic or polar components in petroleum and anthraquinone that is present in seawater can provoke the degradation of n-alkanes into terminal n-alkenes (a carboncarbon double bond at position 1) and low-molecular-weight carbonyl compounds (Ehrhardt and Weber, 1991).

The water-soluble components of petroleum exert a toxic effect on marine organisms. In general, aromatic compounds are more toxic than aliphatic compounds, and smaller molecules are more toxic than larger ones in the same series. Solar irradiation affects oil toxicity: Surface films become less toxic due to the loss of polycyclic aromatic hydrocarbons, but the toxicity of the water-soluble fraction increases as its concentration increases (Nicodem et al., 1997).

Many catastrophic oil spills from large tanker accidents have attracted public attention to the fate of petroleum hydrocarbons in marine environments. In response to this concern, research into the biodegradation of petroleum in natural environments has been intensified. The pioneering studies by Atlas, Bartha and their colleagues (Atlas and Bartha, 1993; Prince, 1993) have demonstrated that the available concentrations of nitrogen and phosphorus in seawater are limiting factors for the growth of hydrocarbondegrading microorganisms. Thus, the addition of nitrogen and phosphorus fertilizers stimulates the biodegradation of petroleum. In general, small hydrocarbon molecules are more easily biodegraded than larger ones, and aromatics are degraded at a much slower rate than that of alkanes in marine environments (Oudot, 1984; Kennicut, 1988; Ishihara et al., 1995; Sugiura et al., 1997; Sasaki et al., 1998; Wang et al., 1998).

Many oil spills in the sea cause shoreline pollution, despite efforts to prevent the drift of a spill toward the coastline. Cleaning up a polluted coastline by enhancing microbial activities was first attempted in 1989 after the spill from the Exxon Valdez. The initial measure taken after this accident was physical washing with high-pressure water. Subsequently, fertilizers were applied to the polluted beaches to accelerate the growth and activities of petroleum-degrading microorganisms. Two to three weeks later, pebbles on the beaches that had been treated with fertilizers had become significantly cleaner than those in the control area (Pritchard and Costa, 1991). Nevertheless, it was difficult to evaluate the effect of the treatment due to heterogeneity in the oil contamination of samples. Fivering cycloalkanes, hopanes (Figure 1), are frequently used as a conserved quantitative internal standard because they are resistant to biodegradation. Using hopanes as an internal standard, it has been demonstrated that the fertilizer application significantly increased the rate of petroleum biodegradation, and that about half of the petroleum had been removed within three months after applying fertilizers in sufficient amounts (Bragg et al., 1994).

Although hydrocarbons have usually been found to persist under strict anaerobic conditions, these compounds are degraded in some types of marine harbor sediment under sulfate-reducing conditions. When hydrocarbondegrading sediment is used to inoculate a type of sediment that shows no hydrocarbon-degrading activity under anaerobic conditions, such activity is generated. This observation indicates that hydrocarbon contamination could be treated under sulfate-reducing conditions, and that the seeding (introduction of foreign microorganisms or equivalent samples) of anaerobic microbial consortia adapted to specific hydrocarbons would be effective to enhance the anaerobic biodegradation of these hydrocarbons (Coates et al., 1997; Weiner and Lovley, 1998)."

As taken from Harayama et al., Petroleum Biodegradation in Marine Environments, J. Molec. Microbiol. Biotechnol. (1999) 1(1): 63-70, available at http://www.horizonpress.com/jmmb/v1/v1n1/10.pdf

According to the Ecological Categorization results from the Canadian Domestic Substances List, petroleum resins are of uncertain inherent toxicity to aquatic organisms [no further data given].

Data accessed December 2014 on the OECD website: http://webnet.oecd.org/CCRWeb/Search.aspx

"Embryonic exposures to the components of petroleum, including polycyclic aromatic hydrocarbons (PAHs), cause a characteristic suite of developmental defects and cardiotoxicity in a variety of fish species. We exposed zebrafish embryos to reference sediment mixed with laboratory weathered South Louisiana crude oil and to sediment collected from an oiled site in Barataria Bay, Louisiana in December 2010. Laboratory oiled sediment exposures caused a reproducible set of developmental malformations in zebrafish embryos including yolk sac and pericardial edema, craniofacial and spinal defects, and tissue degeneration. Dose-response studies with spiked sediment showed that total polycyclic aromatic hydrocarbons (tPAH) concentrations of 27mg tPAH/kg (dry weight normalized to 1 percent organic carbon [1 percent OC]) caused a significant increase in defects, and concentrations above 78mg tPAH/kg 1 percent OC caused nearly complete embryo mortality. No toxicity was observed in Barataria sediment with 2mg tPAH/kg 1 percent OC. Laboratory aging of spiked sediment at 4°C resulted in a nearly 10-fold decrease in sensitivity over a 40-day period. This study demonstrates oiled sediment as an exposure pathway to fish with dose-dependent effects on embryogenesis that are consistent with PAH mechanisms of developmental toxicity. The results have implications for effects on estuarine fish from oiled coastal areas during the Deepwater Horizon spill." As taken from Raimondo S et al. 2014. Ecotoxicol. Environ. Saf. 108, 265-272. PubMed, 2014 available at http://www.ncbi.nlm.nih.gov/pubmed/25105486

10.3. Sediment toxicity

"A study evaluating sediment toxicity in an estuarine marshland near Houston, Texas, was conducted following a major petroleum pipeline rupture during a flood event. Acute sediment toxicity was measured by performing the Microtox® bioassay 100% Test on elutriates from wet sediment samples collected from experimental plots in the study area. Samples were collected over a seven-month period following the spill. Toxic responses were examined for spatial and temporal relationships within the cove and compared to total extractable materials (TEM), total petroleum hydrocarbon (TPH), and GC-MS quantified total saturates and aromatics and target polycyclic aromatic hydrocarbons (PAHs). Sediment toxicity was elevated near the mouth of the study cove and decreased rapidly with time. Acute toxicity was correlated with TPH and GC-MS quantified saturate concentrations. However, toxicity levels were not correlated with TEM or GC-MS aromatic summations and target PAHs. The rapid decrease in sediment toxicity corresponded with a rapid decrease in oil levels suggesting that the intrinsic recovery of the site was due to acclimated populations of hydrocarbon-degrading microorganisms."

As taken from Mueller et al., Acute Toxicity of Estuarine Wetland Sediments Contaminated by Petroleum, Environmental Technology, Volume 20 Issue 8, 1999, available at http://www.informaworld.com/smpp/content~db=all~content=a794045103

10.4. Terrestrial toxicity

"The potential of fungal co-culture of the filamentous Pestalotiopsis sp. NG007 with four different basidiomycetes--Trametes versicolor U97, Pleurotus ostreatus PL1, Cerena sp. F0607, and Polyporus sp. S133--for accelerating biodegradation of petroleum hydrocarbons (PHCs) was studied using three different physicochemical characteristic PHCs in soil. All the combinations showed a mutual intermingling mycelial interaction on the agar plates. However, only NG007/S133 (50/50) exhibited an optimum growth rate and enzymatic activities that supported the degradation of asphalt in soil. The co-culture also degraded all fractions at even higher concentrations of the different PHCs. In addition, asphaltene, which is a difficult fraction for a single microorganism to degrade, was markedly degraded by the co-culture, which indicated that the simultaneous biodegradation of aliphatic, aromatic, resin, and asphaltene fractions had occurred in the co-culture. An examination of in-vitro degradation by the crude enzymes and the retrieval fungal culture from the soil after the experiment confirmed the accelerated biodegradation due to enhanced enzyme activities in the co-culture. The addition of piperonyl butoxide or AgNO3 inhibited biodegradation by 81-99%, which demonstrated the important role of P450 monooxygenases and/or dioxygenases in the initial degradation of the aliphatic and aromatic fractions in PHCs." As taken from Yanto DH and Tachibana S. 2014. J. Hazard. Mater. 278, 454-463. PubMed, 2014 available at http://www.ncbi.nlm.nih.gov/pubmed/24997261

10.5. All other relevant types of ecotoxicity

Bioaccumulation factor: 1.0-67.0 (estimated). Bioaccumulation is low (US EPA, 2011).

The Ecological Categorization Results from the Canadian Domestic Substances List simply state that petroleum resins are of uncertain bioaccumulative potential in the environment.

Data accessed December 2014 on the OECD website: http://webnet.oecd.org/CCRWeb/Search.aspx

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12. Other information

No data available to us at this time.

13. Last audited

August 2022

SCREENING-LEVEL HAZARD CHARACTERIZATION

Asphalt Category

SPONSORED CHEMICALS

Asphalt	**CASRN 8052-42-4**
Residues (petroleum), vacuum	**CASRN 64741-56-6**
Raffinates (petroleum), residual oil decarbonization	**CASRN 64742-07-0**
Petroleum resins	**CASRN 64742-16-1**
Residues (petroleum), hydrodesulfurized vacuum	**CASRN 64742-85-4**
Asphalt, oxidized	**CASRN 64742-93-4**

SUPPORTING CHEMICALS

Aromatic extracts	**No CASRN**
Lubricating oil basestocks	**No CASRN**
Asphalt (API Sample 81-13)	**No CASRN**
Asphalt (API Sample 81-14)	**No CASRN**
Penetration Asphalt (DMSO Extraction)	**No CASRN**
Asphalt-Based Paints	**No CASRN**
Paving Asphalt Fume Condensate	**No CASRN**
Penetration Asphalt Fume	**No CASRN**
Fume Condensates (Roofing/Paving Asphalts)	**No CASRN**
Paving Asphalt Fume Condensate	**No CASRN**
Roofing Asphalt Fume Condensate	**No CASRN**
Asphalt Fume Extracts	**No CASRN**

The High Production Volume (HPV) Challenge Program[1] was conceived as a voluntary initiative aimed at developing and making publicly available screening-level health and environmental effects information on chemicals manufactured in or imported into the United States in quantities greater than one million pounds per year. In the Challenge Program, producers and importers of HPV chemicals voluntarily sponsored chemicals; sponsorship entailed the identification and initial assessment of the adequacy of existing toxicity data/information, conducting new testing if adequate data did not exist, and making both new and existing data and information available to the public. Each complete data submission contains data on 18 internationally agreed to "SIDS" (Screening Information Data Set[1,2])

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[1] U.S. EPA. High Production Volume (HPV) Challenge Program; http://www.epa.gov/chemrtk/index.htm.
[2] U.S. EPA. HPV Challenge Program – Information Sources; http://www.epa.gov/chemrtk/pubs/general/guidocs.htm.

endpoints that are screening-level indicators of potential hazards (toxicity) for humans or the environment.

The Environmental Protection Agency's Office of Pollution Prevention and Toxics (OPPT) is evaluating the data submitted in the HPV Challenge Program on approximately 1400 sponsored chemicals by developing hazard characterizations (HCs). These HCs consist of an evaluation of the quality and completeness of the data set provided in the Challenge Program submissions. They are not intended to be definitive statements regarding the possibility of unreasonable risk of injury to health or the environment.

The evaluation is performed according to established EPA guidance[2,3] and is based primarily on hazard data provided by sponsors; however, in preparing the hazard characterization, EPA considered its own comments and public comments on the original submission as well as the sponsor's responses to comments and revisions made to the submission. In order to determine whether any new hazard information was developed since the time of the HPV submission, a search of the following databases was made from one year prior to the date of the HPV Challenge submission to the present: (ChemID to locate available data sources including Medline/PubMed, Toxline, HSDB, IRIS, NTP, ATSDR, IARC, EXTOXNET, EPA SRS, etc.), STN/CAS online databases (Registry file for locators, ChemAbs for toxicology data, RTECS, Merck, etc.) and Science Direct. OPPT's focus on these specific sources is based on their being of high quality, highly relevant to hazard characterization, and publicly available.

OPPT does not develop HCs for those HPV chemicals which have already been assessed internationally through the HPV program of the Organization for Economic Cooperation and Development (OECD) and for which Screening Initial Data Set (SIDS) Initial Assessment Reports (SIAR) and SIDS Initial Assessment Profiles (SIAP) are available. These documents are presented in an international forum that involves review and endorsement by governmental authorities around the world. OPPT is an active participant in these meetings and accepts these documents as reliable screening-level hazard assessments.

These hazard characterizations are technical documents intended to inform subsequent decisions and actions by OPPT. Accordingly, the documents are not written with the goal of informing the general public. However, they do provide a vehicle for public access to a concise assessment of the raw technical data on HPV chemicals and provide information previously not readily available to the public.

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[3] U.S. EPA. Risk Assessment Guidelines; http://cfpub.epa.gov/ncea/raf/rafguid.cfm.

Chemical Abstract Service Registry Number (CASRN)	**<u>Sponsored Chemicals</u>** **See Appendix** **<u>Supporting Chemicals</u>** **See Appendix**
Chemical Abstract Index Name	
Structural Formula	

Summary

Asphalt materials are complex hydrocarbon mixtures with molecular weights ranging from approximately 500 to 2,000 and carbon numbers predominantly higher than C25. These complex mixtures are viscous semi-solid to solid materials possessing low to negligible water solubility and moderate to negligible vapor pressure. They are expected to possess low mobility in soil. Volatilization is considered low to high based on estimated Henry's Law constants for representative components of these mixtures; however, the strong tendency to adsorb to soil or sediment is likely to attenuate volatilization for most constituents of these complex mixtures. The rate of hydrolysis is considered negligible for the components of these complex mixtures. The rate of atmospheric photooxidation is considered rapid to moderate; however, most of the components of these mixtures are not expected to exist in the vapor phase in the ambient atmosphere. The components of the asphalt category are expected to possess high (P3) persistence and low (B1) bioaccumulation potential.

Human Health Hazard

The six members of the asphalt category are complex mixtures. Because of how they are used (i.e., roofing and paving applications), they are often heated to allow for easy application. This heating process generates fumes that are considered the hazard of concern for human health. Data for different fume condensates are used to address the human health endpoints.

The acute toxicity of CASRN 64741-56-6 is low for the oral and dermal routes of exposure in rats and rabbits, respectively. Paving asphalt fume condensate (supporting chemical, no CASRN) was tested for acute inhalation toxicity in rats. The highest concentration tested (0.0944 mg/L) showed no mortality; however, its acute inhalation toxicity is inconclusive because it is not known if the maximum achievable concentration was reached. CASRN 64741-56-6 is irritating to rabbit skin and eyes, but is not a dermal sensitizer in guinea pigs.

Dermal 28-day repeated dose studies in rabbits with two different samples of CASRN 64741-56-6 (identified as API Samples 81-13 and 81-14) showed irritation at all dose levels (lowest tested dose of 200 mg/kg/day). Systemic effects were observed at the highest dose of 2000 mg/kg/day (reduced body weight for both; changes in hematology with 81-13 and mortality with 81-14); the NOAEL for systemic toxicity is 1000 mg/kg/day.

Two different repeat dose inhalation studies were performed with asphalt fume condensates; one with paving asphalt fume condensate (supporting chemical, no CASRN) and one with roofing asphalt fume condensate (supporting chemical, no CASRN). The paving asphalt fume study was a 14 week nose-only study in rats in which the following systemic effects were noted at a concentration of 0.149 mg/L/day: reduced body weight and food consumption, changes in clinical chemistry parameters [all in males] and histopathological changes in the nasal and perinasal cavities [males and females]; the NOAEC for systemic toxicity is 0.028 mg/L/day. In the roofing asphalt fume condensate study, rats were exposed via inhalation (nose-only) in a combined repeated-dose/reproductive/developmental toxicity screening test. For the repeated dose portion of the study, lung effects were observed in females at 0.13 mg/L/day and in males at 0.3 mg/L/day. NOAECs for females and males were 0.03 mg/L/day and 0.1 mg/L/day, respectively.

In the combined repeated-dose/reproductive/developmental toxicity study with the roofing asphalt fume condensate via inhalation, there were no effects on reproduction (highest tested concentration of 0.3 mg/L/day). For developmental toxicity, maternal toxicity (lung effects) was evident at 0.1 mg/L/day and there were no developmental effects at any concentration; the NOAEC for maternal toxicity is 0.03 mg/L/day and the NOAEC for developmental toxicity I 0.3 mg/L/day (highest concentration tested).

A number of genetic toxicity studies were reported on both asphalt mixtures and various fume condensates derived from asphalts. Polycyclic aromatic hydrocarbon (PAH) content is important in these tests, and the PAH content can vary by asphalt type (e.g., crude oil source), fume condensate type (e.g., temperature used) and solvent extraction method. Results show that CASRN 8052-42-4 was not mutagenic in bacteria *in vitro*, and CASRN 64741-56-6 was mutagenic in mammalian cells *in vitro* with metabolic activation. Several fume condensates were also mutagenic in bacteria with metabolic activation *in vitro*. An *in vivo* test for chromosomal aberrations with CASRN 64741-56-6 was negative. In other genotoxicity studies with fume condensates, neither a roofing asphalt nor paving asphalt fume condensate induced micronuclei in rats *in vivo*. However, a paving asphalt fume condensate did induce DNA adduct formation in lung, nasal and alveolar epithelium tissues following inhalation studies in rats. Available studies with asphalts show carcinogenicity via dermal exposures (skin tumors). For fumes generated from asphalts, available data also show carcinogenicity via dermal but not inhalation exposure routes. For the latter, crude oil source and temperature used to generate the fumes are important factors.

No data gaps were identified under the HPV Challenge Program.

Hazard to the Environment

Based on the supporting chemicals (CASRN 1120-36-1), aromatic extracts (no CASRN) and lubricating oil basestocks (no CASRN), acute and chronic toxicity of asphalt category members to fish, aquatic invertebrates and aquatic plants are considered to be no effects at saturation (NES) based on no effects observed at the water solubility limit (saturation).

No data gaps were identified under the HPV Challenge Program.

The sponsor, American Petroleum Institute, submitted a Test Plan and Robust Summaries to EPA for the asphalt category on December 15, 2003. EPA posted the submission on the ChemRTK HPV Challenge website on January 20, 2004 (http://www.epa.gov/oppt/chemrtk/pubs/summaries/asphlcat/c14901tc.htm). EPA comments on the original submission were posted to the website on June 1, 2004. Public comments were also received and posted to the website. The sponsor submitted updated/revised documents on August 15, 2006, which were posted to the ChemRTK website on September 7, 2006. Final documents were submitted by the sponsor on August 3, 2009 and posted to the ChemRTK website on January 6, 2011.

Category Justification

The asphalt category consists of six sponsored mixtures representing asphaltic materials (i.e., heavy refinery streams that are derived from the high temperature vacuum distillation of petroleum; see Table 1). Asphalts are complex mixtures that include asphaltenes, resins, aromatic oils and saturated oils; these components consist mostly of paraffinic, naphthenic and aromatic hydrocarbons. Asphalts may also include low levels of 3- to 7-ring polycyclic aromatic compounds (PACs), naphthalenes, substances containing nitrogen, sulfur and oxygen, and metals such as vanadium, nickel and iron; the proportion of these components varies depending upon the source of the crude oil and/or the process used to generate these streams. Asphaltic compounds share several physico-chemical properties, including high molecular weight (e.g., 500 – 2000), high carbon-to-hydrogen ratio with carbon number predominantly greater than C25, high boiling point range (> 400 °C), high viscosity, low solubility, high log K_{ow} (> 10) and negligible vapor pressure.

EPA agrees that grouping these six substances into a single category is appropriate on the basis of similar physicochemical properties and production processes. The high molecular weights and similar hydrocarbon distributions among asphalt category members support the conclusion that the toxicity of asphalts, in general, is not expected to vary significantly across members. Moreover, environmental fate, health effects and ecotoxicity are not expected to vary significantly across category members.

Table 1: Asphalt Category: Sponsored Mixtures		
Name	**CASRN**	**Description**[1]
Asphalt	8052-42-4	A very complex combination of high molecular weight organic compounds containing a relatively high proportion of hydrocarbons >C25 with high carbon-to-hydrogen ratios. Contains small amounts of metals (nickel, iron or vanadium). Obtained as the non-volatile residue from distillation of crude oil or by separation as the raffinate from a residual oil in a deasphalting or decarbonization process.
Residues, petroleum, vacuum	64741-56-6	A complex residue from the vacuum distillation of the residue from atmospheric distillation of crude oil. Consists of hydrocarbons >C34 and boiling above 495°C.
Raffinates, petroleum, residual oil decarbonizatin	64742-07-0	A complex combination of hydrocarbons obtained as the solvent insoluble fraction from C5-C7 solvent decarbonization of a residual oil. Consists predominantly of aromatic hydrocarbons >C34 and boiling above 495°C.
Petroleum resins	64742-16-1	A complex combination of organic compounds, predominantly hydrocarbons, obtained as a fraction of the extract of solvent extraction of residuum. Consists predominantly of high molecular weight compounds with high carbon-to-hydrogen ratios.
Residues, petroleum, hydrodesulfurized vacuum	64742-93-4	A complex combination of hydrocarbons obtained by treating a vacuum residuum with hydrogen in the presence of a catalyst under conditions to remove organic sulfur. Consists of hydrocarbons >C34 and boiling above 495°C.
Asphalt, oxidized	64742-93-4	A complex black solid obtained by blowing air through a heated residuum, or raffinate from a deasphalting process with or without a catalyst. The process is oxidative condensation which increases the molecular weight.

[1] Taken almost verbatim from Appendix 1, p. 50 in 2009 Category Assessment Document submitted by API and found at (http://www.epa.gov/oppt/chemrtk/pubs/summaries/asphlcat/c14901tc.htm).

Justification for Supporting Chemical

Asphalt fume condensate (no CASRN) is a visible airborne condensation product of lower boiling volatile components of petroleum asphalt that may be inhaled or deposited on skin and clothing. When asphalts are heated to facilitate paving or roofing applications, the lighter, more volatile components are distilled into the atmosphere. As these components cool, they condense and form small droplets of liquid (fume), some of which are considered respirable.
In their final submission (2009, posted on the EPA website in January, 2011), the sponsor presented details of the polycyclic aromatic hydrocarbon (PAH) content of the fume atmospheres that were tested. Table 9 in the Appendix identifies the test mixtures used for the human health endpoints and Table 10 in the Appendix provides a listing of the individual PAHs and their concentrations for the fume condensates. EPA agrees that the use of data for asphalt fume condensate to support the human health toxicity endpoints is appropriate.

Aromatic extracts (no CASRN) and lubricating oil basestocks (no CASRN) are petroleum hydrocarbon streams (C15 or higher) that contain similar types of hydrocarbon constituents (aromatic and saturated) as chemicals in the asphalt category and are used as supporting chemicals for the aquatic toxicity endpoints. The chemicals in the asphalt category have lower water solubility than aromatic extracts and lubricating oil basestocks. Therefore, the data submitted by the sponsor on the supporting chemicals are considered a worst-case scenario for evaluating aquatic toxicity. Although no clear compositional information was provided on aromatic extracts and lubricating oil basestocks [see two other HPV categories with these data and for which hazard characterizations will be forthcoming: the aromatic extracts category (http://www.epa.gov/chemrtk/pubs/summaries/aroexcat/c14900tc.htm) and the lubricating oil basestocks category (http://www.epa.gov/chemrtk/pubs/summaries/lubolbse/c14364tc.htm)], based on the premise that these streams contain hydrocarbons mainly C15 or higher, EPA accepts the use of aromatic extracts and lubricating oil basestocks as supporting chemicals for the aquatic toxicity endpoints. In both of these test plans, there are narratives describing the mixtures that are used as supporting chemicals for this hazard characterization. In addition, data for 1-tetradecene (CASRN 1120-36-1), which has been evaluated as an OECD HPV chemical (SIAM 11, http://www.chem.unep.ch/irptc/sids/OECDSIDS/AOalfaolefins.pdf), were used in this assessment in consideration of worst-case scenario for evaluating aquatic toxicity.

1. Chemical Identity

1.1 Identification and Purity

Table 1 above is a listing of the category member mixtures. Because each of the category members is a complex mixture, purity is not a parameter of concern. Representative structures for individual components are provided in the Appendix.

1.2 Physical-Chemical Properties

The physical-chemical properties of the asphalt category are provided in Tables 2 and 3. Asphalt materials are complex hydrocarbon mixtures with molecular weight ranging from approximately 500 to 2,000 and carbon numbers predominantly higher than C25. These complex mixtures are viscous semi-solid to solid materials possessing low to negligible water solubility and moderate to negligible vapor pressure.

Table 2. Physical-Chemical Properties of the Asphalt Category[1]

Property	SPONSORED CHEMICAL Asphalt	SPONSORED CHEMICAL Residues (petroleum), vacuum	SPONSORED CHEMICAL Raffinates (petroleum), residual oil decarbonization	SPONSORED CHEMICAL Petroleum resins	SPONSORED CHEMICAL Residues (petroleum), hydrodesulfurized vacuum	SPONSORED CHEMICAL Asphalt, oxidized
CASRN	8052-42-4	64741-56-6	64742-07-0	64742-16-1	64742-85-4	64742-93-4
Molecular Weight	Complex mixture	Complex mixture	Complex mixture	Complex mixture	Complex mixture	Complex mixture
Physical State	Viscous, semi-solid to solid material	Viscous, semi-solid to solid material	Viscous, semi-solid to solid material	Viscous, semi-solid to solid material	Viscous, semi-solid to solid material	Viscous, semi-solid to solid material
Melting Point	30–60°C (measured softening point); 60–75°C (measured softening point)	No data. 30–60°C (measured softening point)[2]; 60–75°C (measured softening point)[3]	No data. 30–60°C (measured softening point)[2]; 60–75°C (measured softening point)[3]	No data. 30–60°C (measured softening point)[2]; 60–75°C (measured softening point)[3]	No data. 30–60°C (measured softening point)[2]; 60–75°C (measured softening point)[3]	60–130°C (measured softening point)
Boiling Point	>470°C (measured); >550°C (measured)	>495°C (measured)	>495°C (measured)	>482°C (measured)	>495°C (measured)	>400°C (measured)
Vapor Pressure	$<1.0\times10^{-10}$ to 1.6×10^{-5} mm Hg (estimated)[4,5]	$<1.0\times10^{-10}$ to 2.6×10^{-7} mm Hg (estimated)[4,5]	$<1.0\times10^{-10}$ to 2.6×10^{-7} mm Hg (estimated)[4,5]	$<1.0\times10^{-10}$ to 2.6×10^{-7} mm Hg (estimated)[4,5]	$<1.0\times10^{-10}$ to 2.6×10^{-7} mm Hg (estimated)[4,5]	$<1.0\times10^{-10}$ to 2.6×10^{-7} mm Hg (estimated)[4,5]
Dissociation Constant (pK_a)	Not applicable					
Henry's Law Constant	$<1.0\times10^{-10}$ to 494 atm-m^3/mol (estimated)[4,5]	$<1.0\times10^{-10}$ to 4,760 atm-m^3/mol (estimated)[4,5]	$<1.0\times10^{-10}$ to 4,760 atm-m^3/mol (estimated)[4,5]	$<1.0\times10^{-10}$ to 4,760 atm-m^3/mol (estimated)[4,5]	$<1.0\times10^{-10}$ to 4,760 atm-m^3/mol (estimated)[4,5]	$<1.0\times10^{-10}$ to 4,760 atm-m^3/mol (estimated)[4,5]
Water Solubility	$<1.0\times10^{-5}$ mg/L (estimated)[4,5]	$<1.0\times10^{-5}$ mg/L (estimated)[4,5]	$<1.0\times10^{-5}$ mg/L (estimated)[4,5]	$<1.0\times10^{-5}$ mg/L (estimated)[4,5]	$<1.0\times10^{-5}$ mg/L (estimated)[4,5]	$<1.0\times10^{-5}$ mg/L (estimated)[4,5]
Log Kow	>10 (estimated)[4,5]	>10 (estimated)[4,5]	>10 (estimated)[4,5]	>10 (estimated)[4,5]	>10 (estimated)[4,5]	>10 (estimated)[4,5]

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[1] American Petroleum Institute. 2006. Revised Robust Summary and Test Plan for the Asphalt Category. Available online at http://www.epa.gov/chemrtk/pubs/summaries/asphlcat/c14901tc.htm as of December 15, 2010.

[2] Data presented for a penetration grade asphalt.

[3] Data presented for a hard grade asphalt.

[4] Data range presented for representative structures provided in the Appendix.

[5] U.S. EPA. 2010. Estimation Programs Interface Suite™ for Microsoft® Windows, v4.00. U.S. Environmental Protection Agency, Washington, DC, USA. Available online at http://www.epa.gov/opptintr/exposure/pubs/episuitedl.htm as of December 15, 2010.

Table 3. Physical-Chemical Properties of the Asphalt Category (Supporting Chemicals)

Property	SUPPORTING CHEMICAL Aromatic extracts	SUPPORTING CHEMICAL Lubricating oil basestocks	SUPPORTING CHEMICAL Asphalt fume condensate
CASRN	Not applicable	Not applicable	Not applicable
Molecular Weight	Complex mixture	Complex mixture	Complex mixture
Physical State	Highly viscous to mobile liquids[1,2]	Liquid[3,4]	Volatile components of heated asphalt[5]
Melting Point	-6 to 36°C (measured pour point)[1,2]	-60 to -6°C (measured softening point)[3,4]	9.9 to 28.2°C (measured)[6,7]
Boiling Point	250–680°C (measured)[1,2]	300–800°C (measured)[3,4]	270.6–316.3°C (measured)[6,7]
Vapor Pressure	<0.075 mm Hg at 25°C (measured)[1,2]	1.3×10^{-6} mm Hg at 25°C (measured)[3,4]	0.003 to 3.0×10^{-5} mm Hg at 25°C (measured)[6,7]
Dissociation Constant (pK_a)	Not applicable		
Henry's Law Constant	3.4×10^{-5} – 0.012 atm-m^3/mol (estimated)[6,8]	4.0×10^{-5} – 21.9 atm-m^3/mol (estimated)[6,8]	0.019 – 12.6 atm-m^3/mol (estimated)[6,8]
Water Solubility	1.4 to 5.8 mg/L (measured)[1]	$<1 \times 10^{-10}$ to 0.004 mg/L (estimated)[6,8]	7.6×10^{-5} to 0.006 mg/L (measured)[6,7]
Log Kow	8.0 to >10 (estimated)[6,8]	7.3 to >10 (estimated)[6,8]	7.7 to 9.2 (estimated)[6,8]

[1] American Petroleum Institute Petroleum HPV Testing Group. 2009. Revised Test Plan and Robust Summary for Aromatic Extracts. Available online at http://www.epa.gov/chemrtk/pubs/summaries/aroexcat/c14900tc.htm as of December 7, 2010.

[2] Data presented for a distillate aromatic extract, Extracts (Petroleum), Heavy Paraffinic Distillate Solvent (CASRN 64742-04-7).

[3] American Petroleum Institute. 2004. Revised Robust Summary and Test Plan for the Lubricating Oil Basestock Category available online at http://www.epa.gov/chemrtk/pubs/summaries/lubolbse/c14364tc.htm as of December 15, 2010.

4 Data presented for selected lubricating oil basestocks.

[5] American Petroleum Institute. 2006. Revised Robust Summary and Test Plan for the Asphalt Category available online at http://www.epa.gov/chemrtk/pubs/summaries/asphlcat/c14901tc.htm as of December 15, 2010.

[6] Data range presented for representative structures provided in the Appendix.

7 SRC. The Physical Properties Database (PHYSPROP). Syracuse, NY: Syracuse Research Corporation. Available online at http://www.syrres.com/esc/physprop.htm as of December 15, 2010.

8 U.S. EPA. 2010. Estimation Programs Interface Suite™ for Microsoft® Windows, v4.00. U.S. Environmental Protection Agency, Washington, DC, USA. Available online at http://www.epa.gov/opptintr/exposure/pubs/episuitedl.htm as of December 15, 2010.

2. General Information on Exposure

2.1 Production Volume and Use Pattern

The Asphalt category chemicals had an aggregated production and/or import volume in the United States greater than 3 billion 600 million pounds in calendar year 2005.

- CASRN 8052-42-4:	1 billion pounds and greater;
- CASRN 64741-56-6:	1 billion pounds and greater;
- CASRN 64742-07-0:	500 million to < 1 billion pounds
- CASRN 64742-16-1:	100 million to < 500 million pounds
- CASRN 64742-93-4:	1 billion pounds and greater;

CASRN 64742-85-4 was not reported in the 2006 IUR.

CASRN 64742-07-0:
No industrial processing and uses, and commercial and consumer uses were reported for this chemical.

CASRN 8052-42-4:
Non-confidential information in the IUR indicated that the industrial processing and uses for the chemical include petroleum refineries as intermediate; asphalt paving, roofing and saturated materials manufacturing as adhesives and binding agents. Non-confidential commercial and consumer uses of this chemical include transportation products.

CASRN 64741-56-6:
Non-confidential information in the IUR indicated that the industrial processing and uses for the chemical include petroleum refineries as fuels and not readily obtainable (NRO.) Non-confidential commercial and consumer uses of this chemical include adhesives and sealants; and "other."

CASRN 64742-16-1
Non-confidential information in the IUR indicated that the industrial processing and uses for the chemical include other miscellaneous durable goods merchant wholesaler as not readily obtainable (NRO.) Non-confidential commercial and consumer uses of this chemical include not readily obtainable (NRO.)

CASRN 64742-93-4:
Non-confidential information in the IUR indicated that the industrial processing and uses for the chemical include other rubber product manufacturing as intermediates. Non-confidential commercial and consumer uses of this chemical include rubber and plastic products.

2.2 Environmental Exposure and Fate

The environmental fate properties are provided in Tables 4 and 5. The components of the asphalt category are expected to possess low mobility in soil. Standard biodegradation studies are not available for the asphalt category members; however, various microorganisms have been isolated that are able to utilize asphalt as a source of carbon for growth. Strains of Pseudomonas, Chromobacterium, and Bacillus were capable of degrading thin films of asphalt painted on culture flasks. Degradation between 3 and 25% was measured after 1 week of incubation, and one experiment measured 90% after 1 month. These results are unlikely to represent the degradation of asphalt category members under environmental conditions. The low solubility and bioavailability of asphalt is likely to result in these category members being persistent in the environment. The Henry's Law constants for representative structures range from low to high; however, volatilization is expected to be low for most components of the asphalts category with the exception of the lower boiling volatile components of petroleum asphalt. The rate of hydrolysis is expected to be negligible since the substances in this category do not possess functional groups that hydrolyze under environmental conditions. The constituents of the asphalt category are expected to have high persistence (P3) and low (B1) bioaccumulation potential.

Table 4. Environmental Fate Properties of the Asphalt Category[1]

Property	SPONSORED CHEMICAL Asphalt	SPONSORED CHEMICAL Residues (petroleum), vacuum	SPONSORED CHEMICAL Raffinates (petroleum), residual oil decarbonization	SPONSORED CHEMICAL Petroleum resins	SPONSORED CHEMICAL Residues (petroleum), hydrodesulfurized vacuum	SPONSORED CHEMICAL Asphalt, oxidized
CASRN	8052-42-4	64741-56-6	64742-07-0	64742-16-1	64742-85-4	64742-93-4
Photodegradation Half-life	0.7–3.7 hours (estimated)[2,3]	0.7–2.1 hours (estimated)[2,3]	0.7–2.1 hours (estimated)[2,3]	0.7–2.1 hours (estimated)[2,3]	0.7–2.1 hours (estimated)[2,3]	0.7–2.1 hours (estimated)[2,3]
Hydrolysis Half-life	Stable					
Biodegradation	Various microorganisms have been isolated that are able to utilize asphalt as a source of carbon for growth.					
Bioaccumulation Factor	1.0–999.4 (estimated)[2,3]	1.0–67.0 (estimated)[2,3]	1.0–67.0 (estimated)[2,3]	1.0–67.0 (estimated)[2,3]	1.0–67.0 (estimated)[2,3]	1.0–67.0 (estimated)[2,3]
Log K_{oc}	7.2–13.5 (estimated)[2,3]	9.0–13.5 (estimated)[2,3]	9.0–13.5 (estimated)[2,3]	9.0–13.5 (estimated)[2,3]	9.0–13.5 (estimated)[2,3]	9.0–13.5 (estimated)[2,3]
Fugacity (Level III Model)[2,3]						
Air (%)	<0.1–3.9	<0.1–0.8	<0.1–0.8	<0.1–0.8	<0.1–0.8	<0.1–0.8
Water (%)	2.5–87.4	3.9–37.2	3.9–37.2	3.9–37.2	3.9–37.2	3.9–37.2
Soil (%)	4.2–93.8	62.0–93.8	62.0–93.8	62.0–93.8	62.0–93.8	62.0–93.8
Sediment (%)	<0.1–61.3	<0.1–25.1	<0.1–25.1	<0.1–25.1	<0.1–25.1	<0.1–25.1
Persistence[4]	P3 (high)	P3 (high)	P3 (high)	P3 (high)	P3 (high)	P3 (high)
Bioaccumulation[4]	B1 (low)	B1 (low)	B1 (low)	B1 (low)	B1 (low)	B1 (low)

[1] American Petroleum Institute. 2006. Revised Robust Summary and Test Plan for the Asphalt Category available online at http://www.epa.gov/chemrtk/pubs/summaries/asphlcat/c14901tc.htm as of December 15, 2010.

[2] Data range presented for representative structures provided the Appendix

[3] U.S. EPA. 2010. Estimation Programs Interface Suite™ for Microsoft® Windows, v4.00. U.S. Environmental Protection Agency, Washington, DC, USA. Available online from: http://www.epa.gov/opptintr/exposure/pubs/episuitedl.htm as of December 15, 2010.

[4] Federal Register. 1999. Category for persistent, bioaccumulative, and toxic new chemical substances. *Federal Register* 64, Number 213 (November 4, 1999) pp. 60194–60204.

Table 5. Environmental Fate Properties of the Asphalt Category (Supporting Chemicals)			
Property	**SUPPORTING CHEMICAL Aromatic extracts**	**SUPPORTING CHEMICAL Lubricating oil basestocks**	**SUPPORTING CHEMICAL Asphalt fume condensate**
CASRN	Not applicable	Not applicable	Not applicable
Photodegradation Half-life	0.6–1.7 hours (estimated)[2,3]	0.6–7.0 hours (estimated)[2,3]	5.7–7.0 hours (estimated)[2,3]
Hydrolysis Half-life	Stable		
Biodegradation	No data	No data	No data
Bioaccumulation Factor	1.0 to 2.6 ×10^5 (estimated)[2,3]	1.0 to 1.5×10^5 (estimated)[2,3]	4.3×10^4 to 6.3×10^4 (estimated)[2,3]
Log K_{oc}	5.6–13 estimated[2,3]	4.3–13.3(estimated)[2,3]	4.5–5.2 (estimated)[2,3]
Fugacity (Level III Model)[2,3]			
Air (%)	<0.1–0.3	<0.1–6.7	6.9–11.1
Water (%)	6.3–15.4	6.3–63.5	72.4–82.2
Soil (%)	58.2–93.7	1.3–93.7	5.3–5.6
Sediment (%)	<0.1–26.2	<0.1–31.8	5.6–10.9
Persistence[4]	P2 (moderate) to P3 (high)	P2 (moderate) to P3 (high)	P2 (moderate) to P3 (high)
Bioaccumulation[4]	B1 (low) to B3 (high)	B1 (low) to B3 (high)	B3 (high)

[1] American Petroleum Institute Petroleum HPV Testing Group. Revised Test Plan and Robust Summary for Aromatic Extracts. January 14, 2009. Available online at http://www.epa.gov/chemrtk/pubs/summaries/aroexcat/c14900tc.htm as of December 7, 2010.

[2] Data range presented for representative structures provided the Appendix

[3] U.S. EPA. 2010. Estimation Programs Interface Suite™ for Microsoft® Windows, v4.00. U.S. Environmental Protection Agency, Washington, DC, USA. Available online from: http://www.epa.gov/opptintr/exposure/pubs/episuitedl.htm as of December 15, 2010.

4 Federal Register. 1999. Category for persistent, bioaccumulative, and toxic new chemical substances. *Federal Register* 64, Number 213 (November 4, 1999) pp. 60194–60204.

3. Human Health Hazard

A summary of health effects data submitted for SIDS endpoints is provided in Table 7. The table also indicates where data for tested category members are read-across (RA) to untested members of the category.

Acute Oral Toxicity

Residues (petroleum), vacuum (CASRN 64741-56-6)
Sprague-Dawley rats (5/sex/dose) were administered residues (petroleum), vacuum (API sample 81-13) in corn oil via gavage at 5000 mg/kg and observed for 14 days. No mortality was observed.
LD_{50} > 5000 mg/kg

Acute Inhalation Toxicity

Paving Asphalt fume condensate (No CASRN, supporting chemical)
Wistar rats (5/sex/dose) were exposed via nose-only inhalation to paving asphalt fume condensate at 0.0944 mg/L (measured as total hydrocarbons, or THC[4]) for 4.5 hours and observed for 14 days. No mortality was observed.
LC_{50} > 0.0944 mg/L

Acute Dermal Toxicity

Residues (petroleum), vacuum (CASRN 64741-56-6)
New Zealand White rabbits (4/sex) were administered residues (petroleum), vacuum (API sample 81-13) via the dermal route at 2000 mg/kg-bw to either intact (2/sex) or abraded (2/sex) skin under occlusive conditions for 24 hours and observed for 14 days. No mortalities or visible lesions were reported.
LD_{50} > 2000 mg/kg

Repeated-Dose Toxicity

Members of the asphalt category are heavy liquids or solids that are heated during their use (i.e., for the application of asphalt to either roads or roofs). What is of most toxicological concern is the formation of fumes from heating and the potential for dermal and inhalation hazard to workers applying the asphalt. Thus, tests using some heated form of different asphalts have been

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[4] In their original submission, the sponsor provided data on the single acute inhalation study in rats using asphalt condensate at a target dose of 100 mg/m^3 (0.100 mg/L THC). EPA noted in its 2004 comments that the sponsor needed to indicate whether the administered concentration was close to the maximum achievable concentration. In the cover letter with their 2006 revised submission, the sponsor indicated that the study from which the data were derived (Fraunhofer 2000) did not determine the maximum achievable concentration. The authors of the study report stated that the target concentration of 100 mg/m^3 was five times the current German occupational exposure limit for indoor working with hot bitumen (asphalt).

done and the PAH content of these fume condensates are important. Table 8 in the Appendix provides a list of the PAHs measured in the various test mixtures used below.

Residues (petroleum), vacuum (CASRN 64741-56-6)
(1) New Zealand White rabbits (5/sex/dose) were administered residues (petroleum), vacuum (API sample 81-13) via the dermal route at 0, 200, 1000 or 2000 mg/kg-bw/day to clipped dorsal skin under occluded conditions for 6 hours/day, 3 days/week for 4 weeks. Two animals died and two were sacrificed moribund during the study – the identity of the dose groups for these mortalities were not reported in the robust summary; however the full report is available in TSCATS (OTS 0000186-1) and shows that one control female and one high dose male were found dead, and one control male and a mid-dose female were sacrificed during the study. This supports the conclusion that these deaths were not likely treatment-related. Treatment-related clinical signs observed in survivors included thin appearance, decreased food intake, flaking skin and wheezing (doses not stated). All animals treated with residues (petroleum), vacuum exhibited slight edema. Decreased body weight gain was observed in males at 2000 mg/kg-bw/day. There were no treatment-related changes in the hematology parameters. Alkaline phosphatase was reduced by 50% in males at 2000 mg/kg-bw/day. Changes in absolute and/or relative organ weights were observed at 2000 mg/kg-bw/day (adrenal, kidney, pituitary and spleen), but were not considered to be treatment-related. Treatment-related gross necropsy and microscopic findings were confined to the skin. The skin of females appeared to be more severely affected. Effects in females were limited to the point of contact with the test substance. Incidental findings were observed and were consistent with *Encephalitozoon* infection.
LOAEL (systemic) = 2000 mg/kg-bw/day (based on decreased body weight gain and reduced alkaline phosphatase in males)
NOAEL (systemic) = 1000 mg/kg-bw/day
LOAEL (local) = 200 mg/kg-bw/day (lowest tested dose) (based on irritation)
NOAEL (local) = Not established

(2) New Zealand White rabbits (5/sex/dose) were administered residues (petroleum), vacuum (API sample 81-14) via the dermal route at 0, 200, 1000 or 2000 mg/kg-bw/day using the same methodology described above for API sample 81-3. Details of this study were not presented in the robust summary, but the full report is available in TSCATS (OTS 0000186-1). Two animals died during the study (low dose male and high dose female), and the deaths were considered treatment-related due to clinical signs observed prior to death being consistent with clinical signs observed in survivors. Treatment-related clinical signs included thin appearance, decreased food intake, flaking skin and wheezing at ≥ 200 mg/kg-bw/day. Reduced body weight was observed in females at 2000 mg/kg-bw/day and considered treatment-related. Changes in hematological, clinical chemistry, and organ weights were observed, but not in a dose-related manner and so were not considered treatment-related (limited information supporting this was presented in the TSCATS report, but associated tables and appendices with the raw data were not provided). Edema was observed at ≥ 200 mg/kg-bw/day and erythema was observed at ≥ 1000 mg/kg-bw/day. Histopathological observations were confined to the skin and consisted of subacute acanthotic dermatitis and hyperkeratosis at 2000 mg/kg-bw/day. Incidental findings associated with *Encephalitozoon* infection included meningoencephalitis, nephritis and periportal lymphoid infiltrates.

LOAEL (systemic) = 2000 mg/kg-bw/day (based on reduced body weight in females and mortality)
NOAEL (systemic) = 1000 mg/kg-bw/day
LOAEL (local) = 200 mg/kg/day (lowest tested dose) (based on irritation)
NOAEL (local) = Not established

Paving Asphalt fume condensate (supporting chemical; derived from CASRN 8052-42-4)
Wistar rats (16/sex/dose) were exposed nose-only to paving asphalt fume condensate at nominal concentrations of 0.004, 0.02, and 0.1 mg/L for 6 hours/day, 5 days/week for 14 weeks. Measured concentrations were 0.006, 0.028 and 0.149 mg/L. Bronchoalveolar lavage (BAL) was performed on 6 of the 16 rats per group to examine several cellular and biochemical indicators of lung damage including differential cell count, total protein, and cellular enzyme levels. In addition, cell proliferation was examined in terminal bronchioles and lung parenchyma of select animals during necropsy. This was achieved by infusing 5 animals per group with 5-bromo-2'-deoxyuridine (BrdU) following the 90-day treatment period. No treatment-related mortality occurred (one mortality was reported in the robust summary, but there was no details on whether it was a control or treated animal). No clinical signs of toxicity were observed. Reduced body weights were noted in females at ≥ 0.006 mg/L (all about 5% less, significance not stated) and in males at 0.149 mg/L (10% lower than controls and considered treatment-related, but statistical significance not stated). Reduced food consumption was also observed in males at 0.149 mg/L. No toxicologically relevant findings were reported for the hematological parameters measured. Changes in clinical chemistry were seen only in males and included increased urea and potassium at 0.149 mg/L, and a decrease in calcium at ≥ 0.028 mg/L. No treatment-related differences in urinalysis data were reported. The BAL revealed increased lactic dehydrogenase at 0.149 mg/L in males and increased cell concentration at 0.149 mg/L in females, which is indicative of lung damage. No treatment-related effects were reported upon necropsy for organ weights or gross abnormalities. Changes noted in the nasal and paranasal cavities of test animals at 0.149 mg/L include very slight to moderate eosinophilic cytoplasmic inclusions in epithelial cells and focal/multifocal mucous cell hyperplasia, as well as very slight to slight multifocal mucosal inflammatory cell infiltration. A marked increase of multifocal very slight to slight tubular basophilia was noted in the kidneys of males exposed to 0.149 mg/L, but was not found to be statistically significant. No other treatment-related histological changes were noted.
LOAEC (systemic) = 0.149 mg/L/day (based on reduced body weight and histopathological changes in the nasal and perinasal cavities)
NOAEC (systemic) = 0.028 mg/L/day

Roofing Asphalt fume condensate (supporting chemical; derived from CASRN 64742-93-4)
In a combined repeated-dose/reproductive/developmental toxicity study [OECD TG 422] Wistar rats (12/sex/dose) were exposed via inhalation (nose-only) to roofing asphalt fume condensate to target concentrations of 0, 0.03, 0.10 and 0.30 mg/L total hydrocarbons (actual concentrations were 0.03, 0.10 and 0.297 mg/L). Animals were acclimated for three weeks prior to treatment to acquaint them with the nose-only apparatus. Also, male animals were appropriately randomized to test groups but females were not (due to an error in the randomization program, females were assigned by body weight so that at study start there was a statistically significant difference in body weight between control and treated animals (controls were lower than treated groups)). Males were exposed to control and test atmospheres for six hours/day, seven days/week for 28

days, and females were exposed for 35-48 days (14 days pre-mating, up to 14 days during mating and for 20 days during gestation; with some animals receiving less based on the date of conception). There were no exposures to females after gestation day 20 or during the four-day post-partum lactation period. Females for which there was no evidence of copulation were dosed for 54 days. The robust summary is ambiguous about the number of females treated since there appear to be two groups – one pregnant and one not pregnant (termed the "breeding" and "subchronic" female groups, respectively).

In this section, only systemic effect endpoints will be discussed. See *Reproductive and Developmental Toxicity* below for a discussion of those endpoints. Body weights, clinical signs, food consumption, clinical chemistry, hematology, certain neurobehavioral evaluations (locomotor and functional observational battery), organ weights and pathology and sperm evaluations were all performed on parental animals.

There were no deaths and no adverse clinical signs observed during the study. Body weight gain and food consumption were significantly reduced in high concentration males. There was no effects on female body weight; however as stated above, females were inappropriately randomized by body weight. There were no treatment-related changes in clinical chemistry or hematology parameters (details not provided in robust summary). The following statistically significant organ weight changes were observed: increases in absolute and relative lung weights (high concentration males; mid and high concentration "subchronic females[5]" and high concentration "breeding females"); increases in absolute lung weights only (low concentration "subchronic females" and mid concentration "breeding females"); and increases in absolute liver weight (high concentration "subchronic females"; although non-significant increases were noted in low and mid concentration "subchronic females"). There were no pathological lesions in the liver or any other organs except for the lung and nasal cavity. Lung effects observed included a slight increase in alveolar macrophage accumulation in conjunction with minimal mononuclear/inflammatory cell infiltration. Also, there was a minimal to slight increase in alveolar hyperplasia in the bronchioles (considered adaptive). These lung effects were seen only in high concentration animals. There was a statistically significant decrease of inflammatory cell infiltration in high concentration "subchronic females". Also, an increase in inflammatory cell infiltration was seen in the nasal cavity of high concentration males. There were no adverse effects noted for any group in the neurobehavioral tests that were performed.
LOAEC (systemic, males) = 0.3 mg/L/day (based on decreased body weight gain, increased absolute and relative lung weight, and histopathology in the lungs)
NOAEC (systemic, males) = 0.1 mg/L/day
LOAEC (systemic, females) = 0.1 mg/L/day (based on increased absolute and relative lung weight)
NOAEC (systemic, females) = 0.03 mg/L/day

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[5] As noted earlier, the robust summary is not clear about the number of females in the "subchronic" satellite group. Therefore, any note of statistical significance should be considered with caution.

Reproductive and Developmental Toxicity

Roofing Asphalt fume condensate (supporting chemical; derived from CASRN 64742-93-4)
Wistar rats (12/sex/dose) were exposed via inhalation (nose-only) to roofing asphalt fume condensate to target concentrations of 0, 0.03, 0.10 and 0.30 mg/L total hydrocarbons (actual concentrations were 0.03, 0.10 and 0.297 mg/L) in a combined repeated-dose/reproductive/developmental toxicity study following the OECD 422 protocol that has already been summarized above. On post-partum day 4, all lactating females and offspring were sacrificed. In addition to reproductive organs evaluated for the repeated-dose portion of the study (along with sperm evaluations), the following reproductive parameters were evaluated: pregnancy incidence, number of pregnant animals delivering, mating index, fertility index, gestation length, number of implantation sites, number of corpora lutea. There were no treatment-related affects on any reproductive toxicity parameter. Although there were no statistically significant differences observed in any of the sperm parameters assessed, there was a dose-dependent trend (not statistically significant) in a decrease in sperm number (38.125, 32.250, 27.125, and 24.542 million sperm/epididymis for the control, low, mid and high concentrations, respectively). There were no effects observed in developing animals (no differences in pup sex ratio, pup body weight, pup body weight gain, and no apparent, gross malformations).
NOAEC (reproductive) > 0.3 mg/L/day (highest tested concentration)
LOAEC (maternal) = 0.1 mg/L/day (based on lung effects in females; see Repeated-Dose Toxicity section for description)
NOAEC (maternal) = 0.03 mg/L/day
NOAEC (developmental) > 0.3 mg/L/day (highest tested concentration)

Genetic Toxicity

A number of genetic toxicity studies were reported on both asphalt mixtures and various fume condensates derived from asphalts. Table 6 below provides an overview of the studies; some of which are not summarized in detail either in the robust summaries submitted by the sponsor and/or in this hazard characterization (reasons provided in footnotes in the table). PAH content is important in these tests, and Table 10 in the Appendix provides these values for each substance used in the different tests. The PAH content can vary by asphalt type (e.g., crude oil source), fume condensate type (e.g., temperature used) and solvent extraction method. In addition, the values presented in Table 10 are in different units and care should be taken in comparing values across mixtures.

Table 6. Genetic Toxicity Tests Performed for the Asphalt Category*

Type	Test Substance Name	In Vitro Gene Mutation (Bacteria)[1]	In vitro gene mutation (mammalian)	In vivo chromosome [2]	In vivo DNA effects (DNA adduct formation)
Asphalt (aka Bitumen)	Penetration Asphalt (DMSO extraction) pp28-30	X[3]			
	Asphalt Paint Samples pp 31-32	X			
	API Sample 81-13 (p 35, 38)		X	X	
	API Sample 81-14 (p 37, 39)		X	X	
Fume (Asphalts that have been heated)	Penetration Asphalt fume sample (ether extract) pp 28-30	X			
	Penetration Asphalt fume sample (acetone extract) pp 28-30	X			
	Fume condensates of coal tar pitches, roofing asphalts and paving asphalts pp32-34	X[4]			
	Fume condensate ("Paving 2007")*			X	X
	Roofing asphalt fume condensate*			X	
	NTP Fume condensate studies	X[5]			

* Page numbers refer to pages in the robust summaries from the 2009 submission. The exceptions are the 2007 studies (paving fume condensate and roofing asphalt fume condensate) which were included at the end of the submission and are printouts from HPVIS and do not have page numbers.

[1] In addition to the studies in this column, references were provided for other Ames tests (see p. 35 in 2009 robust summaries). These studies were identified in the robust summary, but not summarized either in the robust summary or in this hazard characterization (Reinke et al., 2000; DeMeo et al., 1998; Blackburn and Kriech, 1990; and Pasquini, 1989).

[2] Studies summarized on pp. 40-43 in the 2009 robust summaries are not included here because they used intratracheal instillation as the route of exposure in in vivo micronucleus assays. This route of exposure is not considered relevant for this hazard characterization.

[3] Only two *Salmonella* strains were used (instead of the required four).

[4] Only one *Salmonella* strain was used (instead of the required four). These results are not presented in Table 7.

[5] Studies performed by the NTP and not in the sponsor-submitted submission. See text for details and link to data tables.

Genetic Toxicity – Gene Mutation

In vitro

Asphalt (penetration, CASRN 8052-42-4; and asphalt fume condensates [derived from the penetration asphalt, CASRN 8052-42-4])

Salmonella typhimurium strains TA98 and TA100 were exposed to asphalt ("penetration bitumen", three separate samples) or asphalt fume condensate (two separate samples); both with and without metabolic activation. The asphalt samples were first dissolved in benzene and treated with n-heptane to precipitate asphaltenes; the soluble substances were then extracted with DMSO. These DMSO extracts were used in the test at four doses between 0.1 mg/plate up to 65 mg/plate. Asphalt fume condensates were collected from actual paving operations and were subjected to either ether or acetone extractions and then dissolved in DMSO. These two separate extracts were evaluated in the test at four doses (from 0.1 to 12.5 mg/plate for the ether extract and from 0.05 to 60 mg/plate for the acetone extracts). Positive and negative controls were used and yielded the expected results. Cytotoxicity data were not provided. Neither asphalt (unspecified composition) nor asphalt fume condensate increased the mutation frequency in *Salmonella typhimurium* strains TA98 and TA100 with or without activation.
Asphalt and asphalt fume condensate were not mutagenic in this assay.

Asphalt (CASRN 8052-42-4)
Salmonella typhimurium strains TA98, TA100, TA1535, TA1537 and TA1538 were exposed to asphalt paint at concentrations of 0.005, 0.01, 0.1, 1, 5 and 10 µL/plate with and without metabolic activation. Asphalt was first cut back to 64% solid with mineral spirits, then a small amount of xylene was added to create the asphalt paint samples. Positive and negative controls were used, but their responses were not provided in the study summary. Cytotoxicity was not observed at any of the concentrations tested. Asphalt was not mutagenic with or without activation for any of the *Salmonella typhimurium* strains tested.
Asphalt was not mutagenic in this assay.

Asphalt fume condensate (supporting chemical; derivation assumed to be from CASRN 64742-93-4)
Salmonella typhimurium strain TA98 was exposed to asphalt fume condensate extracted in DMSO from three different types of mixtures: coal tar pitches (two samples), roofing asphalts (two samples) and paving asphalts (18 samples). The paving asphalts samples came from 14 different crude oil sources and different process conditions. Although coal tar condensate is not relevant to asphalts, it is important in this study which tested the correlation of PAH type and content to mutagenicity. All fumes were generated in the laboratory by heating 10 kg of each sample and trapping the condensates. The oil phase of the condensates were extracted with DMSO and used in the tests. The PAH content reported in Table XXX in the Appendix was obtained from a publication (Machado et al., 1993) since it was not reported in the robust summary (pp. 32-34 of 2009 robust summary submission). Positive (an ASTM residual fuel oil) and negative controls were used and stated to "yield(ed) the expected results"; however, neither the robust summary nor the publication provided the data. Results showed that both coal tar pitch fume condensates were strongly mutagenic; and the roofing and paving asphalt samples were approximately 100X less mutagenic. The data presented in both the robust summary and the publication show results in terms of a mutagenicity index (MI; which is the number of revertants per microliter of DMSO extract). Neither positive nor negative control MIs are presented. Cytotoxicity data were not provided. The assay was positive for mutagenicity.
Asphalt fume condensate was mutagenic in this assay.

Residues (petroleum), vacuum (CASRN 64741-56-6)

In two studies, mouse lymphoma cells (L5178Y TK+/-) were exposed to residues (petroleum), vacuum (API samples 81-13 or 81-14) at concentrations ranging from 62.5 to 1000 nL/mL with and without metabolic activation. Positive and negative controls were tested concurrently and yielded expected results (data provided in robust summary). In both studies, residues (petroleum), vacuum was not mutagenic without activation, but was weakly mutagenic with metabolic activation (TSCATS: OTS0000175-1).
Residues (petroleum), vacuum was mutagenic in this assay (only under metabolic activation conditions).

Asphalt Fume Condensates (No CASRN, supporting chemical)

The National Toxicology Program (NTP) provides results and data tables for seven different genotoxicity tests with various strains of *Salmonella typhimurium*. All tests were conducted with asphalt fume extracts and results showed that 4/7 were positive (with metabolic activation only); two were considered weakly positive (again, with metabolic activation only) and one was negative. Unfortunately, the information on the website does not describe how the test materials were derived; nor does it provide any details beyond the data tables. Following are the results:

Asphalt fume extract, neat, EMTDP-79 → positive (activation only)
Asphalt fume extract, Fraction A, EMTDP-80 → weak positive (activation only)
Asphalt fume extract, Fraction B, EMTDP-81 → positive (activation only)
Asphalt fume extract, Fraction C, EMTDP-82 → positive (activation only)
Asphalt fume extract, Fraction D, EMTDP-83 → weak positive (activation only)
Asphalt fume extract, Fraction E, EMTDP-84 → negative (with & w/o activation)
Asphalt fume extract, Fraction A-E, EMTDP-85 → positive (activation only)

All the data are available at the following URL: http://ntp-apps.niehs.nih.gov/ntp_tox/index.cfm?fuseaction=ntpsearch.searchresults&searchterm=asphalt&crumbspot=2

Genetic Toxicity – Chromosomal Aberrations

In vivo

Residues (petroleum), vacuum (CASRN 64741-56-6)

In two bone marrow cytogenetic studies, Sprague-Dawley rats (10/sex/dose) were administered residues (petroleum), vacuum (API samples 81-13 and 81-14) as solutions in corn oil via gavage for 5 days at 300, 1000 or 3000 mg/kg-bw/day for API 81-13 or 400, 1300 or 4000 mg/kg-bw/day for API 81-14. Positive and negative controls were tested concurrently and yielded expected results. No increase in the number of chromosomal aberrations was found in rats treated with either test substance, compared to the negative controls. Sialodacryoadenitis (SDAV) infections were observed in several rats upon necropsy, but SDAV is common among rats and is not believed to have influenced the results.
Residues (petroleum), vacuum did not induce chromosomal aberrations in this assay.

Roofing Asphalt fume condensate (supporting chemical; derived from CASRN 64742-93-4)
Wistar rats (12/sex/dose) were exposed via inhalation (nose-only) to roofing asphalt fume condensate to target concentrations of 0, 0.03, 0.10 and 0.30 mg/L total hydrocarbons (actual concentrations were 0.03, 0.10 and 0.297 mg/L) in a combined repeated-dose/reproductive/developmental toxicity study following the OECD 422 protocol. Five rats/sex/group were used to evaluate the induction of micronuclei in bone marrow erthryocytes. A positive control group (cyclophosphamide) was also used. One femur from each rat was collected 24 hours after the last day of 28 days of exposure to the test material. Results showed the positive control responded appropriately and there was no induction of micronuclei in any treated group (all data presented in robust summary).
Roofing asphalt fume condensate did not induce micronuclei in rat bone marrow erythrocytes.

Paving Asphalt fume condensate (supporting chemical; derived from CASRN 64742-93-4)
Some rats from the two year cancer bioassay described below (Fuhst et al., 2007) were evaluated for chromosome damage via a micronucleus assay (the results of which are reported separately in Halter et al., 2007). Wistar rats (6/sex/dose/exposure period for this portion of the study) were exposed via inhalation (nose-only) to roofing asphalt fume condensate to target concentrations of 0, 0.004, 0.020 and 0.100 mg/L total hydrocarbon concentration (THC). Peripheral blood was taken from the tail vein at 5 days, 20 days and 12 months of exposure and analyzed for micronuclei in erythrocytes. These animals were sacrificed at 12 months and the bone marrow was also extracted and analyzed for micronuclei formation. There were no positive controls. Results showed no increases in micronuclei formation in peripheral blood after 5 days, 20 days, or 12 months of exposure; and no increases in micronuclei formation in bone marrow after 12 months.
Paving asphalt fume condensate did not induce micronuclei formation in peripheral blood or bone marrow erythrocytes in this assay

Genetic Toxicity – Other

In vivo

Paving Asphalt fume condensate (supporting chemical; derived from CASRN 64742-93-4)
Some rats from the two year cancer bioassay described below (Fuhst et al., 2007) were evaluated for DNA adduct formation (results of which are reported separately in Halter et al., 2007). Wistar rats (8/sex/dose/exposure period for this portion of the study) were exposed via inhalation (nose-only) to roofing asphalt fume condensate to target concentrations of 0, 0.004, 0.020 and 0.100 mg/L THC. DNA from the lung, nasal and alveolar epithelium was collected from rats at 5 days, 30 days and 12 months. Results showed an increase in 3-4 stable DNA adducts in these tissues over the endogenous adducts seen in the clean air control animals.
Paving asphalt fume condensate did induce DNA adduct formation in lung, nasal and alveolar epithelium in this assay.

Additional Information

Skin Irritation

Residues (petroleum), vacuum (CASRN 64741-56-6)

In two studies, six male New Zealand White rabbits were exposed to 0.5 mL of undiluted residues (petroleum), vacuum (API samples 81-13 and 81-14) on abraded or intact skin under occluded conditions for 24 hours and observed for 14 days. No visible lesions were observed at necropsy. Residues (petroleum), vacuum was slightly irritating to the skin; exposed rabbits received a primary dermal irritation index of 0.2 (for API 81-13) to 0.4 (for API 81-14). The sponsor submitted only the study for API-81-13; the study summary/results for API sample 81-14 was found in TSCATS (OTS 0000171-00).
Residues (petroleum), vacuum was slightly irritating to rabbit skin in this study.

Eye Irritation

Residues (petroleum), vacuum (CASRN 64741-56-6)

Rabbits were exposed to 0.1 mL of undiluted residues (petroleum), vacuum (API samples 81-13) into one eye of each of nine animals (sex and strain not specified). After 30 seconds, treated eyes from each of three rabbits were washed for 1 minute, while the treated eyes of the other six rabbits remained unwashed. Observations were made for up to 7 days. No abnormalities were reported and body weights were normal. Mild to moderate eye irritation was observed in both washed and unwashed eyes for both API samples. Study details for API 81-14 are available from TSCATS (OTS 0000171-1). The sponsor provided a robust summary for API 81-13.
Residues (petroleum), vacuum) was slightly to moderately irritating to rabbit eyes in this study.

Sensitization

Residues (petroleum), vacuum (CASRN 64741-56-6)

In two related studies, guinea pigs (10 males, strain not specified) were treated with 0.4 mL of undiluted residues (petroleum), vacuum (API samples 81-13 and 81-14) under an occlusive dressing to shorn skin once/week for 3 weeks. The dressing was in place for six hours each time. Two weeks after the last dose the challenge dose was applied to a virgin skin site on the opposite flank of the test animal. As with the induction dose, the skin was clipped, the test material applied and an occlusive dressing was used for six hours. Evaluations of skin reactions were made 24 and 48 hours later. Positive and negative controls were tested concurrently and responded appropriately. No skin reactions were observed in animals exposed to residues (petroleum), vacuum. (NOTE: The sponsor provided a robust summary for the API 81-13 study only, and a reference to the API 81-14 study was listed. The API 81-14 study is available in TSCATS (OTS0000186-2).
Residues (petroleum), vacuum was not a dermal sensitizer in guinea pigs in this study.

Carcinogenicity

The sponsor provided a robust summary consisting of very general information on the wealth of cancer information available on asphalts and their fumes (p. 43 in 2009 robust summary document). The sponsor included a two page table summarizing 19 studies (pp.50-51 in the same document). However, the list does not contain sufficient information to make determinations of cancer for this hazard characterization. Also, in the HPVIS portion of the submitted robust summaries (again, no page numbers listed), there is a recent two-year cancer bioassay that was performed and published in the open literature that is summarized below (Fuhst et al., 2007). Finally, the Agency identified a recent review on the potential carcinogenicity of asphalts (Schreiner et al., in press) which will not be summarized in this hazard characterization, but is offered as a recent reference for interested readers.

As noted in the "general" robust summary, there are dermal (i.e., skin painting) studies with asphalts, extracts of asphalts and asphalt fume condensates as well as inhalation studies with fume condensates. As stated earlier in the genetic toxicity section, the PAH content of generated fumes depend on a number of factors, including the source of the petroleum, heating temperatures and solvent used for extraction. This is important because the PAH content – and concentration – are thought to be related to mutagenicity and possible carcinogenicity.

TSCATS does contain more detailed information on some of the studies cited and this information is briefly summarized below. In addition, the International Agency for Research on Cancer (IARC) is planning to review asphalts and their fumes (identified as "bitumen and bitumen fumes") in October, 2011 (http://monographs.iarc.fr/ENG/Meetings/index.php).

Summary of Fuhst et al., 2007 (taken from both the submitted HPVIS robust summary and the publication)

Paving Asphalt fume condensate (supporting chemical; derived from CASRN 64742-93-4)
Fume condensate was collected from an operating asphalt mixing plant. Specifically, the condensate was collected from a storage tank heated to 175^{o} C (to mimic road paving heating processes). Wistar rats (50/sex/group; with an additional 36/sex/group for control and high dose for bronchio-alveloar lavage (BAL) at various times over the two-year period were exposed via nose-only inhalation to the fumes at the target concentrations of 0, 0.004, 0.02, and 0.10 mg/L (actual concentrations of 0, 0.0068, 0.034 and 0.173 mg/L total hydrocarbon concentration, or THC). According to both the robust summary (HPVIS document in 2009 submission) and Fuhst et al (2007), there were no statistically significant differences in tumor types between control and treated animal or in the number of tumor-bearing animals. There was one male with an adenocarcinoma of the nasal cavity (high concentration group) that may have been treatment related. Full data tables and discussion are in Fuhst et al. The following non-carcinogenic effects were observed (all taken from Fuhst et al.): 1) statistically significant decreases in body weight in males and females at the mid and high concentrations; 2) slight evidence of inflammatory effects in the lung (evaluated in high concentration group only via BAL); and 3) irritant effects (histological) in the nasal cavity (dose-related degenerative, inflammatory and proliferative lesions; the latter being basal cell hyperplasia) and lung (dose-related bronchio-alveolar hyperplasia).

Summaries of Studies in Table on pp. 50-51 in Robust Summary [with added details provided from TSCATS (http://www.srcinc.com/what-we-do/databaseforms.aspx?id=384) information, where available]

The 19 studies that are presented in the table in the robust summaries provide little information that is useful. The sponsor summary on p. 43 provides the following general statements assumed to be based on the data from the 19 studies: 1) whole asphalts appear to be carcinogenic in dermal studies when organic solvents are used as solvents but not when applied undiluted; and 2) fume condensates are also carcinogenic in dermal studies in animals – again with the use of organic solvents – but not in inhalation studies.

EPA did not retrieve the 19 original articles to review the carcinogenicity of asphalts for this screening-level hazard characterization.

Residues (petroleum), vacuum (CASRN 64741-56-6)
C3H/HeJ mice (25/sex/dose) were exposed to 50 µL of residues (petroleum), vacuum (API samples 81-13 and 81-14) via the dermal route as a 50% (w/w) solution in toluene via the dermal route twice per week for 12 months. This was part of a large study in which 1700 mice were used to evaluate 12 different petroleum refinery streams. For API samples 81-13 and 81-14, there were no increases in tumors observed. Study details are available from TSCATS (OTS 0000426-8 and OTS 0000298-4; the former presenting the cancer evaluation and the latter presenting the chronic toxicity evaluation). Assumed to represent the study identified in the robust summary table as Vacuum Residuum, API 1989
Residues (petroleum), vacuum was not carcinogenic to mice in this study.

Asphalt (CASRN 8052-42-4) and Residues (petroleum), vacuum (CASRN 64741-56-6)
C3H/HeN mice (50 males) were exposed to 80 µL of a mixture of asphalt (CASRN 8052-42-4) which was “back-blended” vacuum residual bottoms (CASRN 64741-56-6) via the dermal route as a 50% (w/v) solution in cyclohexane to clipped skin twice per week for the lifetime of the animals (≤ 97 weeks). Squamous cell carcinomas and papillomas were observed (8 observed in the treated group and none in the sham or vehicle control groups; there was one fibrosarcoma observed in the vehicle control group). Non-neoplastic skin lesions observed microscopically were believed to have been induced by the cyclohexane vehicle and not by the test chemical mixture. Study details are available from TSCATS (OTS 0533996). It is not clear whether this study is in the table submitted by the sponsor.
A mixture of asphalt and residues (petroleum), vacuum was carcinogenic to mice in this study.

Asphalt fume condensate (no CASRN, supporting chemical)
Two different strains of male mice (C3H and CD-1) were evaluated in a dermal cancer study in which two different asphalt samples (and two different coal tar pitch samples – not mentioned further in this summary) were used under several conditions: with and without simulated sunlight exposures and using two different heating temperatures to generate the fume condensates. Fifty males per group were exposed to asphalt fume condensate via dermal application twice per week as a 50% solution in cyclohexane/acetone for 72 – 104 weeks. The positive control group was treated with benzo(a)pyrene. Greater tumor response was observed

from fumes generated at higher temperatures. C3H mice were more sensitive to the test substance than CD-1 mice. Additional study details are available from TSCATS (OTS 0539444) for the study using C3H and CD-1 mice; assumed to represent the study identified in the robust summary table as Niemeier, 1988.
Asphalt fume condensate was carcinogenic to mice in these studies.

Conclusion: The six members of the asphalt category are complex mixtures. Because of how they are used (i.e., roofing and paving applications), they are often heated to allow for easy application. This heating process generates fumes that are considered the hazard of concern for human health. Data for different fume condensates are used to address the human health endpoints.

The acute toxicity of CASRN 64741-56-6 is low for the oral and dermal routes of exposure in rats and rabbits, respectively. Paving asphalt fume condensate (supporting chemical, no CASRN) was tested for acute inhalation toxicity in rats. The highest concentration tested (0.0944 mg/L) showed no mortality; however, its acute inhalation toxicity is inconclusive because it is not known if the maximum achievable concentration was reached. CASRN 64741-56-6 is irritating to rabbit skin and eyes, but is not a dermal sensitizer in guinea pigs.

Dermal 28-day repeated dose studies in rabbits with two different samples of CASRN 64741-56-6 (identified as API Samples 81-13 and 81-14) showed irritation at all dose levels (lowest tested dose of 200 mg/kg/day). Systemic effects were observed at the highest dose of 2000 mg/kg/day (reduced body weight for both; changes in hematology with 81-13 and mortality with 81-14); the NOAEL for systemic toxicity is 1000 mg/kg/day.

Two different repeat dose inhalation studies were performed with asphalt fume condensates; one with paving asphalt fume condensate (supporting chemical, no CASRN) and one with roofing asphalt fume condensate (supporting chemical, no CASRN). The paving asphalt fume study was a 14 week nose-only study in rats in which the following systemic effects were noted at a concentration of 0.149 mg/L/day: reduced body weight and food consumption, changes in clinical chemistry parameters [all in males] and histopathological changes in the nasal and perinasal cavities [males and females]; the NOAEC for systemic toxicity is 0.028 mg/L/day. In the roofing asphalt fume condensate study, rats were exposed via inhalation (nose-only) in a combined repeated-dose/reproductive/developmental toxicity screening test. For the repeated dose portion of the study, lung effects were observed in females at 0.13 mg/L/day and in males at 0.3 mg/L/day. NOAECs for females and males were 0.03 mg/L/day and 0.1 mg/L/day, respectively.

In the combined repeated-dose/reproductive/developmental toxicity study with the roofing asphalt fume condensate via inhalation, there were no effects on reproduction (highest tested concentration of 0.3 mg/L/day). For developmental toxicity, maternal toxicity (lung effects) was evident at 0.1 mg/L/day and there were no developmental effects at any concentration; the NOAEC for maternal toxicity is 0.03 mg/L/day and the NOAEC for developmental toxicity I 0.3 mg/L/day (highest concentration tested).

A number of genetic toxicity studies were reported on both asphalt mixtures and various fume condensates derived from asphalts. Polycyclic aromatic hydrocarbon (PAH) content is important in these tests, and the PAH content can vary by asphalt type (e.g., crude oil source), fume condensate type (e.g., temperature used) and solvent extraction method. Results show that CASRN 8052-42-4 was not mutagenic in bacteria *in vitro*, and CASRN 64741-56-6 was mutagenic in mammalian cells *in vitro* with metabolic activation. Several fume condensates were also mutagenic in bacteria with metabolic activation *in vitro*. An *in vivo* test for chromosomal aberrations with CASRN 64741-56-6 was negative. In other genotoxicity studies with fume condensates, neither a roofing asphalt nor paving asphalt fume condensate induced micronuclei in rats *in vivo*. However, a paving asphalt fume condensate did induce DNA adduct formation in lung, nasal and alveolar epithelium tissues following inhalation studies in rats. Available studies with asphalts show carcinogenicity via dermal exposures (skin tumors). For fumes generated from asphalts, available data also show carcinogenicity via dermal but not inhalation exposure routes. For the latter, crude oil source and temperature used to generate the fumes are important factors.

Table 7. Summary of Screening Information Data Set Under the U.S. HPV Challenge Program - Human Health Data

Endpoint	**SPONSORED CHEMICAL Asphalt Mixtures**						**SUPPORTING CHEMICAL Fume Condensates Generated from Different Asphalt Mixtures**				
	Asphalt (8052-42-4)	**Residues (petroleum), vacuum (64741-56-6)**	**Residues (petroleum), residual oil decarbonization (64742-07-0)**	**Petroleum resins (64742-16-1)**	**Residues (petroleum), hydro-desulfurized vacuum (64742-85-4)**	**Asphalt, oxidized (64742-93-4)**	**Paving Asphalt fume condensate (2000) (No CASRN)**	**Paving Asphalt fume condensate (2007) (No CASRN)**	**Penetration Asphalt Fume condensate (ether & acetone extracts) (No CASRN)**	**Roofing Asphalt Fume Condensate (No CASRN)**	**NTP Fume Condensates (No CASRN)**
Acute Oral Toxicity LD$_{50}$ (mg/kg-bw)	No Data > 5000 (RA)	**> 5000**	No Data > 5000 (RA)	No Data > 5000 (RA)	No Data > 5000 (RA)	No Data > 5000 (RA)	–	**–**	**–**	**–**	**–**
Acute Inhalation Toxicity LC$_{50}$ (mg/L)	No Data > 0.0944 (RA)	No Data > 0.0944 (RA)	No Data > 0.0944 (RA)	No Data > 0.0944 (RA)	No Data > 0.0944 (RA)	No Data > 0.0944 (RA)	**> 0.0944**	**–**	**–**	**–**	**–**
Acute Dermal Toxicity LD$_{50}$ (mg/kg-bw)	No Data > 2000 (RA)	**> 2000**	No Data > 2000 (RA)	No Data > 2000 (RA)	No Data > 2000 (RA)	No Data > 2000 (RA)	–	**–**	**–**	**–**	**–**
Repeated-Dose Toxicity NOAEC/LOAEC Inhalation (mg/L/day)	No Data NOAEC = 0.03 LOAEC = 0.1 (RA)	No Data NOAEC = 0.03 LOAEC = 0.1 (RA)	No Data NOAEC = 0.03 LOAEC = 0.1 (RA)	No Data NOAEC = 0.03 LOAEC = 0.1 (RA)	No Data NOAEC = 0.03 LOAEC = 0.1 (RA)	No Data NOAEC = 0.03 LOAEC = 0.1 (RA)	**90-Day NOAEC = 0.028 LOAEC = 0.149**	**–**	**–**	**NOAEC = 0.03 LOAEC = 0.1**	–
Repeated-Dose Toxicity NOAEL/LOAEL Dermal (mg/kg-bw/day)	No Data NOAEL = 1000 LOAEL = 2000 (RA)	**(28-d; rabbit) NOAEL = 1000 LOAEL = 2000**	No Data NOAEL = 1000 LOAEL = 2000 (RA)	No Data NOAEL = 1000 LOAEL = 2000 (RA)	No Data NOAEL = 1000 LOAEL = 2000 (RA)	No Data NOAEL = 1000 LOAEL = 2000 (RA)	**–**	**–**	**–**	**–**	**–**
Reproductive Toxicity (Inhaltion, mg/L/day)	No Data NOAEC>0.3 RA)	No Data NOAEC>0.3 RA)	No Data NOAEC>0.3 RA)	No Data NOAEC>0.3 RA)	No Data NOAEC>0.3 RA)	No Data NOAEC>0.3 (RA)	**–**	**–**	**–**	**NOAEC > 0.3 (highest concentration tested)**	**–**

Table 7. Summary of Screening Information Data Set Under the U.S. HPV Challenge Program - Human Health Data											
Endpoint	**SPONSORED CHEMICAL Asphalt Mixtures**						**SUPPORTING CHEMICAL Fume Condensates Generated from Different Asphalt Mixtures**				
	Asphalt (8052-42-4)	**Residues (petroleum), vacuum (64741-56-6)**	**Residues (petroleum), residual oil decarbonization (64742-07-0)**	**Petroleum resins (64742-16-1)**	**Residues (petroleum), hydro-desulfurized vacuum (64742-85-4)**	**Asphalt, oxidized (64742-93-4)**	**Paving Asphalt fume condensate (2000) (No CASRN)**	**Paving Asphalt fume condensate (2007) (No CASRN)**	**Penetration Asphalt Fume condensate (ether & acetone extracts) (No CASRN)**	**Roofing Asphalt Fume Condensate (No CASRN)**	**NTP Fume Condensates (No CASRN)**
Developmental Toxicity Inhalation (mg/L/day)							–	–	–		–
Maternal	No Data NOAEC = 0.03 LOAEC = 0.1	No Data NOAEC = 0.03 LOAEC = 0.1	No Data NOAEC = 0.03 LOAEC = 0.1	No Data NOAEC = 0.03 LOAEC = 0.1	No Data NOAEC = 0.03 LOAEC = 0.1	No Data NOAEC = 0.03 LOAEC = 0.1				**NOAEC = Not established** **LOAEC = 0.03**	
Developmental	NOAEC>0.3 (RA)	NOAEC>0.3 (RA)	NOAEC>0.3 (RA)	NOAEC>0.3 (RA)	NOAEC>0.3 (RA)	NOAEC>0.3 (RA)				**NOAEC>0.297 (highest concentration tested)**	
Genetic Toxicity – Gene Mutation *In vitro*	**Negative**	**(Mammalian) Positive (w/metabolic activation only)**	No Data Positive (metabolic activation only) (RA)	No Data Positive (metabolic activation only) (RA)	No Data Positive (metabolic activation only) (RA)	No Data Positive (metabolic activation only) (RA)	–	–	**(Bacteria) Negative**	–	**(Bacteria) Positive (w/ metabolic activation only)**
Genetic Toxicity – Chromosomal Aberrations *In vivo*	No Data Negative (RA)	**Negative**	No Data Negative (RA)	No Data Negative (RA)	No Data Negative (RA)	No Data Negative (RA)	–	**Negative**	–	**Negative**	–
Genetic Toxicity – Other *In vivo* DNA adducts	No Data Positive (RA)	No Data Positive (RA)	No Data Positive (RA)	No Data Positive (RA)	No Data Positive (RA)	No Data Positive (RA)	–	**Positive**	–	–	–

Table 7. Summary of Screening Information Data Set Under the U.S. HPV Challenge Program - Human Health Data

Endpoint	**SPONSORED CHEMICAL Asphalt Mixtures**						**SUPPORTING CHEMICAL Fume Condensates Generated from Different Asphalt Mixtures**				
	Asphalt (8052-42-4)	**Residues (petroleum), vacuum (64741-56-6)**	**Residues (petroleum), residual oil decarbonization (64742-07-0)**	**Petroleum resins (64742-16-1)**	**Residues (petroleum), hydro-desulfurized vacuum (64742-85-4)**	**Asphalt, oxidized (64742-93-4)**	**Paving Asphalt fume condensate (2000) (No CASRN)**	**Paving Asphalt fume condensate (2007) (No CASRN)**	**Penetration Asphalt Fume condensate (ether & acetone extracts) (No CASRN)**	**Roofing Asphalt Fume Condensate (No CASRN)**	**NTP Fume Condensates (No CASRN)**
Additional Information											
Skin Irritation	−	**Irritating**	−	−	−	−	−	−	−	−	−
Eye Irritation	−	**Irritating**	−	−	−	−	−	−	−	−	−
Dermal Sensitization	−	**Not sensitizing**	−	−	−	−	−	−	−	−	−
Carcinogenicity	**Available studies show carcinogenicity via dermal exposures (skin tumors)**						**Available data show carcinogenicity via dermal (but not inhalation exposure). Depends on fume and temperature.**				

Measured data in bold text; (RA) = read-across; − endpoint not addressed for this chemical

4. Hazard to the Environment

A summary of aquatic toxicity data submitted for SIDS endpoints is provided in Table 8. The table also indicates where data for tested category members are read-across (RA) to untested members of the category.

The supporting chemical, 1-tetradecene (CASRN 1120-36-1) has been previously assessed in the OECD HPV program at SIAM 11 (http://www.chem.unep.ch/irptc/sids/OECDSIDS/AOalfaolefins.pdf). For the supporting chemicals, aromatic extracts and lubricating oil basestocks, although the sponsor did not provide full robust summaries for the aquatic toxicity studies (i.e., many experimental details were not specified), the data have been accepted on a weight-of-evidence basis. The sponsor's submission indicates that the chemicals in this category may contain sulfur (trace to 8 %). The sulfur comes in the following forms: (1) heterocyclic sulfur compounds with multiple fused rings and large molecular weights due to alkylation, and (2) sulfur released in the form of H_2S and mercaptans in very low concentrations (Gamble et al., 1999; Fraunhofer, 2003). Based on these considerations, EPA is not considering the toxicity of hydrogen sulfide or mercaptans to characterize the hazard of members of this asphalt category to aquatic organisms.

Acute Toxicity to Fish

Aromatic extracts (no CASRN, supporting chemical)
Rainbow trout (*Oncorhynchus mykiss*) were exposed to aromatic extracts as a water accommodated fraction (WAF) under unspecified conditions for 96 hours. The loading rates were as high as 1000 mg/L and no analytical measurements were made on the WAFs. No effects were noted at any of the WAF loading rates. EPA does not consider the loading rate as the no-effect concentration when the concentration exceeds the water solubility of the substance.
No effects at saturation.

Lubricating oil basestocks (no CASRN, supporting chemical)
Rainbow trout (*Oncorhynchus mykiss*) were exposed to lubricating oil basestocks as a WAF under unspecified conditions for 96 hours. The loading rates were as high as 1000 mg/L and no analytical measurements were made on the WAFs. No effects were noted at any of the WAF loading rates. EPA does not consider the loading rate as the no-effect concentration when the concentration exceeds the water solubility of the substance.
No effects at saturation.

Acute Toxicity to Aquatic Invertebrates

Aromatic extracts (no CASRN, supporting chemical)
Daphnia magna were exposed to aromatic extracts as a WAF under unspecified conditions for 48 hours. The loading rates were as high as 1000 mg/L and no analytical measurements were made on the WAFs. No effects were noted at any of the WAF loading rates. EPA does not consider the loading rate as the no-effect concentration when the concentration exceeds the water solubility of the substance.
No effects at saturation.

Lubricating oil basestocks (no CASRN, supporting chemical)
Daphnia magna were exposed to lubricating oil basestocks as a WAF under unspecified conditions for 48 hours. The loading rates were as high as 10,000 mg/L and no analytical measurements were made on the WAFs. No effects were noted at any of the WAF loading rates. EPA does not consider the loading rate as the no-effect concentration when the concentration exceeds the water solubility of the substance.
No effects at saturation.

Toxicity to Aquatic Plants

Aromatic extracts (no CASRN, supporting chemical)
Green algae (*Scenedesmus subspicatus*) were exposed to aromatic extracts as a WAF under unspecified conditions for 96 hours. The loading rates were as high as 1000 mg/L and no analytical measurements were made on the WAFs. No effects were noted at any of the WAF loading rates. EPA does not consider the loading rate as the no-effect concentration when the concentration exceeds the water solubility of the substance.
No effects at saturation.

Lubricating oil basestocks (no CASRN, supporting chemical)
Green algae (*Scenedesmus subspicatus*) were exposed to lubricating oil basestocks as a WAF under unspecified conditions for 96 hours. The loading rates were as high as 1000 mg/L and no analytical measurements were made on the WAFs. No effects were noted at any of the WAF loading rates. EPA does not consider the loading rate as the no-effect concentration when the concentration exceeds the water solubility of the substance.
No effects at saturation.

Chronic Toxicity to Aquatic Invertebrates

Aromatic extracts (no CASRN, supporting chemical)
Daphnia magna were exposed to aromatic extracts as a WAF under unspecified conditions for 21 days. The loading rates were as high as 1000 mg/L and no analytical measurements were made on the WAFs. No effects were noted at any of the WAF loading rates. EPA does not consider the loading rate as the no-effect concentration when the concentration exceeds the water solubility of the substance.
No effects at saturation.

Lubricating oil basestocks (no CASRN, supporting chemical)
Daphnia magna were exposed to lubricating oil basestocks as a WAF under unspecified conditions for 21 days. The loading rates were as high as 1000 mg/L and no analytical measurements were made on the WAFs. No effects were noted at any of the WAF loading rates. EPA does not consider the loading rate as the no-effect concentration when the concentration exceeds the water solubility of the substance.
No effects at saturation.

Conclusion: Based on the supporting chemicals, CASRN 1120-36-1, aromatic extracts (no CASRN) and lubricating oil basestocks (no CASRN), acute and chronic toxicity of asphalt category members to fish, aquatic invertebrates and aquatic plants are considered to be no effects at saturation (NES) based on no effects observed at the water solubility limit (saturation).

Table 8. Summary of Environmental Effects – Aquatic Toxicity Data

Endpoints	Fish 96-h LC_{50} (mg/L)	Aquatic Invertebrates 48-h EC_{50} (mg/L)	Aquatic Plants 72-h EC_{50} (mg/L)	Chronic Toxicity to Invertebrates 21-d EC_{50} (mg/L)
SPONSORED CHEMICAL Asphalt (8052-42-4)	No Data NES (RA)	No Data NES (RA)	No Data NES (RA)	No Data NES (RA)
SPONSORED CHEMICAL Residues (petroleum), vacuum (64741-56-6)	No Data NES (RA)	No Data NES (RA)	No Data NES (RA)	No Data NES (RA)
SPONSORED CHEMICAL Residues (petroleum), residual oil decarbonization (64742-07-0)	No Data NES (RA)	No Data NES (RA)	No Data NES (RA)	No Data NES (RA)
SPONSORED CHEMICAL Petroleum resins (64742-16-1)	No Data NES (RA)	No Data NES (RA)	No Data NES (RA)	No Data NES (RA)
SPONSORED CHEMICAL Residues (petroleum), hydrodesulfurized vacuum (64742-85-4)	No Data NES (RA)	No Data NES (RA)	No Data NES (RA)	No Data NES (RA)
SPONSORED CHEMICAL Asphalt, oxidized (64742-93-4)	No Data NES (RA)	No Data NES (RA)	No Data NES (RA)	No Data NES (RA)
SUPPORTING CHEMICAL 1-Tetradecene (1120-36-1)	**NES**	**NES**	**NES**	NES (e)
SUPPORTING CHEMICAL Aromatic extracts (No CASRN)	**NES**	**NES**	**NES**	**NES**

Table 8. Summary of Environmental Effects – Aquatic Toxicity Data

Endpoints	Fish 96-h LC_{50} (mg/L)	Aquatic Invertebrates 48-h EC_{50} (mg/L)	Aquatic Plants 72-h EC_{50} (mg/L)	Chronic Toxicity to Invertebrates 21-d EC_{50} (mg/L)
SUPPORTING CHEMICAL Lubricating oil basestocks (No CASRN)	**NES**	**NES**	**NES**	**NES**

NES = No effects at saturation (water solubility limit); (RA) = Read Across; (e) = ECOSAR predicted value

5. References

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Gamble, J.F., Nicholich, M.J., Baron, N.J., and Vincent, W.J. 1999. Exposure-response of asphalt fumes with changes in pulmonary function and symptoms. *Scand J Work Environ Health* 25: 186-206

Halter R, T Hansen, A Seidel, C Ziemann,and J Borlak. 2007. Importance of DNA-Adduct Formation and Gene Expression Profiling of Disease Candidate Genes in Rats Exposed to Bitumen Fumes *Journal of Occupational and Environmental Hygiene*, 4(S1): 44–64

Machado, ML, PW Beatty, JC Tetzer, AH Glickman and EI McGinnis. 1993. Evaluation of the relationship between PAH content and mutagenic activity of fumes from roofing and paving asphalts and coal tar pitch. *Fund. Appl. Tox.,* v. 21, pp 492-499.

Schreiner, C. 2010. Review of mechanistic studies relevant to the potential carcinogenicity of asphalts. *Reg. Tox. Pharm.* (accessed online on January 19, 2011; doi:10.1016/j.yrtph.2010.10.010)

APPENDIX

The following pages show:

- Table 9: Asphalt and Fumes Mixtures Used for Human Health Endpoints Testing in the Asphalt Category
- Table 10: PAH Content of Various Fume Condensates Used in Toxicity Tests
- Table 11: PAH Content of Various Test Mixtures Used in Genotoxicity Tests
- A short narrative followed by a table with Chemical Names, CASRNs and Descriptions for the Asphalt Category members.

Table 9: Asphalt and Fumes Mixtures Used for Human Health Endpoints Testing in the Asphalt Category

Type	Test Material Name	Associated Sponsored Category Member	Endpoint(s) Used
Asphalt (aka Bitumen)	API Sample 81-13	CASRN 64741-56-6	Acute oral and acute dermal toxicity, skin and eye irritation, sensitization in guinea pig, dermal repeated dose study in rabbits (4 wks), in vitro gene mutation test in mouse cell line, in vivo chromosomal aberration test
	API Sample 81-14	CASRN 64741-56-6	Sensitization in guinea pigs, dermal repeated dose study in rabbits (4 wks), in vitro gene mutation test in mouse cell line, in vivo chromosomal aberration test
	Penetration Asphalt (DMSO extraction)	CASRN 8052-42-4	Ames test
	Asphalt-based paints	CASRN 8052-42-4	Ames test
Fumes (Asphalts that have been heated)	Paving asphalt fume condensate (2000)	CASRN 8052-42-4	Acute inhalation toxicity, inhalation 90-day study
	Penetration asphalt fume (various solvent extractions)	CASRN 8052-42-4	Ames test
	Fume condensates of coal tar pitches, roofing asphalts and paving asphalts	CASRN 64742-93-4 (for the roofing/paving asphalts)	Ames test
	Paving asphalt fume condensate (2007)	CASRN 64742-93-4	2-year inhalation study with several peripheral studies (DNA adducts, micronucleus assay, (nose-only inhalation study)[1]
	Roofing asphalt fume condensate (RAFC)	CASRN 64742-93-4	OECD 422 study with a peripheral group evaluated for genetic toxicity (micronucleus assay)
	Asphalt fume extract from NTP studies	Not provided	Ames test

[1] This study is published in a special supplement of the *Journal of Occupational and Environmental Hygiene* which is dedicated to the toxicity and exposure of asphalt and asphalt fumes (J. Occup. Env. Hyg [2007], Volume 4, Suppl 1).

Table 10: PAH Content of Various Fume Condensates Used in Toxicity Tests*[1]

PAH	Paving (2000)[2]	Paving (2000)[3]			Paving (2007)[4]			RAFC[5]
		Low	Mid	High	Low	Mid	High	
Naphthalene	4709	409	1641	8304	1014	3687	19578	164
Acenaphthylene	ND	ND	ND	ND	ND	ND	ND	ND
Acenaphthene	96	222	1046	4754	477	1923	10093	29
Fluorene	42	505	2296	11162	801	3520	21690	254
Phenanthrene	111	449	2450	15743	331	1641	11580	248
Anthracene	42	ND	ND	ND	ND	ND	ND	32
Fluoranthene	39	26	150	631	43	220	1295	11
Pyrene	95	57	303	1311	45	219	1139	49
Benz(a)anthracene	30	8	46	217	7	31	169	8
Chrysene	31	13	78	377	10	45	251	20
Benzo(b)fluoranthene	11	5	23	116	4	15	83	5
Benzo(k)fluoranthene	ND	ND	ND	ND	ND	ND	7	2
Benzo(e)pyrene	23	9	46	222	4	19	200	8
Benzeo(a)pyrene	4	2	10	54	ND	5	30	4
Indeno(1,2,3-cd)pyrene	ND	ND	ND	ND	ND	ND	5	1
Dibenz (ah)anthracene	ND	ND	3	21	ND	ND	7	1
Benzp(ghi)perylene	3	2	10	50	3	4	24	3
Triphenylene	NR	NR	NR	NR	11	47	249	21

* ND= Not detected; NR = not reported as being analyzed for in this sample.

[1] Units are ng/m^3 (or ug/g for the RAFC in the last column) and are taken from the robust summaries submitted in 2009. All values were rounded to the nearest whole number.

[2] Refers to the paving asphalt fume condensate from the studies performed in 2000 (acute inhalation study).

[3] Refers to the paving asphalt fume condensate from the studies performed in 2000 (90-day inhalation study; low, mid and high concentrations were 4, 20 and 100 mg/m^3, respectively).

[4] Refers to the paving asphalt fume condensate from the studies performed in 2007 (2-year inhalation study with peripheral evaluations of DNA adduct formation and a micronucleus assay).

[5] Refers to the roofing asphalt fume condensate (AFC) from studies performed in 2007 (OECD 422 with a peripheral evaluation using a micronucleus assay).

Table 11 : PAH Content of Various Test Mixtures Used in Genotoxicity Tests*[1]

PAH	Penetration Asphalt[2]		Asphalt Paint Samples[5]	Asphalt Fume Condensates[6]	
	DMSO Extractions of solid asphalt[3]	Ether Extractions of fumes[4]		Roofing asphalt[7]	Paving Asphalt[8]
Naphthalene	ND	240	300	39.2	1.70
Acenaphthalene	ND	ND	>10	ND	0.73
Acenaphthene	4	1260	ND	12	0.3
Fluorene	1	80	>10	28.4	0.26
Phenanthrene	11	220	>10	8.4	1.04
Anthracene	7	130	ND	0.6	0.63
Fluoranthene	40	1130	ND	0.6	1.59
Pyrene	8	540	ND	8.5	2.09
Benz(a)anthracene	5	3500	ND	2.6	0.68
Chrysene	72	200	ND	19.4	1.45
Benzo(b)fluoranthene	36	1030	NR	1.2	0.91
Benzo(k)fluoranthene	8	670	NR	0.4	1.31
Benzo(e)pyrene	NR	NR	ND	NR	
Benzeo(a)pyrene	7	610	ND	1.6	1.97[9]
Indeno(1,2,3-cd)pyrene	7	50	NR	ND	1.44
Dibenz (ah)anthracene	9	980	NR	ND	0.82
Benzp(ghi)perylene	2	190	NR	5.4	1.56
Biphenyl	NR	NR	>10	NR	NR
9-H-Fluorene	NR	NR	ND	NR	NR
Acridine	NR	NR	>10	NR	NR
2-Methylphenanthrene	NR	NR	>10	NR	NR
2-Methylanthracene	NR	NR	?	NR	NR
1-Methylpyrene	NR	NR	NR	NR	NR
Perylene	NR	NR	NR	NR	0.83

* ND= Not detected; NR = not reported as being analyzed for in this sample;? = left blank in the robust summary.

[1] All units are in ng/m^3 (unless otherwise noted) and are taken from the robust summaries submitted in 2009. All values were rounded to the nearest whole number.

[2] Three samples of solid, penetration asphalt were collected and dissolved in benzene. Asphaltenes were separated from the samples using n-heptane; and this fraction was extracted with DMSO to produce the test substance. The values under "DMSO extractions…" represent Sample 3, the one with the highest PAH content. In addition, two fume samples were taken during paving operations; the values listed under "Ether extractions…" represent Sample 2 (again, the highest PAH content).

[3] Units are in **ug/g**.

[4] Units were converted from **ug/m^3 to ng/m^3** (multiply by 1000) for ease in comparing with other values for fumes.

[5] The asphalt paint samples were derived from petroleum asphalt which was "cut back" to 64% solid by the addition of mineral spirits. A small amount of

Table 11 : PAH Content of Various Test Mixtures Used in Genotoxicity Tests*[1]					
PAH	**Penetration Asphalt[2]**		**Asphalt Paint Samples[5]**	**Asphalt Fume Condensates[6]**	
	DMSO Extractions of solid asphalt[3]	**Ether Extractions of fumes[4]**		**Roofing asphalt[7]**	**Paving Asphalt[8]**

xylene was then added to create the four different asphalt paint samples evaluated. Units were converted from **mg/g to ug/g** (multiply by 1000) for ease in comparing with other values. (Sample D values were used as reported on p. 32 in the 2009 robust summaries).

[6] Refer to the Ames test summarized on pp. 32-34 of th e2009 robust summaries.

[7] Taken from Table 2 in Machado et al., 1993. The highest sample is presented (Asphalt No. 3 Fume, generated at 316 °C; the last column on the right). The reported values **are in ppm, which is equivalent to ug/g.**

[8] Taken from Table 3 in Machado et al., 1993. In this case, the data show individual results from 18 different samples. The highest “sum” value is presented here (Sample 11). Again, the reported values **are in ppm, which is equivalent to ug/g.**

[9] B(a)P is listed twice in Table 3 in Machado et al., 1993. The first value was taken.

Chemical Names, CASRNs and Descriptions.

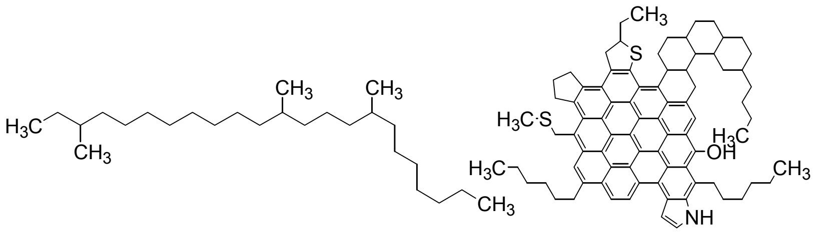
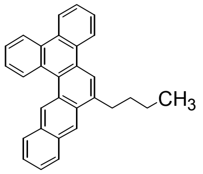
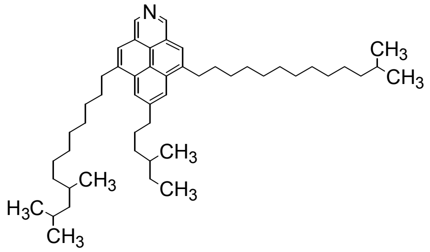
The major chemical groups in produced asphalt are:

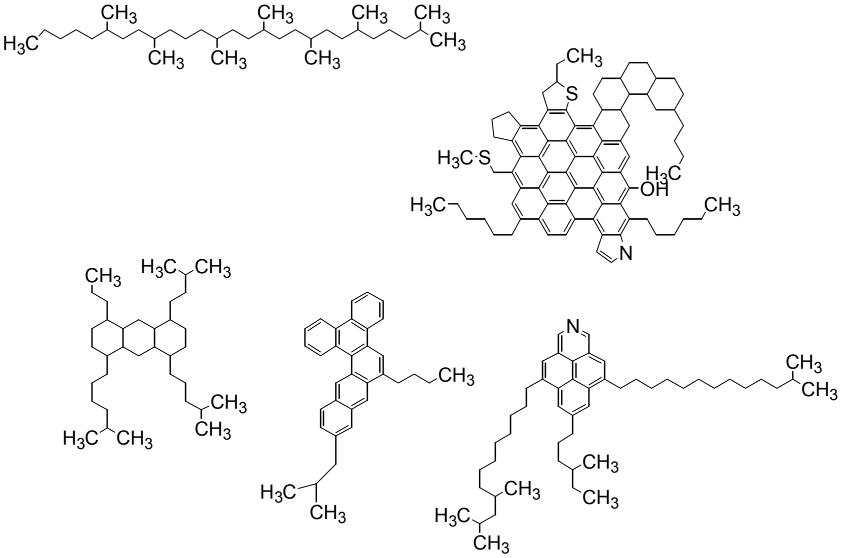
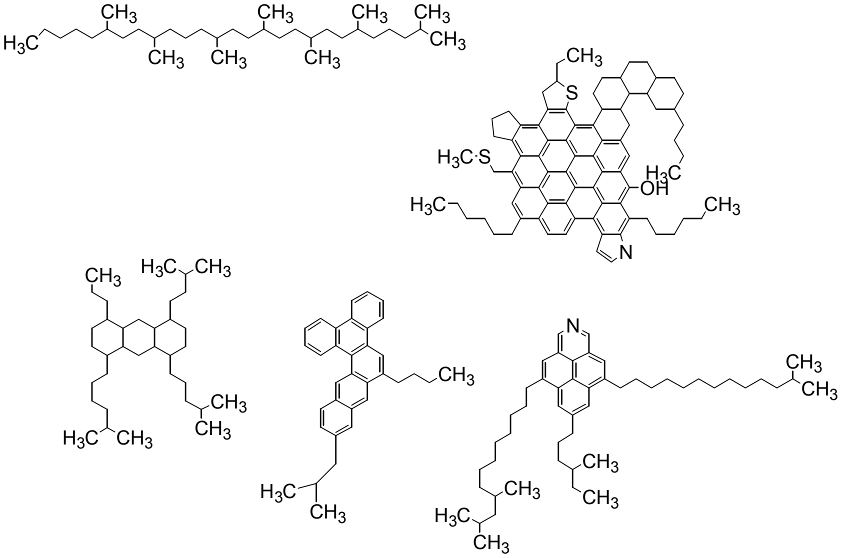
1) Asphaltenes: brittle brown-black amorphous solids, which are highly condensed aromatic compounds with molecular weight 2,000–5,000, constituting 5–25% of the weight of asphalts. They comprise one or two chromophores containing 4 to 10 fused rings each, with a significant number of alkyl substituents. A higher proportion of asphaltenes are present in the harder asphalts.

2) Resins: brown-black, adhesive, shiny solids or semi-solids. Composed of heterogeneous polar aromatic compounds with small amounts of oxygen, nitrogen, and sulfur with molecular weights of 800–2,000, constituting 15–25% of the weight of asphalts.

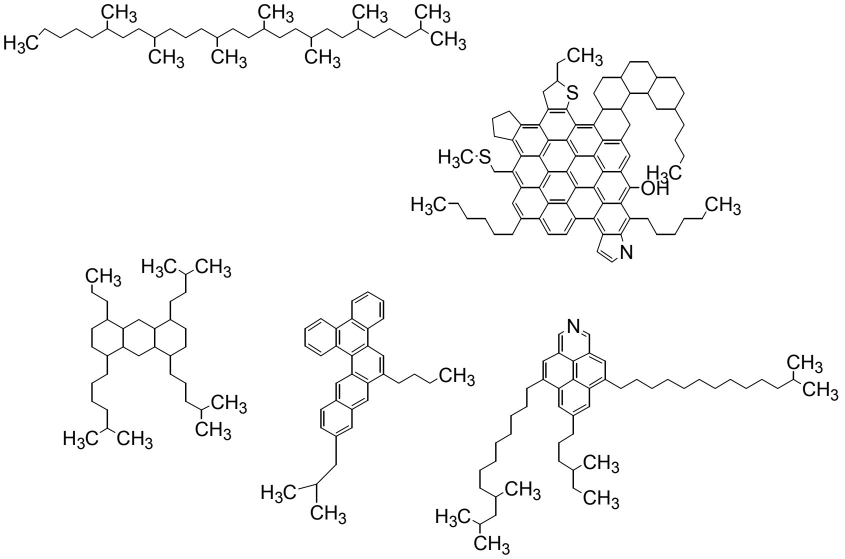
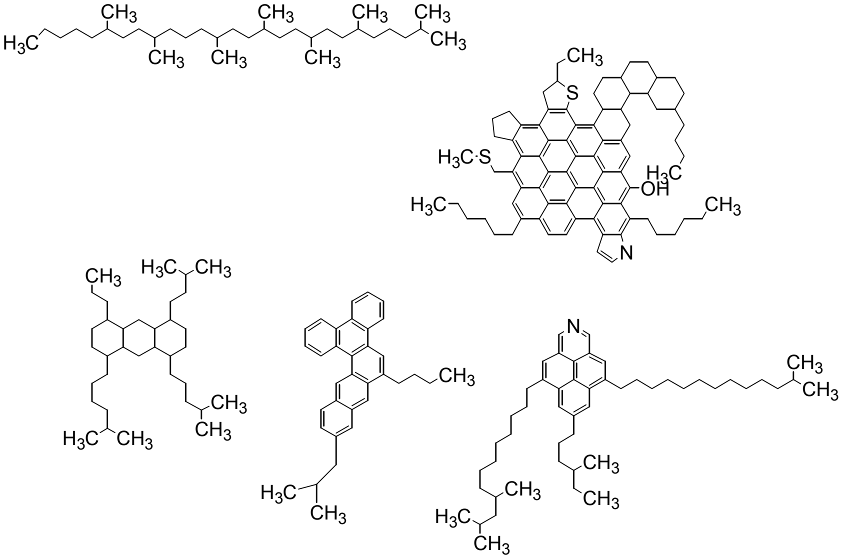
3) Aromatic oil components: viscous dark brown liquids containing mainly carbon, hydrogen, and sulfur with minor amounts of oxygen and nitrogen, with a molecular weight of 500–900, constituting 45–60% of the weight of the asphalt.

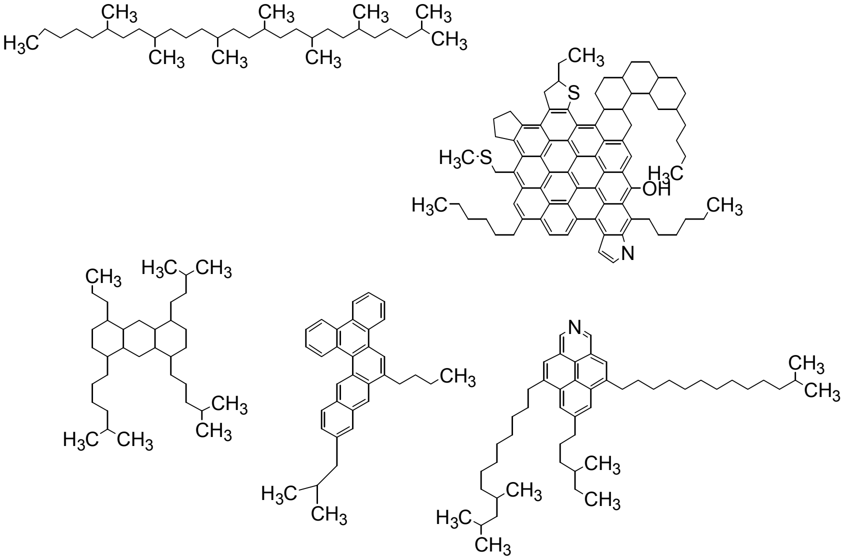
4) Saturated oil components: viscous liquids or solids ranging from straw to water-white in color, consisting mainly of long chain saturated hydrocarbons with some branched chain compounds, alkyl aromatics with long side chains, and cyclic paraffins (naphthenes), with a molecular weight of 500–1,000, constituting 5–20% of the weight of the asphalt.

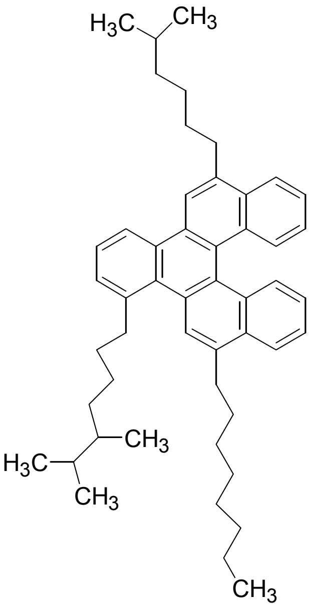
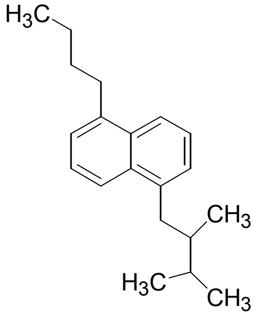
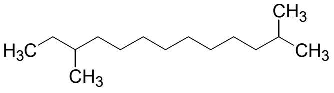
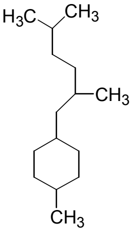
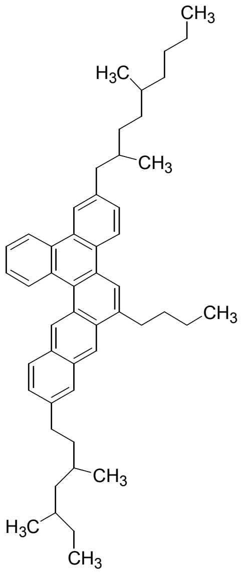
Sponsored Chemicals		
Chemical Name	**CASRN**	**Description or Chemical Structure**
Asphalt	8052-42-4	   A very complex combination of high molecular weight organic compounds containing a relatively high proportion of hydrocarbons with carbon numbers predominantly greater than C25 with high carbon-to-hydrogen ratios. It also contains small amounts of various metals such as nickel, iron, or vanadium. It is obtained as the non-volatile residue from distillation of crude oil or by separation as the raffinate from a residual oil in a deasphalting or decarbonization process.

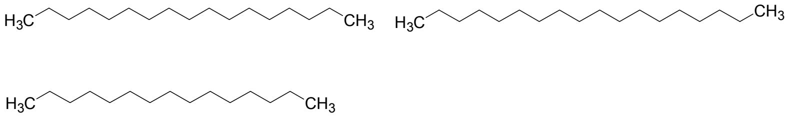
Sponsored Chemicals		
Chemical Name	**CASRN**	**Description or Chemical Structure**
Residues (petroleum), vacuum	64741-56-6	 A complex residuum from the vacuum distillation of the residuum from atmospheric distillation of a crude oil. It consists of hydrocarbon having carbon numbers predominantly greater than C34 and boiling above approximately 495°C.
Raffinates (petroleum), residual oil decarbonization	64742-07-0	 A complex combination of hydrocarbons obtained as the insoluble fraction from C5–C7 solvent decarbonization of a residual oil. It consists predominantly of aromatic hydrocarbons having carbon numbers predominantly higher than C34 and boiling above approximately 495°C.

Sponsored Chemicals

Chemical Name	CASRN	Description or Chemical Structure
Petroleum resins	64742-16-1	 A complex combination of organic compounds, predominantly hydrocarbons, obtained as a fraction of the extract of solvent extraction of residuum. It consists predominantly of high molecular weight compounds with high carbon-to-hydrogen ratios.
Residues (petroleum), hydrodesulfurized vacuum	64742-85-4	 A complex combination of hydrocarbons obtained by treating a vacuum residuum with hydrogen in the presence of a catalyst under conditions primarily to remove organic sulfur compounds. It consists of hydrocarbons having carbon numbers predominantly greater than C34 and boiling above approximately 495°C.

Sponsored Chemicals		
Chemical Name	CASRN	Description or Chemical Structure
Asphalt, oxidized	64742-93-4	 A complex, black solid obtained by blowing air through a heated residuum, or raffinate from a deasphalting process with or without a catalyst. The process is principally one of oxidative condensation which increases the molecular weight.

Supporting Chemicals		
Chemical Name	**CASRN**	**Description or Chemical Structure**
Aromatic extracts	Multiple CASRN	 
Lubricating oil basestocks	Multiple CASRNs	   Lubricating oil basestocks contain saturated as well as aromatic hydrocarbons of C15–C50

Supporting Chemicals		
Chemical Name	**CASRN**	**Description or Chemical Structure**
Asphalt fume condensate	Multiple CASRN	 Asphalt fume is a visible airborne condensation product of lower boiling volatile components of petroleum asphalt. Most of the compounds identified by GC/MS were characterized as n-alkanes (73%). Polycyclic aromatic hydrocarbons and thia-arenes contributed roughly 8%.[1]

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[1] Rogge,WF; Hildemann, LM; Mazurek, MA; Cass, GR; Simoneit, BRT. 1997. Sources of fine organic aerosol. Hot asphalt roofing tar pot fumes. Environ Sci Technol 31:2726–2730.

GRENHALL

- SAFETY DATA SHEET -

SECTION 1: IDENTIFICATION

Product Name: **PETRORESIN GC-100**
Chemical Name: Petroleum Hydrocarbon Resin
Recommended use: Tackifier used in the manufacture of Adhesives, Inks & Coatings
Restrictions on use: None

MANUFACTURER / SUPPLIER:
Grenhall Industries Inc.
1 Imperial Court
Brampton, Ontario, Canada L6T 4X4
Tel: 905-458-8549
Fax: 905-458-8363
www.grenhall.com

EMERGENCY CONTACT:
Chemtrec: 1-800-424-9300
Canutec: 613-996-6666

SECTION 2: HAZARDS IDENTIFICATION

Hazard Classification: This product is considered non-hazardous.
This product is not classified according to OSHA regulations (CFR 29 1910.1200)

OSHA Specified Hazards:
Combustible dust — If converted to small particles during further processing, handling or by other means may form combustible dust concentrations in air.

Warning label items including precautionary statement:

Signal Word(s): **WARNING!**

Hazard Statement(s): If converted to small particles during further processing, handling or by other means, product may form combustible dust concentrations in air.

Precautionary Statement:
Disposal: Dispose of contents/container to an appropriate treatment and disposal facility in accordance with applicable laws and regulations, and product characteristics at time of disposal.

Hazard(s) not otherwise classified (HNOC): None known

SECTION 3: COMPOSITION / INFORMATION ON INGREDIENTS

Substances / Mixtures

General Information:

Chemical Name	CAS No.	Concentration
Petroleum Resins	64742-16-1	100%

SECTION 4: FIRST-AID MEASURES

INHALATION:	In pellet form, this product is not expected to be a problem. If resin dust is inhaled, move victim to fresh air. Treat symptomatically. Get medical attention if symptoms persist.
EYE CONTACT:	Any material that contacts the eye should be washed out immediately with water. Get medical attention if symptoms persist.
SKIN CONTACT:	Wash contact areas with soap and water. Get medical attention if symptoms persist.
INGESTION:	Seek medical advice. Material is not expected to be absorbed from the gastrointestinal tract so that induction of vomiting should not be necessary.

SECTION 5: FIRE-FIGHTING MEASURES

General Fire Hazards:	Material can accumulate static charges which may cause an electrical spark (ignition source). Use proper bonding and/or grounding procedures.
Extinguishing media	
Suitable extinguishing media:	Water spray. Carbon Dioxide. Dry chemical.
Unsuitable extinguishing media:	None known
Special hazards arising from the substance: or mixture	Powdered material may form explosive dust-air mixture
Advice for firefighters	
Special fire fighting procedures:	Minimize dust generation and accumulation.
Special protective equipment for fire-fighters:	Self-contained breathing apparatus and full protective clothing must be worn in case of fire.

SECTION 6: ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures:	Wear appropriate personal protective equipment.
Environmental Precautions:	Not regarded as dangerous for the environment.
Methods and material for containment and cleaning up:	Sweep up and place in a clearly labeled container for chemical waste. Large Spillages: Flush spill area with water spray. Prevent runoff from entering drains, sewers, or streams. Dike for later disposal.
Notification Procedures:	In the event of a spill or accidental release, notify relevant authorities in accordance with all applicable regulations.

SECTION 7: HANDLING AND STORAGE

Precautions for safe handling:	Minimize dust generation and accumulation.
Conditions for safe storage, including any incompatibilities:	Keep container closed.
Specific end use(s):	Chemical Intermediate

SECTION 8: EXPOSURE CONTROLS / PERSONAL PROTECTION

Control Parameters

Occupational Exposure Limits	Country specific exposure limits have not been established or are not applicable unless listed below.

Exposure controls

Appropriate engineering controls:	Good general ventilation (typically 10 air changes per hour) should be used. Ventilation rates should be matched to conditions. If applicable, use process enclosures, local exhaust ventilation, or other engineering controls to maintain airborne levels below recommended exposure limits. If exposure limits have not been established, maintain airborne levels to an acceptable level.

Individual protection measures, such as personal protective equipment

General information:	Eye bath. Washing facilities.
Eye/face protection:	It is a good industrial hygiene practice to minimize eye contact.

Skin protection

Hand Protection:	It is a good industrial hygiene practice to minimize skin contact.
Other:	No data available.
Respiratory Protection:	If engineering controls do not maintain airborne concentrations below recommended exposure limits (where applicable) or to an acceptable level (in countries where exposure limits have not been established), an approved respirator must be worn. In the United States of America, if respirators are used, a program should be instituted to assure compliance with OSHA Standard 63 FR 1152, January 8, 1998. Respirator type: Air-purifying respirator with an appropriate, government approved (where applicable), air-purifying filter, cartridge or canister. Contact health and safety professional or manufacturer for specific information.
Hygiene measures:	Observe good industrial hygiene practices.
Environmental Controls:	No data available.

SECTION 9: PHYSICAL AND CHEMICAL PROPERTIES

Appearance

Physical State:	Solid
Form:	Flakes or Prills
Color:	Amber or light yellow

Odour: weak, characteristic hydrocarbon
Odour Threshold: Not determined.
pH: No data available.
Softening Point: 130-135°C (Ring and Ball Method)
Boiling Point: No data available.
Flash Point: 260°C (Cleveland open cup)
Evaporation Rate: Not applicable.
Flammability (solid, gas): Product is not flammable.
Flammability Limit - Upper (%)–: Not applicable.
Flammability Limit - Lower (%)–: Not applicable.
Vapour pressure: Not determined.
Vapour density (air=1): No data available.
Specific Gravity: 1.06 (25°C)
Solubility(ies)
Solubility in Water: Negligible
Solubility (other): No data available.
Partition coefficient (n-octanol/water): No data available.
Autoignition Temperature: Product is not self-igniting.
Decomposition Temperature: Not determined.
Dynamic Viscosity: Not determined.
Kinematic viscosity: Not determined.
Explosive properties: Product is not explosive.
Oxidizing properties: Not oxidizing.

SECTION 10: STABILITY AND REACTIVITY

Reactivity: None known
Chemical Stability: Product is stable under ordinary conditions.
Possibility of Hazardous Reactions: None known
Conditions to avoid: None at ambient temperature
Incompatible Materials: Strong oxidizing agents
Hazardous Decomposition Products: Carbon Monoxide. Carbon Dioxide.

SECTION 11: TOXICOLOGICAL INFORMATION

Information on likely routes of exposure
Inhalation: None known.

Ingestion: None known.

Skin Contact: None known.

Eye contact: None known.

Information on toxicological effects

Oral
Product: No data available.

Dermal
Product: No data available.

Inhalation
Product: No data available.

Repeated Dose Toxicity

Product:	No data available.

Skin Corrosion/Irritation

Product:	No data available.

Serious Eye Damage/Eye Irritation

Product:	No data available.

Respiratory or Skin Sensitization

Product:	No data available.

Carcinogenicity

Product:	No data available.

Specified substance(s):

additive(s)/colorant(s) IARC Not Listed. NTP Not Listed. OSHA Not Listed.

Toxicity to reproduction

Product:	No data available.

Developmental Toxicity

Product:	No data available.

Germ Cell Mutagenicity

In vitro Product:	No data available.
In vivo Product:	No data available.

Specific Target Organ Toxicity - Single Exposure

Product:	No data available.

Specific Target Organ Toxicity - Repeated Exposure

Product:	No data available.

Aspiration Hazard

Product:	No data available.

Other Effects:	No data available.

SECTION 12: ECOLOGICAL INFORMATION

Ecotoxicity:

Acute hazards to the aquatic environment:

Fish

Product:	No data available.

Aquatic Invertebrates

Product:	No data available.

Chronic hazards to the aquatic environment:

Fish

Product:	No data available.

Aquatic Invertebrates

Product:	No data available.

Toxicity to Aquatic Plants

Product:	No data available.

Persistence and Degradability

Biodegradation

Product:	Not easily biodegradable. Easily eliminable from water.

BOD/COD Ratio

Product:	No data available.

Bioaccumulative Potential

Bioconcentration Factor (BCF)

Product:	No data available.

Partition Coefficient n-octanol / water (log Kow)

Product:	No data available.

Mobility in Soil:	Adsorption to solid phase is possible.

Known or predicted distribution to environmental compartments

hydrocarbon resin	No data available.
additive(s)/colorant(s)	No data available.

Other Adverse Effects:	No data available.

SECTION 13: DISPOSAL CONSIDERATIONS

Waste treatment methods

General information:	No data available.
Disposal methods:	Dispose of waste and residues in accordance with local authority requirements. Incinerate.

SECTION 14: TRANSPORT INFORMATION

TDG:	Class not regulated
DOT:	Class not regulated
IMDG:	Class not regulated
IATA:	Class not regulated

SECTION 15: REGULATORY INFORMATION

Safety, health and environmental regulations/legislation specific for the substance or mixture:

WHMIS (Canada) Status: non-controlled

SARA 311-312 Hazard Classification(s):
fire hazard

US EPCRA (SARA Title III) Section 313 - Toxic Chemical List
NONE
NONE

OSHA: non-hazardous
DSL (Canada - Domestic Substances List) Inventory: Product is listed

TSCA (US Toxic Substances Control Act): All components of this product are listed on the TSCA inventory. Any impurities present in this product are exempt from listing.

MITI (Japanese Handbook of Existing and New Chemical Substances): All components of this product are listed in the Handbook or have been approved in Japan by new substance notification.

Philippines Inventory (PICCS) : All components of this product are listed on the Philippine inventory or otherwise comply with PICCS.

Inventory of Existing Chemical Substances in China: All components of this product are listed on the Inventory of Existing Chemical Substances in China (IECSC).

Australian Inventory of Chemical Substances: Product is listed.

New Zealand Inventory of Chemical Substances: Product is listed.

SECTION 16: OTHER INFORMATION

According to REACH, this product does not legally require a SDS. However, it is used to communicate information according to Article 32 of REACH.

Revision Date:	**October 2021**
Supersedes Date:	April 2018
Supersedes Date:	April 2015
Revision Information:	New Format

Asphalt: Human health tier II assessment

29 June 2018

Chemicals in this assessment

Chemical Name in the Inventory	CAS Number
Asphalt	8052-42-4
Gilsonite	12002-43-6
Residues, petroleum, vacuum	64741-56-6
Raffinates, petroleum, residual oil decarbonization	64742-07-0
Petroleum resins	64742-16-1
Residues, petroleum, hydrodesulfurized vacuum	64742-85-4
Asphalt, oxidized	64742-93-4

Preface

This assessment was carried out by staff of the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) using the Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework.

The IMAP framework addresses the human health and environmental impacts of previously unassessed industrial chemicals listed on the Australian Inventory of Chemical Substances (the Inventory).

The framework was developed with significant input from stakeholders and provides a more rapid, flexible and transparent approach for the assessment of chemicals listed on the Inventory.

Stage One of the implementation of this framework, which lasted four years from 1 July 2012, examined 3000 chemicals meeting characteristics identified by stakeholders as needing priority assessment. This included chemicals for which NICNAS already held exposure information, chemicals identified as a concern or for which regulatory action had been taken overseas, and chemicals detected in international studies analysing chemicals present in babies' umbilical cord blood.

Stage Two of IMAP began in July 2016. We are continuing to assess chemicals on the Inventory, including chemicals identified as a concern for which action has been taken overseas and chemicals that can be rapidly identified and assessed by using Stage One information. We are also continuing to publish information for chemicals on the Inventory that pose a low risk to human health or the environment or both. This work provides efficiencies and enables us to identify higher risk chemicals requiring assessment.

The IMAP framework is a science and risk-based model designed to align the assessment effort with the human health and environmental impacts of chemicals. It has three tiers of assessment, with the assessment effort increasing with each tier. The Tier I assessment is a high throughput approach using tabulated electronic data. The Tier II assessment is an evaluation of risk on a substance-by-substance or chemical category-by-category basis. Tier III assessments are conducted to address specific concerns that could not be resolved during the Tier II assessment.

These assessments are carried out by staff employed by the Australian Government Department of Health and the Australian Government Department of the Environment and Energy. The human health and environment risk assessments are conducted and published separately, using information available at the time, and may be undertaken at different tiers.

This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

For more detail on this program please visit:**www.nicnas.gov.au**

Disclaimer

NICNAS has made every effort to assure the quality of information available in this report. However, before relying on it for a specific purpose, users should obtain advice relevant to their particular circumstances. This report has been prepared by NICNAS using a range of sources, including information from databases maintained by third parties, which include data supplied by industry. NICNAS has not verified and cannot guarantee the correctness of all information obtained from those databases. Reproduction or further distribution of this information may be subject to copyright protection. Use of this information without obtaining the permission from the owner(s) of the respective information might violate the rights of the owner. NICNAS does not take any responsibility whatsoever for any copyright or other infringements that may be caused by using this information.

ACRONYMS & ABBREVIATIONS

Grouping Rationale

The chemicals in this group are naturally formed by the evaporation of crude petroleum followed by chemical reactions such as oxidation and polymerisation (gilsonite, CAS No. 12002-43-6) or heavy residual streams derived from the vacuum distillation of petroleum. The chemicals have high carbon to hydrogen ratios, with carbon numbers predominantly greater than C25, high molecular weights (MWs) (500–5000 Da) and high viscosities. The chemicals are categorised as being of unknown or variable composition, complex reaction products or biological materials (UVCBs). The asphalts (with CAS Nos 8052-42-4 and 64742-93-4), are more commonly referred to as bitumen in Europe, and are the material that remains after all refinery products have been extracted from cracked crude oil. Gilsonite (CAS No. 12002-43-6) is considered as natural asphalt and is also referred to as bitumen. The other chemicals in this group (with CAS Nos 64741-56-6, 64742-07-0, 64742-16-1, and 64742-85-4) are not considered asphalts; however, they consist primarily of high molecular weight hydrocarbons and have similar physical properties to asphalt. The chemicals in this group can be sold as is, or blended together, or subsequently processed to meet certain physicochemical (performance) specifications for a variety of asphalt end-use products (API Petroleum HPV Testing Group, 2009).

The exact composition of the chemicals in this group is dependent on the chemical complexity of the original crude petroleum and the refining process. The composition of the crude petroleum may vary between oil fields and even within the same oil field (at different locations). The refining process can result in changes to the physical properties of the chemicals; however, its chemical nature only changes if thermal cracking occurs (API Petroleum HPV Testing Group, 2009).

Elemental analyses indicate that most asphalts typically contain 79–88 % (w/w) carbon, 7–13 % hydrogen, up to 3 % nitrogen, up to 8 % sulfur, up to 8 % oxygen with trace amounts of vanadium, nickel, aluminium and silicon. The major chemical groups present within asphalt are as follows:

- Asphaltenes (5–25 % by weight of asphalt, typical MW 2000–5000 Da)—highly condensed aromatic compounds comprised of one or two chromophores containing 4–10 fused rings in each, and a significant number of alkyl substituents;
- Resins (15–25 % by weight of asphalt, typical MW 800–2000 Da)—heterogeneous polar aromatic compounds with small amounts of oxygen, nitrogen and sulfur;
- Aromatic oil components (45–60 % by weight of asphalt, typical MW 500–900 Da)—compounds with aromatic and naphthenic-aromatic rings with alkyl side chains. These contain mainly carbon, hydrogen and sulfur, and small amounts of oxygen and nitrogen; and
- Saturated oil components (5–20 % by weight of asphalt, typical MW 500–1000 Da)—mainly long chain saturated hydrocarbons with some branched alkyl chain compounds, alkyl aromatics with long side chains and cyclic paraffins (naphthenes).

Natural asphalt gilsonite (CAS No. 12002-43-6) is unrefined and contains mainly of asphaltenes (approximately 60–80%, w/w) and resins (20–40%, w/w) while the aromatics and saturates account only for <1% and <2% (w/w), respectively (Nciri et al., 2014; HSDB).

Oxidised asphalt (CAS No. 64742-93-4) is asphalt which has been air-blown at elevated temperatures to produce a final product that meets technical specifications for the intended use. The process of air blowing causes oxidation and condensation polymerisation, which produces higher molecular weight compounds that give a harder, less temperature sensitive product. The asphaltene content is increased while the cyclic aromatic content of the resins decreases (API Petroleum HPV Testing Group 2009). There are two main grades of oxidised asphalt: fully oxidised and semi-blown; each has different characteristics and uses (IARC, 2013).

The temperature and pressure used to manufacture asphalt do not facilitate formation of polycyclic aromatic compounds (PACs) with 3–7 fused rings. These are typically present in the low parts per million (ppm) range (US EPA, 2006).

The chemicals in this group have similar uses which may involve heating and the generation of fumes. The chemical composition of these fumes is affected by the temperature of fume generation. There is strong evidence that higher application temperatures are associated with higher exposures of PACs (US EPA, 2006).

Import, Manufacture and Use

Australian

The following Australian industrial uses were reported under previous mandatory and/or voluntary calls for information.

Asphalt (CAS No. 8052-42-4) and petroleum resins (CAS No. 64742-16-1) have reported commercial use including as:

- additives in construction materials; and
- lubricants and additives.

Petroleum resins (CAS No. 64742-16-1) has reported potential domestic use in adhesives and binding agents.

Petroleum resins (CAS No. 64742-16-1) has reported site-limited use in stabilisers.

The following chemicals are listed on the 2006 High Volume Industrial Chemicals List (HVICL):

- asphalt (CAS No. 8052-42-4) with a total reported volume of 100000–999999 tonnes; and

- petroleum resins (CAS No. 64742-16-1) with a total reported volume of 1000–9999 tonnes.

International

The following international uses have been identified through the European Union (EU) Registration, Evaluation and Authorisation of Chemicals (REACH) dossiers (REACHa; REACHb); the Substances and Preparations in the Nordic countries (SPIN) database; the OECD High Production Volume chemical program (OECD HPV); Galleria Chemica; Hazardous Substances Data Bank (HSDB); and various international assessments/documents (IPCS, 2004; US EPA, 2006; Asphalt Institute, 2011; IARC, 2013).

The main uses of asphalts are paving (such as roads, airport runways and car parks) (85 %), roofing applications (10 %) and specialised applications such as waterproofing, damp proofing, pipe coatings, insulation, lubricants and paints and protective coatings to prevent corrosion of metals (5 %) (IPCS, 2004; API Petroleum HPV Testing Group, 2009; IARC, 2013; Galleria Chemica; HSDB). Asphalts are categorised into different classes based on performance specification, manufacture and use (IARC, 2013). There are currently six classes of asphalt, with the main types in use being hard-grade straight run, oxidised and cutback asphalts. Straight-run asphalts are mixed with mineral aggregates and are used in road-paving, generally at temperatures from 110 to 160 °C. Oxidised asphalts are used in hot-roofing applications (180–230 °C). Mastic asphalt is a type of straight-run asphalt used in specialised applications at higher temperatures (200–250 °C). Mastic asphalts are used in Europe, but are rarely used in the USA and Canada (IARC, 2013). Gilsonite is similar to a hard petroleum asphalt and has a softening point between 115 and 190°C (The Shell Bitumen Handbook).

A survey of asphalt producers in seven European countries showed that more than 95 % of the asphalt chemicals produced were covered by three CAS Nos; asphalt (CAS No. 8052-42-4), residues, petroleum, vacuum (CAS No. 64741-56-6) and oxidized asphalt (CAS No. 64742-93-4) (Asphalt Institute 2011). Asphalt (CAS No. 8052-42-4) and oxidized asphalt (CAS No. 64742-93-4) are reported to represent >99 % of asphalt material used in the United States of America (USA).

In addition to the commercial applications described above, the chemicals with CAS Nos 8052-42-4, 12002-43-6, 64742-16-1, 64742-85-4 and 64742-93-4 have reported commercial uses including:

- as absorbents;
- as fuel additives;
- as impregnation materials;
- as lubricants and additives;
- in processing for rubber production; and
- as reprographic agents.

Domestic uses have been identified for chemicals with CAS Nos 8052-42-4, 12002-43-6, 64742-16-1, 64742-85-4 and 64742-93-4.

Asphalt (CAS No. 8052-42-4) and oxidized asphalt (CAS No. 64742-93-4) have been reported as present in numerous consumer products at concentrations up to 75 % while gilsonite (CAS No 12002-43-6) may be present as binder additive. These products include:

- roof coatings, cements and adhesives;
- insulation batts; and
- driveway fillers and sealers (US Dept. of Health and Human Services).

The chemicals with CAS Nos 64742-16-1 and 64742-85-4 have reported domestic uses in the Substances and Preparations in the Nordic countries (SPIN) database including:

- as adhesives, binding agents;

- as corrosion inhibitors;
- as fillers;
- in insulating materials; and
- in paints, lacquers and varnishes.

However, it should be noted that SPIN does not distinguish between direct use of the chemicals, or use of the materials that are produced from chemical reactions involving the chemical.

The chemical residues, petroleum, hydrodesulfurized vacuum (CAS No. 64742-85-4) has a reported domestic use in consumer coating products.

Raffinates, petroleum, residual oil decarbonization (CAS No. 64742-07-0) has no identified uses.

Asphalt (CAS No. 8052-42-4) and the chemical with CAS No. 64742-85-4 have reported site-limited use as intermediates.

Asphalt (CAS No. 8052-42-4) and gilsonite (CAS No. 12002-43-6) has have reported non-industrial uses in non-agricultural pesticides and preservatives.

Restrictions

Australian

No known restrictions have been identified.

International

No known restrictions have been identified.

Existing Worker Health and Safety Controls

Hazard Classification

The chemicals are not listed on the Hazardous Chemicals Information System (HCIS) (Safe Work Australia).

Exposure Standards

Australian

Asphalt (CAS No. 8052-42-4), as bitumen fumes, has an occupational exposure limit (OEL) of 5 mg/m^3 time weighted average (TWA).

International

The following exposure standards are identified (Galleria Chemica) for asphalt (CAS No. 8052-42-4):

An OEL of 0.5–10 mg/m^3 TWA and 1.5–10 mg/m^3 short-term exposure limit (STEL) in different countries such as Canada, Chile, China, Germany, Indonesia, Ireland, Malaysia, Mexico, Norway, Singapore, South Africa, Spain, Switzerland, United Kingdom, and USA. The American Conference of Governmental Industrial Hygienists (ACGIH) recommends a threshold limited value (TLV) of 0.5 mg/m^3 (TWA) as benzene-soluble (or equivalent method) inhalable aerosol. 'This value is intended to minimize the potential for mucous membrane and ocular irritation' (ACGIH, 2011).

Health Hazard Information

Due to the uses of the chemicals (which could involve heating), the hazardous properties of two forms (ambient temperature and fumes produced in use) of the chemicals are evaluated. Heated asphalts release vapours that condense upon cooling, termed asphalt or bitumen fume. Asphalt fume refers only to the aerosolised fraction of total emissions (i.e. solid particulate matter, condensed vapour, and liquid bitumen droplets). Asphalt fume comprises a complex mixture of constituents, which is strongly dependent on how the fume is generated and contains higher concentrations of the volatile components present in asphalt that are toxicologically distinct from the bulk asphalt. The temperature of fume generation affects the chemical composition, including levels of PACs in the fume. Residues of two- to seven-ring PACs are found in both solid asphalts and asphalt fume. The fumes tend to contain proportionally more two-ring PACs (such as naphthalene), and less five-ring PACs (such as benzo[*a*]pyrene), than solid asphalts.

To understand the potential hazard of asphalt fume, many of the toxicological studies have used experimental systems that attempt to reproduce asphalt fume similar to occupational inhalation exposure, or condense generated fumes in a form for oral or dermal exposure to test animals.

Toxicokinetics

The chemicals are complex mixtures; as such, the pharmacokinetic behaviour will be dependent on the properties of the individual constituents. Therefore, it is considered inappropriate to generalise the extent of the chemicals' absorption, distribution and metabolism. The statements below, regarding the expected behaviour of the chemicals, are based on the properties of the bulk material; however, the presence of other chemicals in the mixture (expected to be of low concentration) for which these statements do not hold, cannot be ruled out.

The chemicals are not expected to be absorbed dermally, due to their high molecular weight and large molecular size, low water solubility and negligible vapour pressure.

Long chain aliphatic hydrocarbons are a major component of asphalt. Following inhalation of such chemicals, hydrocarbons of 9–16 carbon atoms were found in the blood, brain, liver, kidneys and fat of rats. Aerosols of hydrocarbons with >16 carbon atoms were found in the lung and liver in mice. They are expected to be oxidatively metabolised and slowly eliminated in the urine and faeces (IARC, 2013). PACs are also a component of the chemicals (at ppm levels), or may be present at higher concentrations in fumes generated during use. Following inhalation, ingestion, or skin contact, they are expected to be metabolised and subsequently eliminated by urinary or biliary excretion. Whole body studies in rodents have also demonstrated detectable levels of polycyclic aromatic hydrocarbons in the majority of internal organs (IARC, 2013).

Acute Toxicity

Oral

Based on the data available, the chemicals in this group have low acute toxicity based on results from animal tests following oral exposure to residues, petroleum, vacuum (CAS No. 64741-56-6). The median lethal dose (LD50) in rats is >5000 mg/kg bw. Observed sub-lethal effects included hypoactivity and diarrhoea (US EPA, 2006).

Dermal

Based on the data available, the chemicals in this group have low acute toxicity based on results from animal tests following dermal exposure to residues, petroleum, vacuum (CAS No. 64741-56-6). The LD50 value in rats is >2000 mg/kg bw.

Observed sub-lethal effects included hypoactivity and diarrhoea (US EPA, 2006).

Inhalation

Based on the data available, the chemicals in this group have low acute toxicity following inhalation exposure.

No mortality or significant signs of toxicity were noted in rats exposed to fumes generated from condensates collected from the headspace of a bitumen storage unit. Mean exposures were estimated to be 182 mg/m^3 for four hours (US EPA, 2006). No mortality or toxic effects have been reported in several other studies in which rats were repeatedly exposed up to 300 mg/m^3 (API Petroleum HPV Testing Group, 2009).

The chemicals may contain trace quantities of hydrogen sulfide (CAS No. 7783-06-4) which can be emitted in uses involving heating or can build up in the headspace of storage or transport containers (US EPA 2006; API Petroleum HPV Testing Group, 2009). Hydrogen sulfide is classified as hazardous with hazard category 'Acute Toxicity Category 2' and hazard statement 'Fatal if inhaled' (H330) in the HCIS (Safe Work Australia). Hydrogen sulfide may be released from asphalts at elevated temperatures.

Corrosion / Irritation

Skin Irritation

Based on the available data, the chemicals in this group may slightly irritate skin in animal studies, particularly following repeated exposure. The effects were not sufficient to warrant a hazard classification.

Studies were performed similarly to OECD Test Guideline (TG) 404. Residues, petroleum, vacuum (CAS No. 64741-56-6) applied to intact rabbit skin produced slight erythema and oedema (mean scores for 24 and 72 hours were both 0.1). After 14 days, oedema had resolved, but the mean erythema score was 0.8 (REACHa).

Eye Irritation

Based on the available data, the chemicals in this group may be, at most, slightly irritating to the eye in animal studies. The effects were not sufficient to warrant a hazard classification.

In two studies, vacuum residuum (CAS No. not stated but likely 64741-56-6) was reported to not irritate the eyes of rabbits when tested according to OECD TG 405. The mean primary eye irritation scores (out of 110) at 1 h, 24 h, 48 h, 72 h and 7 days were given as 1, 4.7, 1.8, 0 and 0 (study 1), or 2, 4, 4.2, 1.8 and 0 (study 2), respectively. The effects were reported to be reversible within 72 hours after application to the eyes of rabbits, in these studies (REACHa).

Exposure to asphalt vapours was reported to cause only minor, transient conjunctivitis in the eyes of rabbits (IPCS, 2004).

Observation in humans

Irritant effects (skin, eye, nasal and throat) and respiratory effects have been reported among workers in various sectors of the asphalt industry. The effects were typically mild in severity and transitory in nature. Lower respiratory tract effects including wheezing, coughing, shortness of breath and changes in pulmonary functions have also been reported. There are insufficient data available to determine the relationship between exposure to asphalt fumes and the reported health effects due to other potential confounding exposures (e.g. diesel exhaust and road dust) (IPCS, 2004; IARC, 2013).

Severe burns to the skin have been reported in workers from hot asphalt (usually used at temperatures from 150 to 190 °C). Additionally, it can harden and adhere to skin upon contact, retaining sufficient heat to continue to cause damage at the affected area (IPCS, 2004; IARC, 2013).

Sensitisation

Skin Sensitisation

The negative results observed for residues, petroleum, vacuum (CAS No. 64741-56-6), in several skin sensitisation animal studies conducted in accordance with OECD TG 406 (Buehler test), support a conclusion that the chemicals in this group are not skin sensitisers. There was no dermal irritation observed in the test groups following the challenge application with the undiluted chemical (REACHa).

Repeated Dose Toxicity

Oral

There are limited data available. The most likely route of human exposure to the chemicals in this group is by the dermal or inhalation routes.

In a 71-day study in pigs, reported in ACGIH (2011), two bitumens (CAS No. not stated) were not harmful following repeated exposure by the oral route.

Dermal

Based on the data available, the chemicals in this group are not considered to cause serious damage to health from repeated dermal exposure.

In a GLP-compliant study conducted similarly to OECD TG 410, residues, petroleum, vacuum (CAS No. 64741-56-6) was administered at dosages of 200, 1000, or 2000 mg/kg bw three times a week for four weeks. Clinical observations included slight oedema, flaking skin, wheezing and decreased food-intake (qualitative observation), resulting in reduced body weight gain in all dose groups when compared to controls. There were statistically significant reduced body weight gains in males in the high-dose group. There were no significant changes in clinical chemistry, haematology parameters or reproductive organs reported. A no observed adverse effect level (NOAEL) for local effects of 200 mg/kg bw/day was reported based on dermal irritation. A NOAEL for systemic effects of 1000 mg/kg bw/day was reported based on decreased body weight (which was considered to be secondary to the reduced food intake) (US EPA, 2006; REACHa).

Inhalation

Based on the data available, the chemicals in this group are not considered to cause serious damage to health from repeated inhalation exposure.

The fume condensate from oxidized asphalt (CAS No. 64742-93-4) was tested in rats in a combined repeated dose and reproductive and developmental screening test conducted in accordance with OECD TG 474. Wistar rats were exposed (nose-only) to concentrations of approximately 30, 100 or 300 mg/m^3 for 28 days. A no observed adverse effect concentration (NOAEC) was established as 100 mg/m^3 based on slight histopathological changes observed in the lungs observed at the highest dose (API Petroleum HPV Testing Group, 2009).

Asphalt fume condensate collected over a paving asphalt tank was tested in a repeated dose inhalation study conducted in accordance with OECD TG 413. Wistar rats were exposed (nose-only) to concentrations of approximately 5, 28 or 149 mg/m^3 for 90 days. The NOAEC was established as 28 mg/m^3 based on reduced body weights and histopathological changes in the nasal and paranasal cavities observed at the highest dose (US EPA, 2006; API Petroleum HPV Testing Group, 2009).

Similar irritant effects were observed in a 104-week study with bitumen fume condensate conducted in accordance with OECD TG 451 (see **Carcinogenicity** section).

Genotoxicity

Based on the weight of evidence, the chemicals in this group (as whole materials) are not considered to be mutagenic. Asphalt fume condensates are mutagenic, with the level of mutagenic activity related to the temperature at which they are generated and levels of PACs (US EPA, 2006; IARC, 2013; REACHa).

Asphalts (as whole materials)

In modified Ames tests using asphalts or dimethylsulfoxide (DMSO) extracts of roofing and paving asphalts, there was either no mutagenic activity, or weak mutagenic activity (only with metabolic activation) in *Salmonella typhimurium* (US EPA, 2006).

In an Ames tests, gilsonite was not mutagenic in *S typhimurium* strains TA100, TA 1535, TA97, TA1537, or TA98 with or without metabolic activation (CCRIS).

In a mammalian cell mutation study, DMSO extracts of residues, petroleum, vacuum (CAS No. 64741-56-6) were not mutagenic in mouse lymphoma cells without metabolic activation, but were weakly mutagenic with metabolic activation (US EPA, 2006; REACHa).

In a chromosomal aberration study, SD rats were exposed by oral gavage to vacuum residuum (CAS No. not stated) samples, at doses of 0, 0.3, 1.0 or 3.0 g/kg bw/day for 5 days. There were no chromosomal abnormalities in rat bone marrow cells observed under the conditions of the test (US EPA, 2006).

Asphalt fume condensates

Both positive and negative results have been reported in modified Ames tests with asphalt fume condensates. In general, increased mutagenicity was observed in fumes generated at higher temperatures, with levels reported to correlate with PAC content; in particular, with three to seven ring PACs. In modified Ames tests, laboratory generated fumes at 149 °C or 316 °C, were mutagenic, with the sample generated at 316 °C more mutagenic than that generated at 149 °C. Field samples tested were only weakly mutagenic. Fume condensate from the predominant grades of paving asphalt and roofing asphalt in the USA, generated under realistic end-use temperatures, showed minimal mutagenicity in the modified Ames test (US EPA, 2006; IARC, 2013).

In chromosomal aberration studies, condensates of asphalt fumes, generated in the field and in the laboratory, were negative in Chinese hamster ovary cells. However, fumes generated at 316 °C caused a dose related increase in micronucleus formation in Chinese hamster lung fibroblasts (V79) cells. Field samples of paving and stone mastic fumes, generated at up to 165 °C, induced micronuclei in BEAS 2B human bronchial epithelial cells (US EPA, 2006; IARC, 2013).

Asphalt fume condensates have been reported to induce DNA adducts in vitro in calf thymus DNA, with levels reported to correlate with bacterial mutagenicity (US EPA, 2006; IARC, 2013).

In in vivo studies in rats and Big Blue mice, no significant genotoxic effects were observed following exposure to asphalt fumes (generated at 170 °C) at a dose of 100 mg/m^3 for 6 h/day for five days (nose only inhalation). In various studies in rats, with differing exposure conditions to asphalt fumes, micronucleus formation in the bone marrow was not observed. However, DNA adducts in the lungs and nasal cavities of rats have been observed following inhalation exposure and DNA adducts in the skin, lung and lymphocytes have been observed in rats and mice following dermal application of asphalt fume condensates (US EPA, 2006; IARC, 2013).

In studies in humans, workers exposed to asphalt fumes were reported to have mutagenic urine (measured by Ames test), DNA damage, unidentified DNA adducts, sister chromatid exchange, micronucleus formation and chromosomal aberrations (IARC, 2013).

Carcinogenicity

Based on the available data, the chemicals in this group as whole materials are not considered carcinogenic, although dilution in organic solvents may produce some carcinogenic effects following prolonged dermal exposure. Exposure to asphalt emissions during certain occupations has been linked to increased risks of carcinogenicity.

There are comprehensive summaries of carcinogenicity studies in animals (including mice, rats, guinea pigs or rabbits) available. However, most of the animal studies (including exposure routes such as skin application, subcutaneous injection, intramuscular injection and inhalation), were considered inadequate (including all studies conducted before 1980) by the International Agency for Research on Cancer (IARC). The studies were considered to be inadequate for assessment on the basis of poor study design, poor reporting, no information of controls, limited or no histopathology, or information on survival. In some studies a carcinogenic chemical was used as the vehicle.

Asphalts (as whole materials)

In skin painting studies in mice, considered adequate by IARC, treatment with a sample of 'air refined petroleum asphalt' in toluene resulted in an increase in malignant tumours. However, negative results were reported in three studies using asphalt or oxidised asphalt (IARC, 2013).

IARC concluded that there was 'inadequate evidence in experimental animals for the carcinogenicity of straight run bitumens' and 'limited evidence in experimental animals for the carcinogenicity of oxidized bitumens' (IARC, 2013).

Asphalt fume condensates

Increased incidences of skin tumours were observed in six skin painting studies (considered adequate by IARC), with oxidised asphalt fume condensates generated at temperatures ranging from 199 to 316 °C. These samples have been demonstrated to be compositionally different to fumes generated in the field (US EPA, 2006)

The IARC considered that on the basis of the weight of evidence from studies in experimental animals, it was highly probable that a mechanism involving genotoxicity is responsible for the tumourigenic effects of exposure to bitumen in mouse skin.

In a 104-week inhalation study conducted in accordance with OECD TG 451, Wistar rats were exposed to bitumen fume condensate (derived from the headspace of a storage tank containing semi-blown paving bitumen) at concentrations of 0, 6.8, 34.4 or 172.5 mg/m^3 for 6 h/day, 5 days/week. No significant increase in organ tumours was observed. Histopathology of the respiratory tract in animals after 7 days, 90 days and 12 months, showed slight irritation in the nasal and paranasal cavities (basal cell hyperplasia), larynx (multifocal mucosal mononuclear cell infiltration) and lungs (bronchiolo-alveolar hyperplasia and alveolar histiocytosis) (API Petroleum HPV Testing Group, 2009; REACHa)

IARC concluded that there was 'inadequate evidence in experimental animals for the carcinogenicity of fumes of straight run bitumens' but 'sufficient evidence in experimental animals for the carcinogenicity of fumes of oxidized bitumens' (IARC, 2013).

The carcinogenicity concerns for asphalts are focussed on their use at higher temperatures where fumes which contain PACs may be emitted (such as during road paving and roofing). The IARC report examined a substantial number of cohort and case-control studies in humans to evaluate the cancer incidence, or mortality attributable to exposure to asphalts or asphalt emissions (IARC, 2013).

The case-control studies examined cohorts with specific cancer types, such as cancer of the lung or kidney. There were several meta-analyses included on the epidemiological risk of cancer among asphalt workers. The IARC multicentre cohort study (the largest amongst the cohort studies reported), included quantitative exposure assessment of both inhalation and dermal exposure, incidence and mortality, and adjustment for a range of confounding factors (such as smoking, coal-tar and/or silica exposure) and type of asphalt use (such as paving or roofing). Additionally, the results from genotoxicity studies of workers examining urinary mutagenicity, gene and protein expression, DNA damage, micronucleus formation, chromosomal aberrations were considered.

There was limited evidence of carcinogenicity in humans for occupational exposures to asphalt emissions during roofing and mastic-asphalt work. A positive association was reported for cancers of the lung and upper aerodigestive tract, although confounding factors could not be completely ruled out. There was inadequate evidence of carcinogenicity in humans for occupational exposure to asphalt emissions during road paving (IARC, 2013).

Overall, IARC determined that:

- oxidised asphalts and their emissions during roofing were 'Probably carcinogenic to humans' (Group 2A).
- hard asphalts and their emissions during mastic-asphalt work were 'Possibly carcinogenic to humans' (Group 2B).
- straight-run asphalts and their emissions during road paving were 'Possibly carcinogenic to humans' (Group 2B).

In making the overall evaluation for occupational exposures to asphalts and their emissions during road paving, the IARC Working Group considered the following mechanistic results and other relevant data from independent studies in exposed workers. There were increased levels of DNA damage, sister-chromatid exchange, micronucleus formation and chromosomal aberration. 'Many of these events are known to be associated with human neoplasia. In addition, data from in vitro and in vivo animal studies support these findings' (IARC, 2013).

The absence of a carcinogenic effect for emissions from road paving is supported by results from fluorescence spectroscopy which has been reported to be used as a predictor of carcinogenicity for asphalt fumes. Fluorescence emission levels were reported to be below the value that corresponds to a minimal carcinogenic effect (US EPA, 2006).

Reproductive and Developmental Toxicity

Certain petroleum stream chemicals have been shown to be developmentally toxic by the dermal route of exposure. Effects include increased incidence of early and total resorptions and decrease in foetal body weight (IPIECA 2010; Murray et al. 2013). Similar embryotoxic effects have been described in laboratory animals exposed to PAC such as benz[*a*]anthracene, benzo[*a*]pyrene, and naphthalene (IPCS, 1998).

There are no reproductive or developmental toxicity studies available on asphalt or asphalt fumes. Based on the limited data available, and the low concentration of PACs generated in asphalt fumes, a classification for reproductive or developmental effects is not warranted (API Petroleum HPV Testing Group, 2006; REACH).

In a GLP-compliant two-generation reproduction toxicity study conducted in accordance with OECD TG 416, rats were exposed (oral gavage) to the analogue chemical, distillates (Fischer-Tropsch), heavy, C18-50- branched, cyclic and linear (CAS No. 848301-69-9) at dosages of 0, 50, 250 or 1000 mg/kg bw/day. The analogue chemical is mainly comprised of saturated oil components, which may be found in asphalts (REACHa).

There were histopathological lesions in the lungs (chronic interstitial/alveolar inflammation) of the F0- and F1-generations. There were corresponding macroscopic findings and/or increased lung weights, and effects in the kidneys (renal tubular hyaline droplets likely associated with alpha-2μ-globulin) of the F1 males only. The study authors stated that the lung lesions were most likely secondary to aspiration of the chemical and, therefore, not relevant for human risk assessment. The renal effects are specific to male rats. These are induced by hydrocarbons and have no relevance for humans. An equivocal, nonadverse slight decrease in F2 pup brain weights was reported. A NOAEL of 1000 mg/kg bw/day was determined for reproductive and systemic toxicity, based on no adverse effects on the male and female reproductive systems, non-reproductive tissues, and other parameters (such as body weight, feed consumption, and clinical observations).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation relate to the use of the chemicals at elevated temperatures. Fumes from asphalts have been associated with carcinogenicity and mutagenicity in humans and animals. There is considered to be an increased risk for fumes containing higher levels of PACs. The levels of PACs are affected by the temperature of fume generation. Exposure to asphalt fumes could also cause irritant effects (skin, eye, nasal and throat) and respiratory effects. Severe burns to the skin have been reported in workers from hot asphalt (usually used at temperatures from 150 to 190 °C).

The impurity hydrogen sulfide (CAS No. 7783-06-4) may be released from asphalts at elevated temperatures, where it may accumulate in confined spaces and reach hazardous levels. Inhalation exposure to hydrogen sulfide may cause eye, nose and throat irritation, nausea, headaches and, in extreme cases, may be fatal.

Public Risk Characterisation

The general public could be exposed through the skin or by inhalation when using domestic products containing the chemical. Based on a review of representative safety data sheets for domestic products identified in the USA (US Dept. of Health and Human Services), application of these products does not involve heating. Therefore, it is considered that the public will not be exposed to the chemicals at elevated temperatures where the hazards of the chemicals are increased (from exposure to fumes). In addition, although there is potential for public exposure to fumes during road paving, this is expected to be for short durations and infrequent. Hence, the public risk from the chemicals in this group is not considered to be unreasonable.

Occupational Risk Characterisation

Given the critical systemic long-term and local health effects, the chemicals during use at elevated temperatures could pose an unreasonable risk to workers unless adequate control measures to minimise dermal and inhalation exposure are implemented.

Time-trend data (mainly available for road paving) of exposure to asphalt fume have shown a decrease by a factor of 2–3 each decade since 1970. In the USA and Europe, the introduction of warm-mix asphalts with a lower application temperature (100–140 °C) is expected to further lower exposure to asphalt fume.

Particular concerns exist for workers during mastic-asphalt application and for roofers applying hot asphalt, as these occupations involve higher application temperatures compared with paving, and have been associated with carcinogenicity (IARC, 2013). Whilst cold and soft applications have been developed to lower exposure to fumes in roofers, it is not known if these are used in Australia.

Guidance on the interpretation of workplace exposure standards for airborne contaminants advises that 'exposure to carcinogens should be eliminated or minimised so far as is reasonably practicable' (Safe Work Australia, 2013). Given that lower exposure standards appear achievable (based on international exposure standards), Safe Work Australia should consider whether current controls adequately minimise the exposure risk to workers.

NICNAS Recommendation

It is recommended that Safe Work Australia consider whether current controls adequately minimise the risk to workers. A Tier III assessment could be necessary to provide further information to determine whether the current exposure controls offer adequate protection to workers.

The chemicals are not recommended for classification and labelling aligned with the Globally Harmonized System of Classification and Labelling of Chemicals (GHS), as the critical hazard of carcinogenicity is associated with byproducts generated during use of the chemicals under specific conditions, not with the chemical itself. This does not consider classification of physical hazards and environmental hazards.

Regulatory Control

Advice for industry

Control measures

Control measures to minimise the risk from dermal and inhalation exposure to asphalt fumes should be implemented in accordance with the hierarchy of controls. Approaches to minimise risk include substitution, isolation and engineering controls. Measures required to eliminate, or minimise risk arising from storing, handling and using a hazardous chemical depend on the physical form and the manner in which the chemicals are used. Examples of control measures which could minimise the risk include, but are not limited to:

- isolating operations;
- ensure adequate ventilation and work upwind to prevent the chemicals from entering the breathing zone of any worker;
- health monitoring for any worker who is at risk of exposure to the chemicals, if valid techniques are available to monitor the effect on the worker's health;
- air monitoring to ensure control measures in place are working effectively and continue to do so: and
- minimising manual processes and work tasks through automating processes.

Guidance on managing risks from hazardous chemicals are provided in the *Managing risks of hazardous chemicals in the workplace—Code of practice* available on the Safe Work Australia website.

Personal protective equipment should not solely be relied upon to control risk and should only be used when all other reasonably practicable control measures do not eliminate or sufficiently minimise risk. Guidance in selecting personal protective equipment can be obtained from Australian, Australian/New Zealand or other approved standards.

Obligations under workplace health and safety legislation

Information in this report should be taken into account to help meet obligations under workplace health and safety legislation as adopted by the relevant state or territory. This includes, but is not limited to:

- ensuring that hazardous chemicals are correctly classified and labelled;
- ensuring that (material) safety data sheets ((M)SDS) containing accurate information about the hazards (relating to both health hazards and physicochemical (physical) hazards) of the chemicals are prepared; and
- managing risks arising from storing, handling and using a hazardous chemical.

Your work health and safety regulator should be contacted for information on the work health and safety laws in your jurisdiction.

Information on how to prepare an (M)SDS and how to label containers of hazardous chemicals are provided in relevant codes of practice such as the *Preparation of safety data sheets for hazardous chemicals—Code of practice* and *Labelling of workplace hazardous chemicals—Code of practice*, respectively. These codes of practice are available from the Safe Work Australia website.

A review of the physical hazards of these chemicals has not been undertaken as part of this assessment.

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Last Update 29 June 2018

Chemical Identities

Chemical Name in the Inventory and Synonyms	**Asphalt** asphaltum bitumen mineral pitch
CAS Number	8052-42-4
Structural Formula	

	No Structural Diagram Available
Molecular Formula	Unspecified
Molecular Weight	Unspecified

Chemical Name in the Inventory and Synonyms	**Gilsonite** asphalt, gilsonite bitumens, gilsonite rubber, mineral
CAS Number	12002-43-6
Structural Formula	No Structural Diagram Available
Molecular Formula	Unspecified
Molecular Weight	Unspecified

Chemical Name in the Inventory and Synonyms	**Residues, petroleum, vacuum** vacuum residue (petroleum) vacuum residuum (petroleum)

CAS Number	64741-56-6
Structural Formula	No Structural Diagram Available
Molecular Formula	Unspecified
Molecular Weight	Unspecified

Chemical Name in the Inventory and Synonyms	**Raffinates, petroleum, residual oil decarbonization** residual oil, decarbonization raffinate
CAS Number	64742-07-0
Structural Formula	No Structural Diagram Available
Molecular Formula	Unspecified
Molecular Weight	Unspecified

Chemical Name in the	**Petroleum resins**

Inventory and Synonyms	hydrocarbon resins neopolymer (petroleum resin)
CAS Number	64742-16-1
Structural Formula	No Structural Diagram Available
Molecular Formula	Unspecified
Molecular Weight	Unspecified

Chemical Name in the Inventory and Synonyms	**Residues, petroleum, hydrodesulfurized vacuum** hydrodesulfurized vacuum residuum (petroleum)
CAS Number	64742-85-4
Structural Formula	No Structural Diagram Available
Molecular Formula	Unspecified
Molecular Weight	Unspecified

Chemical Name in the Inventory and Synonyms	**Asphalt, oxidized** asphalt, petroleum, oxidized blown asphalt
CAS Number	64742-93-4
Structural Formula	No Structural Diagram Available
Molecular Formula	Unspecified
Molecular Weight	Unspecified

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Draft Screening Assessment

Assessment of Select Hydrocarbon-based Substances

Environment and Climate Change Canada
Health Canada

January 2022

Synopsis

Pursuant to section 68 or 74 of the *Canadian Environmental Protection Act, 1999* (CEPA), the Minister of the Environment and the Minister of Health have conducted a draft screening assessment of eight hydrocarbon-based substances. These substances were identified as priorities for assessment as they met categorization criteria under subsection 73(1) of CEPA or were considered a priority on the basis of other human health concerns. The Chemical Abstracts Service Registry Numbers (CAS RN[1]), their *Domestic Substances List* (DSL) names, and their common names used throughout the assessment are listed in the table below.

The eight substances assessed in this screening assessment

CAS RN	**DSL name**	**Simplified or common names**
64742-16-1[a]	Petroleum resins	Petroleum resins
68131-77-1 [a, b]	Distillates (petroleum), steam-cracked, polymd.	Hydrocarbon resin
68410-13-9 [a]	Distillates (petroleum), steam-cracked, C_{5-12} fraction, polymd.	Polymerized C_{5-12} distillates
67891-82-1 [a]	Hydrocarbon waxes (petroleum), oxidized, compds. with ethanolamine	Oxidized hydrocarbon waxes with EA
97862-84-5 [a]	Hydrocarbon waxes (petroleum), oxidized, compds. with 2-(methylamino)ethanol	Oxidized hydrocarbon waxes with 2-MAE
68425-94-5 [a]	Residues (petroleum), catalytic reformer fractionator, sulfonated, polymers with formaldehyde, sodium salts	Alkylated naphthalene sulfonate sodium salt polymers with formaldehyde
68526-82-9 [a]	Alkenes, C6-10, hydroformylation products, high-boiling	Heavy oxo ends
68815-10-1 [a]	Petroleum, sulfurized	Sulfurized petroleum

[a] All CAS RNs are UVCB substances (substances of unknown or variable composition, complex reaction products, or biological materials).

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

Petroleum resins is used in asphalt, adhesives and sealants, lubricants and greases, and polishes and waxes. Petroleum resins appears in some cosmetics and natural

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health products as an adhesive. Hydrocarbon resin is used mostly in household and construction adhesive products. The use of these resin substances as adhesives and resin components is expected to have low exposure potential to the environment. Polymerized C_{5-12} distillates, which is also a resin, is an intermediate petrochemical substance that is unlikely to leave petrochemical facilities without further processing, and no uses in products available to consumers in Canada have been identified for this substance. Based on experimental data for petroleum resins, and given their very low water solubility, these substances are also expected to have low ecotoxicity and have low hazard potential to the environment. These three resins have low human health hazard potential based on their high molecular weight (500 to 2000 Daltons), and low volatility. In addition, dermal exposure to the two resin substances, following their specialized uses in high melt adhesives, is not expected to lead to systemic exposure to humans. The use of resin substances as components of asphalt is considered to have been addressed through the Asphalt and Oxidized Asphalt screening assessment, which concluded that these asphalt and oxidized asphalt substances have low potential of concern for the environmental and human health. Therefore, petroleum resins, hydrocarbon resin, and polymerized C_{5-12} distillates are unlikely to be causing harm to the environment, and the potential risk to human health from these substances is considered to be low.

Oxidized hydrocarbon waxes with EA and oxidized hydrocarbon waxes with 2-MAE are made up of oxidized petrolatum and alkanolamine constituents. Based on the available information, oxidized hydrocarbon waxes with EA and oxidized hydrocarbon waxes with 2-MAE are not expected to be in use in Canada. The constituents of these UVCB substances have been assessed previously through the Petrolatum and Waxes and the Alkanolamines and Fatty Alkanolamides Group screening assessments and were concluded not to meet any criteria under section 64 of CEPA. Available data also suggests that these substances are of low toxicity and low bioavailability. Therefore, oxidized hydrocarbon waxes with EA and oxidized hydrocarbon waxes with 2-MAE are unlikely to be causing harm to the environment, and the potential risk to human health from these substances is considered to be low.

Alkylated naphthalene sulfonate sodium salt polymers with formaldehyde is used as a formulant in pest control products (which is addressed under the *Pest Control Products Act*), and industrially as a wetting and penetrating agent. The industrial use of this substance is not expected to lead to environmental exposure, and its ecological hazard is expected to be low. This substance is absent from products available to consumers, and the general population is not expected to be exposed to this substance through environmental media or food. Therefore, alkylated naphthalene sulfonate sodium salt is unlikely to be causing harm to the environment and the potential risk to human health from this substance is considered to be low.

Heavy oxo ends is used as an industrial de-foamer and is not present in products available to consumers. Exposure of the general population in Canada is not expected. Heavy oxo ends are expected to be of low ecological risk based on no effect at high loading rates and limited environmental exposure. On the basis of these considerations, heavy oxo ends is unlikely to be causing harm to the environment and the potential risk to human health from this substance is considered to be low.

Sulfurized petroleum has industrial uses as a metal-working agent in industrial cutting oils where the sulfur additive performs an anti-wear function in the oil. Used metal-working fluids are disposed of as “used oil” and are subject to the Code of Practice for Used Oil Management in Canada. No uses of this substance in products available to consumers were identified. On the basis of these considerations, exposure for the general population to this substance is not expected. Based on modelled data, sulfurized petroleum is expected to have a relatively low toxicity and low bioavailability. Sulfurized petroleum is unlikely to be causing harm to the environment and the potential risk to human health is considered to be low.

Considering all available lines of evidence presented in this draft screening assessment, there is low risk of harm to the environment from the eight hydrocarbon-based substances in this assessment. It is proposed to conclude that the eight hydrocarbon-based substances in this assessment do not meet the criteria under paragraphs 64(*a*) or (*b*) of CEPA as they are not entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity or that constitute or may constitute a danger to the environment on which life depends.

On the basis of the information presented in this draft screening assessment, it is proposed to conclude that the eight hydrocarbon-based substances in this assessment do not meet the criteria under paragraph 64(*c*) of CEPA as they are not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

It is therefore proposed to conclude that the eight hydrocarbon-based substances assessed in this assessment do not meet any of the criteria set out in section 64 of CEPA.

This report also identifies 35 hydrocarbon-based substances (identified in Table A-1 of Appendix A) for which risk assessment activities are considered to have already taken place under CEPA. These substances were identified as priorities for assessment as they met categorization criteria under subsection 73(1) of CEPA or were considered a priority on the basis of other human health concerns. Based on their composition, physical-chemical properties and reported uses, these additional 35 substances were considered to fall within hydrocarbon-based groups that have previously been addressed under CEPA. These substances include 31 low boiling point naphthas; 2 natural gases; 2 substances that align with coal tars and their distillates [pitch and light oil (coal) coke-oven]. Identified uses of these 35 substances are not expected to result in exposures beyond those already considered in previous assessments. Since these substances are not expected to contribute additional concerns to human health or the environment beyond those that have already been identified in past assessments of similar substances, they will not undergo further assessment at this time. In addition, existing or future risk management actions related to the previous assessments, where applicable, are expected to address the risks from these substances.

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1. Introduction

Pursuant to section 68 or 74 of the *Canadian Environmental Protection Act, 1999* (CEPA) (Canada 1999), the Minister of the Environment and the Minister of Health have conducted a draft screening assessment of eight hydrocarbon-based substances to determine whether they present or may present a risk to the environment or to human health. These eight substances were identified as priorities for assessment as they met categorization criteria under subsection 73(1) of CEPA or were considered a priority on the basis of other human health concerns (ECCC, HC [modified 2017]).

The substances addressed in this report are considered to be of Unknown or Variable composition, Complex reaction products, or Biological materials (UVCBs) [2]. This report has two parts. The eight hydrocarbon-based substances being assessed were divided into five subgroups based on their physical and chemical properties and their risk is characterized in five separate sections (Sections 2 to 6).

The second part of this report (Section 7) includes an additional 35 hydrocarbon-based substances (see Table A-1, Appendix A) for which risk assessment activities can be considered as having already taken place under CEPA. These 35 substances were identified as priorities for assessment as they met categorization criteria under subsection 73(1) of CEPA or were considered a priority on the basis of other human health concerns (ECCC, HC [modified 2017]). In addition, proposed or future risk management actions, when applicable, stemming from previous assessments would also be expected to address some of these substances. As such, these 35 substances will not undergo further risk assessment at this time. The identification of, and proposed actions for these 35 hydrocarbon-based substances for which risk assessment activities can be considered as having already taken place follows on previous activities (Environment Canada, Health Canada 2015; ECCC, HC 2017a) where various substances on the *Domestic Substances List* (DSL) were identified as being associated with other risk assessment activities under CEPA and, as a result, were identified as not requiring further risk assessment at that time.

This draft screening assessment includes consideration of information on chemical properties, environmental fate, hazards, uses and exposures, including additional information submitted by stakeholders. Relevant data were identified up to December 2019. Targeted literature searches were conducted up to December 2019. When

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[2] 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.

available and relevant, information presented in assessments from other jurisdictions was considered.

This draft screening assessment was prepared by staff in the CEPA Risk Assessment Program at Health Canada and Environment and Climate Change Canada. The ecological and human health portions of this assessment have undergone external written review and/or consultation. Comments were received from Mr. Geoff Granville (GCGranville Consulting Corp), Dr. Connie Gaudet, Ms. Theresa Lopez, Ms. Jennifer Flippin, and Dr. Joan Garey (the latter three from Tetra Tech). While external comments were taken into consideration, the final content and outcome of the screening assessment remain the responsibility of Health Canada and Environment and Climate Change Canada.

This draft screening assessment focuses on information critical to determining whether substances meet the criteria as set out in section 64 of CEPA by examining scientific information and incorporating a weight of evidence approach and precaution.[3] This draft screening assessment presents the critical information and considerations on which the conclusions are based.

2. Petroleum resins, hydrocarbon resin, polymerized C_{5-12} distillates (CAS RN[4]s 64742-16-1, 68131-77-1, 68410-13-9)

2.1 Substance identity and physical and chemical properties

Petroleum resins (CAS RN 64742-16-1) is a complex combination of organic compounds, predominantly hydrocarbons, obtained as a fraction of the solvent extract of residuum, which is derived from the vacuum distillation of petroleum (API 2017). Petroleum resins is considered within the United States Environmental Protection Agency (US EPA) asphalt category and is described as predominantly comprised of high molecular weight compounds with high carbon-to-hydrogen ratios and a carbon

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

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number greater than 25 (US EPA 2011a, 2011b). Petroleum resins has a boiling point greater than 482°C (US EPA 2011a, 2011b; ECHA 2019a) and is primarily identified as a carbon chain polymer material. Its components have molecular weights in the range of 500 to 2000 Daltons. It is an inert viscous, semi-solid to solid substance, with low water solubility (less than 10^{-5} mg/L), and low vapour pressure (3×10^{-7} mm Hg ($4x10^{-5}$ Pa) or less) (US EPA 2011a, 2011b, Zohuriaan-Mehr and Omidian 2000). At 20°C, this substance is a solid in raw form with a density of 0.9 to 1.1 g/cm^3 and has a softening point between 90 and 100°C (MSDS 2016d). The water solubility range of components of petroleum resins is between 3.5×10^{-4} and 8.1×10^{-4} mg/L at 20°C and a pH between 6.22 and 7.42 (ECHA 2019a).

Distillates (petroleum), steam-cracked, polymd. (CAS RN 68131-77-1), hereinafter referred to as hydrocarbon resin, is a complex combination of organic compounds prepared from the C5-C9 fraction of petroleum cracking by-products which are pre-treated, polymerized and distilled (Reehua 2018). This substance is solid light yellow flakes or small granular solids and has a softening point between 80 and 105°C (LookChem 2018). The vapour pressure and water solubility of hydrocarbon resin are considered to be negligible (SDS 2017).

Distillates (petroleum), steam-cracked, C_{5-12} fraction, polymd. (CAS RN 68410-13-9), hereinafter referred to as polymerized C_{5-12} distillates, is a complex combination of organic compounds which includes polymers of dicyclopentadiene, polyvinyltoluene, polystyrene, or copolymers with these building units (Reehua 2018).

As these three substances are polymeric and are expected to have similar physical-chemical properties, they are assessed in a single chapter and read across data, based on available data, is used to conclude on their exposure and hazard characterization.

These three substances are considered to be UVCBs.

2.2 Sources and uses

According to information submitted in response to a CEPA section 71 survey, and a voluntary data gathering initiative, uses of petroleum resins in Canada (ECCC 2015, 2016) include in enamels, primers, and automotive protective paint materials and sealants.

Petroleum resins is primarily manufactured in refineries (Levelton 2011). Production quantities for petroleum resins in Canada were obtained through a voluntary data gathering initiative (ECCC 2016), and were reported to be < 1 000 kg per year.

Petroleum resins is either sold in its original form, or is blended with, or processed with other asphaltic materials to produce end-use asphalt products, including speciality products such as pipe coatings, roofing adhesives, some special-use paints, and lubricants (US EPA 2011a). Searches of material safety data sheet (MSDS) databases identified additional potential uses, including use as adhesives and coatings (MSDS

2015a), as an ingredient in asphalt modifiers (MSDS 2016a), and in lubricants (MSDS 2015b).

With regards to non-asphalt related uses, petroleum resins is one of the components of hot melt glues (including glue sticks used by crafters) and tackifiers, in which it is present up to concentrations of 10-60%. Petroleum resins is used in solvent-based construction adhesives in mass fractions of 1-5% (MSDS 2004, 2011, 2016b).

There is no definitive information on the use of petroleum resins in food packaging or as an incidental additive in Canada (personal communication, email from the Foods Directorate, Health Canada (HC) to the Existing Substances Risk Assessment Bureau, HC, dated June 21, 2019; unreferenced). It does not appear in Therapeutic Products as a medicinal or non-medicinal ingredient (personal communication, email from the Therapeutic Products Directorate, HC to the Existing Substances Risk Assessment Bureau, HC, dated March 7, 2019; unreferenced), however, it appears in one licensed natural health product (LNHPD 2018), and in a limited number of cosmetic products as an adhesive in patch products (personal communication, email from the Consumer and Hazardous Products Safety Directorate, HC to the Existing Substances Risk Assessment Bureau, HC, dated March 14, 2019; unreferenced).

According to information submitted in response to a CEPA section 71 survey and a voluntary data gathering initiative, industrial uses of hydrocarbon resin in Canada (ECCC 2015, 2016) include rubber manufacturing, adhesives, paints and sealants, and as a binder for carbon electrodes used in aluminum smelting. Hydrocarbon resin is found in synthetic resin and raw material for manufacturing adhesives (MSDS 2016b), sealants, coatings, printer inks, and rubber products (Levelton 2011; MSDS 2011, 2012a, 2016c), and in concrete curing (MSDS 2013b). When used in solvent-based construction adhesives, they appear in mass fractions of 10-30%.

Hydrocarbon resin can be manufactured outside the petroleum sector, and is used as an intermediary substance in refineries and petrochemical industries (Levelton 2011). Due to the expected low hazard concern, hydrocarbon resin is exempt from reporting in the US EPA *Toxic Substances Control Act* (TSCA) under “Chemical Data Reporting Rule (flag XU) for polymers” (ChemIDplus 2017; US EPA 2019b, 2020).

Hydrocarbon resin may be used in adhesives used in food packaging in Canada but is not expected to have direct food contact (personal communication, email from the Food Directorate, HC to the Existing Substances Risk Assessment Bureau, HC, dated March 25, 2019; unreferenced). Hydrocarbon resin appears in the Natural Health Products Ingredients Database (NHPID) as a non-medicinal ingredient with adhesive use (NHPID 2019), and appears in some licensed natural health products such as wart, corn and callus removers and other foot-use products (LNHPD 2018).

Polymerized C_{5-12} distillates is an intermediate petrochemical substance (Levelton 2011). Polymerized C_{5-12} distillates was not included in surveys issued pursuant to section 71 of CEPA (Environment Canada 2008, 2011). A search of Material Safety

Data Sheets (MSDS), as well as consultation with petrochemical experts, resulted in no findings of recent information on Canadian manufacturing and import quantities or uses of polymerized C_{5-12} distillates in Canada or identified uses of polymerized C_{5-12} distillates in products available to consumers in Canada was found. Due to its expected low hazard concern, this substance is under the polymer reporting exemptions of the US *Toxic Substances Control Act* (TSCA) (US EPA 2019c).

No uses of polymerized C_{5-12} distillates in foods, natural health products, cosmetics, therapeutic or veterinary medicines have been reported in Canada [Personal communication, email from the Food Directorate, HC to the Existing Substances Risk Assessment Bureau, HC, dated March 25, 2019; unreferenced; email from Personal communication, email from the Natural and Non-prescriptive Health Products Directorate, HC to the Existing Substances Risk Assessment Bureau, HC, dated March 29 2019; unreferenced; Personal communication, email from the Consumer and Hazardous Products Safety Directorate, HC, to the Existing Substances Risk Assessment BureauHC, dated March 14, 2014; unreferenced; personal communication, email from the Therapeutic Products Directorate, HC to the Existing Substances Risk Assessment Bureau, HC, dated March 7, 2019; unreferenced;

2.3 Fate and exposure

Petroleum resins is primarily manufactured in refineries (Levelton 2011). Exposures associated with potential asphalt uses of petroleum resins were previously described in the Asphalt and Oxidized Asphalt screening assessment (ECCC, HC 2017c). Components of petroleum resins are expected to have high persistence and low bioaccumulation potential (ECCC, HC 2017c; US EPA 2011b).

Similar to asphalt, petroleum resins is expected to have low vapour pressure and very low water solubility under environmentally relevant conditions, and its components are not expected to disperse in the environment or to be bioavailable (ECCC, HC 2017c; US EPA 2011b). The hydrocarbon resin and polymerized C_{5-12} distillates have physical chemical properties similar to the petroleum resins and are not expected to disperse in the environment.

Exposures to the general human population from petroleum resins and hydrocarbon resin through the use of construction adhesives are expected to be infrequent and incidental. Based on their uses as hot-melt thermoplastics for consumers, dermal contact to the hot glue is expected to be limited, and physical contact with these substances is expected to occur mostly after the adhesive has hardened into the cool, solid form. Hydrocarbon resin that may be used in adhesives used in food packaging in Canada is not expected to have direct food contact. (personal communication, email from the Food Directorate, HC to the Existing Substances Risk Assessment Bureau, HC, dated March 25, 2019; unreferenced).

More sustained dermal exposures are expected from their use as adhesives or viscosity adjusters in the limited number of natural health and cosmetic products applied as tapes identified as containing petroleum or hydrocarbon resin. However, due to their low water solubility and high molecular weight (TDS 2018), these substances are expected to be associated with very low dermal absorption. Inhalation exposure to these substances is not expected due to their low vapour pressure.

No recent information on exposure of polymerized C_{5-12} distillates on the environment could be identified. Its primary production locations have been refineries and the petrochemical industry, and it is unlikely to leave these facilities without further processing (Levelton 2011). Ecological and human exposures from environmental media to this substance are not expected.

2.4 Ecological and human health effects

2.4.1 Ecological effects

Similar to asphalt, petroleum resins are not expected to cause acute or chronic toxicity to aquatic organisms due to their extremely low water solubility (ECCC, HC 2017c). Petroleum resins were found to have no measurable acute toxicity to *Daphnia magna* in a 48-h toxicity test using water-accommodated fraction (WAF) with a median effect loading rate[5] (EL_{50}) of > 100 mg/L or to the freshwater algae *Desmodesmus subspicatus* in a 72-h toxicity test with an EL_{50} of > 100 mg/L (ECHA 2018c) similar to the low toxicity observed with asphalt (ECCC, HC 2017c). No additional aquatic or terrestrial toxicity data for petroleum resins were identified in literature. No ecotoxicity data were available for hydrocarbon resin or polymerized C_{5-12} distillates; however, they are expected to have similar ecological effects as petroleum resins and are expected to be of low toxicity. They are also expected to have low hazard, consistent with their exemption from reporting under TSCA due to their low hazard concern (US EPA 2019c).

2.4.2 Human health effects

Petroleum resins, hydrocarbon resin, and polymerized C_{5-12} distillates were not identified as posing a hazard to human health based on classifications by other national or international agencies for carcinogenicity, genotoxicity, developmental toxicity, or reproductive toxicity. These three substances are not included on the European Chemicals Agency's Candidate List of Substances of Very High Concern for Authorisation (ECHA 2019c). In their Tier I Human Health Assessment, NICNAS considered the hydrocarbon resin to be in the category of Low Concern Polymer (NICNAS 2018).

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[5] A loading rate is the amount of petroleum substance added to the exposure solution to generate a WAF and is reported in mg/L. When used to describe an effect endpoint, the loading rate is the amount of petroleum substance added to generate a WAF that results in the effect reported; e.g., the lethal loading rate 50 (LL50) is the amount of petroleum substance needed to generate a WAF that is lethal to 50% of the test organisms. A loading rate is not a direct measure of the concentration of the petroleum components dissolved in the exposure solution.

Limited empirical health effects data were identified for these substances, which also included hazard information submitted to European Chemicals Agency (ECHA), under REACH requirements, as registration dossiers by other parties.

In an acute oral toxicity study (OECD test guideline 423), single-dose exposure to 2000 mg/kg-bw (gavage) of petroleum resin (CAS 64742-16-1) did not cause any mortality or signs of toxicity in female rats that were examined daily for up to 14 days post-exposure. A necropsy examination performed at the end of the study did not reveal any gross abnormalities. The study authors determined the LD_{50} value to be >2000 mg/kg-bw (ECHA 2019a; 2018c). Similarly, in an OECD test guideline 471 study, petroleum resin was found to be non-mutagenic in a bacterial reverse mutation assay in the presence and absence of metabolic activation in various *Salmonella typhimurium* strains. This substance did not show potential to cause skin sensitization in standard *in vitro* assays (ECHA 2018c). Due to its low acute oral toxicity, it has been assigned to Category 5 (“Warning; May be harmful if swallowed”. No symbol required) based on the Globally Harmonized System of Classification and Labelling of Chemicals (GHS)(ECHA 2019a).

For hydrocarbon resin (CAS 68131-77-1), the potential of the substance to cause skin sensitization has been identified (ECHA 2018a).

2.5 Risk characterization

The limited information available on the ecological hazard of these substances indicates that they present a low hazard to the environment. Release to the environment of petroleum resins, hydrocarbon resin, and polymerized C_{5-12} distillates are expected to be minimal and their components are not expected to disperse in the environment or to be bioavailable.

There is no indication in published literature that petroleum or hydrocarbon resin pose a hazard to human health. As noted in Section 2.1, the available data indicate that these substances are of low concern, as evidenced from their exemption from reporting status in the US EPA *Toxic Substances Control Act*. Given the physical and chemical properties of these substances, including their high molecular weight and very low vapour pressure and water solubility, they are expected to have low potential for inhalation and dermal absorption.

Releases of petroleum resins, hydrocarbon resin, and polymerized C_{5-12} distillates are expected to be minimal such that exposure to the general population is not expected, and exposure to the environment is expected to be limited.

Therefore, the potential to cause harm to the environment or to human health for the general population of Canada is expected to be low.

3. Oxidized hydrocarbon waxes with EA and oxidized hydrocarbon waxes with 2-MAE (CAS RN 67891-82-1, 97862-84-5)

3.1 Substance identity

Hydrocarbon waxes (petroleum), oxidized, compds. with ethanolamine (CAS RN 67891-82-1), will hereinafter referred to as oxidized hydrocarbon waxes with EA, and hydrocarbon waxes (petroleum), oxidized, compds. with 2-(methylamino)ethanol (CAS RN 97862-84-5), will hereinafter referred to as oxidized hydrocarbon waxes with 2-MAE. These two oxidized hydrocarbon waxes consist of gel-like constituents of oxidized petrolatum and alkanolamines (PubChem 2019). The alkanolamine fatty acid salts are not expected to be volatile since these substances have high molecular weight and are in ionized (salt) form. These substances are considered to be UVCBs.

3.2 Sources and uses

A search of possible uses indicates these substances are primarily produced in refineries (Levelton 2011), and are reported to be used as metal-working fluid, specifically as mold-release agents and as lubricants (Kirk-Othmer 2019). These uses are expected to be restricted to the metalworking industry and would not result in general population exposure; in addition, no product uses in Canada of this nature were identified via a search of available MSDS's.

No uses of oxidized hydrocarbon waxes with EA and oxidized hydrocarbon waxes with 2-MAE in foods, natural health products, cosmetics, therapeutic or veterinary medicines, or pest control products have been reported in Canada [Personal communication, email from the Food Directorate, HC to the Existing Substances Risk Assessment Bureau, HC, dated March 25, 2019; unreferenced; email from Personal communication, email from the Natural and Non-prescriptive Health Products Directorate, HC, to the Existing Substances Risk Assessment Bureau, HC, dated March 29 2019; unreferenced; Personal communication, email from the Consumer and Hazardous Products Safety Directorate, HC, to the Existing Substances Risk Assessment Bureau, HC, dated March 14, 2014; unreferenced; personal communication, email from the Therapeutic Products Directorate, HC to the Existing Substances Risk Assessment Bureau, HC, dated March 7, 2019; unreferenced; Personal communication, email from the Pest Management Regulatory Agency, Health Canada, to the Existing Substances Risk Assessment Bureau, HC, dated March 29, 2019].

3.3 Fate and exposure

Oxidized hydrocarbon waxes with EA and oxidized hydrocarbon waxes with 2-MAE are primarily produced in refineries (Levelton 2011). The persistence and bioaccumulation potential of both oxidized petrolatum and the alkanolamines, which are constituents of these substances, were characterized in the Petrolatum and Waxes and the Alkanolamines and Fatty Alkanolamides Group screening assessments (ECCC, HC

2016b, 2020). Oxidized petrolatums are expected to have negligible water solubility and are therefore expected to have very low exposure to aquatic organisms within the water column (PubChem 2019; ECCC HC 2016a). If released to water, due to the waxy or gel-like form and low water solubility, the oxidized petrolatum constituents of , oxidized hydrocarbon waxes with EA and oxidized hydrocarbon waxes with 2-MAE are not expected to remain in water and can be expected to partition to sediment. Although these substances are expected to be bioaccumulative, oxidative petrolatum constituents are not expected to biomagnify in aquatic or terrestrial webs and release of oxidized petrolatum to municipal wastewater are expected to be mostly removed by wastewater treatment plants as they are insoluble in water (ECCC, HC 2016b). Alkanolamines are not expected to persist in the environment or to bioaccumulate in organisms (ECCC, HC 2020). Given the waxy or gel-like form of the substance, it is expected that the high viscosity of the substance would lead to negligible releases from metalworking fluids as higher viscosity substances tend to remain on metalworking equipment (ECCC 2020). The uses of these two substances are expected to be limited to industrial settings and not lead to exposure of environmental media or the general population.

As these substances have not been identified in products available to consumers and have limited releases, low persistence and low bioavailability, exposure of the general population in Canada is not expected.

3.4 Ecological and human health effects

3.4.1 Ecological effects

Limited information on the ecological toxicity of oxidized hydrocarbon waxes with EA and oxidized hydrocarbon waxes with 2-MAE was identified. The ecological hazard potential of petrolatum and waxes, including oxidized petrolatum and alkanolamines, which are constituents of these substances, were characterized in the Petrolatum and Waxes and the Alkanolamines and Fatty Alkanolamides Group screening assessments (ECCC, HC 2016b, 2020) and was found to have low potential for harm to the environment. Read-across studies for petrolatum and wax suggest that these substances are of low toxicity and low bioavailability (ECCC, HC 2016b). Overall, potential for ecological harm from oxidized hydrocarbon waxes with EA and oxidized hydrocarbon waxes with 2-MAE is expected to be low.

3.4.2 Human health effects

No empirical health effects data was identified for these substances. Oxidized hydrocarbon waxes with EA and oxidized hydrocarbon waxes with 2-MAE were not identified as posing a hazard to human health based on classifications by other national or international agencies for carcinogenicity, genotoxicity, developmental toxicity, or reproductive toxicity. They are also not included on the European Chemicals Agency’s Candidate List of Substances of Very High Concern for Authorisation (ECHA 2019c).

3.5 Risk characterization

Limited information was available on the ecological hazard of the substances. Both oxidized petrolatum and the alkanolamines, which are constituents of these substances, were characterized as being of low ecological hazard in the Petrolatum and Waxes and the Alkanolamines and Fatty Alkanolamides Group screening assessments (ECCC, HC 2016b, 2020). Although the hazard database is limited, the available data indicates that these substances are considered to be of low hazard.

Expected uses of these substances as metalworking fluids/oils are in industrial settings. Metalworking fluids are often captured after use and recycled (Roberts 2015). Used metalworking fluids are disposed of as Used Oil and are subject to the Code of Practice for Used Oil Management in Canada (CCME 1989). Release to the environment of oxidized hydrocarbon waxes with EA and oxidized hydrocarbon waxes with 2-MAE from industrial facilities are expected to be minimal and their constituents are not expected to disperse in the environment or to be bioavailable to aquatic organisms.

The available data also indicates that these types of substances are considered to have a low ecotoxicity and low bioavailability.

The general population in Canada is not expected to be exposed to these substances through environmental media, food, or from the use of products available to consumers.

Therefore, the potential to cause harm to the environment or to human health for the general population of Canada is expected to be low.

4. Alkylated naphthalene sulfonate sodium salt polymers with formaldehyde (CAS RN 68425-94-5)

4.1 Substance identity and physical and chemical properties

Residues (petroleum), catalytic reformer fractionator, sulfonated, polymers with formaldehyde, sodium salts (CAS RN 68425-94-5), hereinafter referred to as alkylated naphthalene sulfonate sodium salt polymers with formaldehyde, has a vapour pressure of less than 130 Pa (1 mm Hg) (non-volatile) and has a specific gravity of less than 1. This substance is soluble in water (ECHA 2019d; MSDS 2013a). It consists of sodium naphthalene sulfonate or sodium alkylated-naphthalene sulfonate units connected by CH_2 linking groups from the process of hydroformylation. This substance is considered to be a UVCB.

4.2 Sources and uses

According to information submitted in response to a CEPA section 71 survey, and a voluntary data gathering initiative, reported industrial uses in Canada (ECCC 2015, 2016) include as a road construction ingredient and in enamels. This substance is primarily produced in the petrochemical industry (Levelton 2011). The primary use of this substance is as a dispersant or surfactant (ECCC 2015, 2016). Alkylated naphthalene sulfonate sodium salt polymers with formaldehyde is used as an additive in the manufacture of concrete products (MSDS 2013a), and as an additive in printer ink (MSDS 2012b). No recent information on Canadian manufacturing and import quantities of this substance in Canada, was available.

In Canada, alkylated naphthalene sulfonate sodium salt polymers with formaldehyde is also used as a formulant (dispersant/surfactant) in pest control products (personal communication, email from the Pesticides Management, HC to the Existing Substances Risk Assessment Bureau, HC, dated March 7, 2019; unreferenced). No uses of alkylated naphthalene sulfonate sodium salt in foods, natural health products, cosmetics, or veterinary medicines were reported in Canada [Personal communication, email from the Food Directorate, HC to the Existing Substances Risk Assessment Bureau, HC, dated March 25, 2019; unreferenced; email from Personal communication, email from the Natural and Non-prescriptive Health Products Directorate, HC, to the Existing Substances Risk Assessment Bureau, HC, dated March 29 2019; unreferenced; Personal communication, email from the Consumer and Hazardous Products Safety Directorate, HC, to the Existing Substances Risk Assessment Bureau, HC, dated March 14, 2014; unreferenced; Personal communication, email from the Therapeutic Products Directorate, HC to the Existing Substances Risk Assessment Bureau, HC, dated March 7, 2019; unreferenced].

Naphthalenesulfonic acid, polymer with formaldehyde, sodium salt (CAS RN 9084-06-4), and 2-naphthalenesulfonic acid, polymer with formaldehyde, sodium salt (CAS RN 36290-04-7) are anionic surfactants which are considered analogues for naphthalenesulfonic acid, polymers with formaldehyde.

4.3 Fate and exposure

No information on environmental fate and behaviour for alkylated naphthalene sulfonate sodium salt polymers with formaldehyde was identified in the literature. Similar polymers are found to have a water extractability of less than 2% by weight (ECCC, HC 2018). Based on available information, including a literature search, MSDS search, and discussions with experts in the petroleum field, this substance is not expected to exist in significant quantities in Canada and exposure to the environment in Canada is not expected.

Based on the available information, which indicates that this substance is not used in any products available to consumers, exposure of the general population in Canada to this substance is not expected.

Exposures to pesticides are evaluated under the *Pest Control Products Act* and are managed by Health Canada’s Pest Management Regulatory Agency (PMRA).

4.4 Ecological and human health effects

4.4.1 Ecological effects

No aquatic or terrestrial toxicity data for alkylated naphthalene sulfonate sodium salt polymers with formaldehyde was identified in the literature.

Substances of similar molecular structure, naphthalenesulfonic acid, polymer with formaldehyde, sodium salt and 2-naphthalenesulfonic acid, polymer with formaldehyde, sodium salt, were used to identify potential ecological effects of alkylated naphthalene sulfonate sodium salt polymers with formaldehyde. These analogues are polymers that are not expected to contain reactive functional groups associated with ecological hazard and, as per the New Substances Notification Regulations for Chemicals and Polymers, are considered to be of low potential to cause ecological harm (ECCC, HC [modified 2016]; 2018).

As such, the ecological effects from alkylated naphthalene sulfonate sodium salt polymers with formaldehyde are expected to be low.

4.4.2 Human health effects

No empirical health effects data was identified for this substance. Alkylated naphthalene sulfonate sodium salt polymers with formaldehyde was not identified as posing a high hazard to human health on the basis of classifications by other national or international agencies for carcinogenicity, genotoxicity, developmental toxicity, or reproductive toxicity. It is also not included on the European Chemicals Agency’s Candidate List of Substances of Very High Concern for Authorisation (ECHA 2019c).

4.5 Risk characterization

On the basis of available information indicating that alkylated naphthalene sulfonate sodium salt polymers with formaldehyde is not in use in Canada, environmental exposure from this substance is expected to be limited.

Although data for alkylated naphthalene sulfonate sodium salt polymers with formaldehyde was not identified in the literature, data on substances similar to alkylated naphthalene sulfonate sodium salt polymers with formaldehyde were found not to meet the criteria set out in section 64 of CEPA and are considered to be of low risk to the environment in Canada and to human health (ECCC, HC [modified 2016]; 2018).

Alkylated naphthalene sulfonate sodium salt polymers with formaldehyde has not been reported to be found in products available to the consumer in Canada.

Release of alkylated naphthalene sulfonate sodium salt polymers with formaldehyde is expected to be minimal such that exposure to the general population is not expected.

Therefore, the potential for harm to the environment or to human health for the general population of Canada is expected to be low.

5. Heavy oxo ends (CAS RN 68526-82-9)

5.1 Substance identity and physical and chemical properties

Alkenes C_{6-10}, hydroformylation products, high-boiling (CAS RN 68526-82-9), hereinafter referred to as heavy oxo ends, consists of the high boiling residuum produced from the distillation of C_{7-11} alcohols (NCI 2015). This substance is a complex mixture of oxygenated branched and multi-isomeric alcohols (C_8-C_{11}) and aliphatic ethers (C_{16}-C_{22}). This substance is also known as octyl alcohols bottoms (US EPA 2019a). This substance is a liquid at room temperature with a boiling point between 136 and 480°C. This substance has a density of 0.86 g/cm^3 and a vapour pressure of 4.3 to 28 hPa between 20 and 50 °C (BASF 2002; ECHA 2019b). The water solubility of this substance was determined by EPIWIN 3.05 using several typical components of this UVCB substance to gain an understanding of the range of water solubilities (BASF 2002). The most water-soluble components have a water solubility in the range of 100 mg/L with the higher limit of the solubility related to unreacted C_9 and C_{11} alcohols. Overall, components of this substance are considered to have solubility in the range of 4×10^{-5} (C_{22}) to 100 mg/L (C_9) at 20°C and, based on typical composition, while 26% of its components have a solubility of 1 mg/L or greater (BASF 2002). This substance is considered to be a UVCB.

5.2 Sources and uses

According to information submitted in response to a targeted CEPA section 71 information gathering initiative, heavy oxo ends is used in Canada as an industrial de-foamer (ECCC 2016).

According to EPA Inventory Update Reporting, the 2006 production volume for heavy oxo ends in the United States was between 1 million and $\leq$ 10 million lbs (453 600 to $\leq$ 4 536 000 kg) (US EPA 2011c). Recent EPA Inventory Update Reports do not report production volumes for heavy oxo ends (US EPA 2012, 2016). No recent information on Canadian manufacturing or import quantities or uses of these substances in Canada outside of qualitative uses reported in response to a CEPA section 71 survey (ECCC 2016) was identified.

Heavy oxo ends is used as an industrial de-foamer and lubricant (Knobeloch and Anderson 2006) as well as an intermediate in manufacturing (ECHA 2019b; Levelton 2011). It is used as a solvent and, in reaction with phthalic anhydride, is used to form phthalates, which are used as vinyl plasticizers (Franke et al. 2012).

No products available to consumers in Canada which include this substance have been identified.

Heavy oxo ends may be used as a component in coatings used on corrugated boxes used as food packaging in Canada with the potential for direct food contact. However, the exposure potential to this substance through food is considered negligible (personal communication, email from the Food Directorate, HC to the Existing Substances Risk Assessment Bureau, HC, dated March 25, 2019; unreferenced). No uses of heavy oxo ends in natural health products, cosmetics, therapeutic or veterinary medicines have been reported [Personal communication, email from the Natural and Non-prescriptive Health Products Directorate, HC, to the Existing Substances Risk Assessment Bureau, HC, dated March 29 2019; unreferenced; Personal communication, email from the Consumer and Hazardous Products Safety Directorate, HC, to the Existing Substances Risk Assessment Bureau, HC, dated March 14, 2014; unreferenced; personal communication, email from the Therapeutic Products Directorate, HC to the Existing Substances Risk Assessment Bureau, HC, dated March 7, 2019; unreferenced].

5.3 Fate and exposure

The higher carbon components of the heavy oxo ends are water insoluble and this substance does not hydrolyze readily in water (BASF 2002). The substance is inherently biodegradable in water with 49.99% biodegradation in 28 days (ECHA 2019c). Data on the most soluble components (C_9-C_{13} alcohols) indicate that linear alcohols are readily biodegradable (OECD 2006b), as well as mixtures of oxo alcohols (e.g., alcohols similar to those found in heavy oxo ends) (OECD 2006a).

Due to the physical-chemical properties of most of its components (relatively low vapour pressure and water solubility) (BASF 2002), environmental exposure is expected to be low. Its soluble components (C_9 – C_{13} alcohols) are expected to be readily degraded. When used as an industrial defoamer, heavy oxo ends will undergo wastewater treatment; wastewater removal modelling (SimpleTreat version 3.1 2003) for soluble C_9 and C_{13} alcohols indicate 88% or greater removal during secondary wastewater treatment.

No uses of this substance in products available to consumers have been identified and, exposure of the general population in Canada to heavy oxo ends through environmental media and product use is not expected.

5.4 Ecological and human health effects

5.4.1 Ecological effects

Heavy oxo ends was found to be of low hazard to rainbow trout (*Oncorhynchus mykiss*) in a 96-h toxicity test utilizing a water-accommodated fraction (WAF) with a non-lethal loading rate (LL_0) of >1000 mg/L or 0.71 mg/L measured (ECHA 2019b). Heavy oxo ends was also found to have no lethal effects on two species of fish (*Lepomis macrochirus* and *Oncorhynchus mykiss (formally referred to as Salmo gairdneri)*) in a 96-h toxicity test with an LC_0 > 1000 mg/L (nominal) (BASF 2002).

This substance was found to be acutely hazardous when administered with a solvent to *Daphnia magna* in a 48-h toxicity test with a NOEC of 0.1 mg/L (nominal) and an EC_{50} of 0.17 mg/L (nominal) (BASF 2002). It is expected that this represents the toxicity of the soluble alcohol components; and is similar to or lower than that observed for invertebrates and fish with C_{9}-$_{13}$ oxo alcohols (0.39 – 17.1 mg/L and 0.42 – 11 mg/L, respectively) (OECD 2006a). Most other components of oxo heavy ends have low to very low water solubility. The use of a solvent in the test with *D. magna* might have increased the bioavailability of components not usually soluble, such as higher carbon-chain alcohols that are more toxic than alcohols with shorter carbon chains (OECD 2006b) or other non-soluble components. Therefore, there is uncertainty regarding the results of this study.

Most of the water-soluble components (water solubility >1mg/L) of heavy oxo ends are C_{9-13} alcohols. Oxo alcohols in this range have toxicities to fish and invertebrates ranging from approximately 0.4 to 20 mg/L (OECD 2006a).

5.4.2 Human health effects

No empirical health effects data was identified for this substance. Heavy oxo ends was not identified as posing a high hazard to human health on the basis of classifications by other national or international agencies for carcinogenicity, genotoxicity, developmental toxicity, or reproductive toxicity. It is also not included on the European Chemicals Agency's Candidate List of Substances of Very High Concern for Authorisation (ECHA 2019c).

5.5 Risk characterization

While certain components of heavy oxo ends (i.e., C_{9-13} alcohols) are soluble and have moderate to high hazard, these same components are expected to be largely removed (approximately 90% removal) during wastewater treatment, and any remaining alcohols are expected to readily degrade if released to the environment. Environmental exposure to the remainder of the components is expected to be minimal due to their low to very low water solubility. A WAF study with fish demonstrated that even at high loading rates (1000 mg/L), only a small amount of heavy oxo ends was soluble in water (0.71 mg/L) and this was determined not to be hazardous. Therefore, the risk to the environment in Canada is expected to be low. The general population in Canada is not expected to be exposed to this substance through environmental media or from the use of products available to consumers. Exposure from food is considered to be negligible. On the basis of the low exposure, the risk to human health is considered to be low.

On the basis of the absence of heavy oxo ends in products available to consumer in Canada, and their limited environmental exposure, the potential to cause harm to the environment or to human health for the general population of Canada is expected to be low.

6. Sulfurized petroleum (CAS RN 68815-10-1)

6.1 Substance identity

Petroleum, sulfurized (CAS RN 68815-10-1) is hereinafter referred to as sulfurized petroleum. Sulfurization of unsaturated compounds involves addition of sulfur compounds (elemental sulfur, hydrogen sulfide, and/or mercaptans) to the substance. For example, sulfurized mineral oils can be made by dissolving elemental sulfur (flowers of sulfur) in mineral oil by heating (Farng and Jao 2017, Rossrucker et al. 2017).

Sulfurized olefins, as found in sulfurized petroleum, function mainly through thermal decomposition mechanisms, whereby the sulfur-carbon bonds dissociate at high temperature and the resulting sulfur combines with a metal to form a ductile metal sulfide surface film. Sulfur prevents contact between interacting ferrous metal surfaces through the formation of an intermediate film of iron sulfide which decreases the wear rate while accelerating the smoothing of the metal surfaces (Farng and Jao 2017). Sulfurized olefins or other oils are usually used under high pressure and temperature conditions. This substance is considered to be a UVCB.

6.2 Sources and uses

According to information submitted under a voluntary data gathering initiative, sulfurized petroleum is used in an industrial cutting fluid in Canada (ECCC 2016). The sulfur additive performs an anti-wear function in the cutting oil by forming a film between cutting tool and work piece. Sulfur-containing additives are used to provide protection against high-pressure, metal-to-metal contacts in boundary lubrication. This substance is primarily produced in refineries (Levelton 2011).

No uses in products available to consumers in Canada were identified for this substance.

No uses of sulfurized petroleum in foods, natural health products, cosmetics, therapeutic or veterinary medicines, or pest control products have been reported in Canada [Personal communication, email from Personal communication, email from the Food Directorate, HC to the Existing Substances Risk Assessment Bureau, HC, dated March 25, 2019; unreferenced; email from the Natural and Non-prescriptive Health Products Directorate, HC, to the Existing Substances Risk Assessment Bureau, HC, dated March 29 2019; unreferenced; Personal communication, email from the Consumer and Hazardous Products Safety Directorate, HC, to the Existing Substances Risk Assessment Bureau, HC, dated March 14, 2014; unreferenced; personal communication, email from the Therapeutic Products Directorate, HC to the Existing Substances Risk Assessment Bureau, HC, dated March 7, 2019; unreferenced; Personal communication, email from the Pest Management Regulatory Agency, HC, to the Existing Substances Risk Assessment Bureau, HC, dated March 29, 2019].

6.3 Fate and exposure

No information on ecological fate and exposure of sulfurized petroleum could be identified in the literature.

Expected uses of the substance in metalworking fluids are primarily in occupational and industrial settings. Metalworking fluids are often captured after use and recycled (Roberts 2015). Used metalworking fluids are disposed of as Used Oil and are subject to the Code of Practice for Used Oil Management in Canada (CCME 1989).

No uses in products available to consumers were identified for this substance. Exposure of the general population in Canada to sulfurized petroleum is not expected.

6.4 Ecological and human health effects

6.4.1 Ecological effects

No aquatic or terrestrial toxicity data for sulfurized petroleum could be identified in the literature. Based on modelled data from ECOSAR 2.0, sulfurized petroleum is insufficiently soluble to cause measurable toxic effects on aquatic organisms at solubility limit (ECOSAR 2017).

6.4.2 Human health effects

No empirical health effects data was identified for this substance. Sulfurized petroleum was not identified as posing a high hazard to human health on the basis of classifications by other national or international agencies for carcinogenicity, genotoxicity, developmental toxicity, or reproductive toxicity. It is also not included on the European Chemicals Agency's Candidate List of Substances of Very High Concern for Authorisation (ECHA 2019c). Further investigation of health effects is not warranted at this time given the low expected exposure of the general Canadian population.

6.5 Risk characterization

On the basis of sulfurized petroleum being used in metalworking in industrial settings where the substances are often captured after use and recycled, it is expected to have a low potential for release, and the environmental exposure from this substance is expected to be limited. Based on modelled data, sulfurized petroleum is expected to have relatively low toxicity, and therefore quantitative characterization of exposure (i.e., derivation of exposure estimates) was not conducted.

Exposure of the general population to sulfurized petroleum through environmental media, food, or the use of products available to consumers is not expected.
On the basis of the absence of sulfurized petroleum from products available to the consumer in Canada and the resulting limited environmental exposure, the exposure from this substance to the general population of Canada is not expected.

Release of sulfurized petroleum is expected to be minimal such that exposure to the general population is not expected, and exposure to the environment is expected to be limited. Therefore, the potential to cause harm to the environment or to human health for the general population of Canada is expected to be low.

7. Substances addressed under previous risk assessments

The identification of, and proposed action for, 35 hydrocarbon-based substances for which risk assessment activities can be considered as having already taken place follows on previous risk assessments under CEPA (Environment Canada, Health Canada 2015; ECCC, HC 2017a). As a result, these 35 substances were identified as not requiring further risk assessment at this time. In addition, existing or future risk management actions resulting from previous assessments, where applicable, are expected to address the risks of these substances.

Additional risk assessment or risk management activities may be undertaken if new information becomes available on these substances as a result of: 1) identification of new hazard or exposure information which may impact previous risk analyses; 2) international activities; or 3) risk management activities including performance measurement.

The list of the 35 hydrocarbon substances for which risk assessment activities can be considered as having already taken place is provided in Table A-1 in Appendix A. These substances are described in the following subsections.

7.1 Low boiling point naphthas

Low boiling point naphthas (LBPNs) are complex and highly variable combinations of volatile hydrocarbons, predominantly in the carbon range of C_4 to C_{12}.

The 31 LBPNs listed in Table A-1 in Appendix A have been identified as petroleum site-restricted or industry-restricted and do not appear in any marketplace products. They may be transported from petroleum sector facilities to other industrial facilities by ship or truck. They can be intermediate streams consumed within a facility, blended into a mixture leaving the facility under different CAS RNs or transported off-site for use as a feedstock in other industrial facilities. LBPNs were previously assessed under the Site-Restricted LBPNs screening assessment and the Industry-Restricted LBPNs screening assessment and were found to not meet the criteria set out in section 64 of CEPA (Environment Canada, Health Canada 2011, 2013a). No additional general population and environmental media exposures to the LBPN substances of this group are expected beyond those already considered in the Site-Restricted LBPNs screening assessment and the Industry-Restricted LBPNs screening assessment (Environment Canada, Health Canada 2013a, 2014).

As such, these substances are considered to have been addressed and will not be subject to further risk assessment at this time.

7.2 Natural gas and natural gas, dried (CAS RNs 8006-14-2 and 68410-63-9)

Natural gas and natural gas, dried, are part of the substances broadly classified as petroleum and refinery gases (PRGs), which also includes liquefied petroleum gases (LPGs).

PRGs are complex and highly variable combinations of volatile hydrocarbons predominantly in the carbon range of C_1 to C_7, and are produced in petroleum facilities (i.e., refineries and natural gas processing facilities).

PRGs were previously assessed under two Petroleum and Refinery Gases screening assessments (Environment Canada, Health Canada 2011, 2013b) as well as the Liquefied Petroleum Gases screening assessment (ECCC, HC 2016a) and were found to meet the criteria under paragraph 64(*c*) of CEPA due to their contribution to overall petroleum refinery emissions, specifically of 1,3-butadiene. The ECCC, HC (2016c) assessment, however, notes that human health risks from volatile emissions at natural gas processing facilities, where natural gas and natural gas, dried are produced, are low, based on laboratory testing and other lines of evidence demonstrating that appreciable concentrations of 1,3-butadiene are not expected to be present in gas streams at natural gas processing facilities.

Potential exposures from natural gas and natural gas, dried are considered to have been addressed under the Liquefied Petroleum Gases screening assessment (ECCC, HC 2016a) and include releases within facilities from activities associated with their production and processing, releases related to their transportation between industrial facilities, and release during consumer uses. No additional general population and environmental media exposures from natural gas and natural gas, dried beyond those already considered in the Liquefied Petroleum Gases screening assessment are expected. Risk management for PRGs occurs under the existing Risk Management Approach for Liquefied Petroleum Gases (ECCC, HC 2017).

As such, natural gas and natural gas, dried, will not be subject to further risk assessment at this time and will be addressed by subsequent risk management measures that are being, or have been, developed for PRGs. The *Regulations Respecting Reduction in the Release of Volatile Organic Compounds (Petroleum Sector)* will also address natural gas and natural gas, dried (CAS RNs 8006-14-2 and 68410-63-9).

7.3 Pitch (CAS RN 61789-60-4)

Pitch is a residue resulting from the distillation of coal tar. Coal tars and their distillates, which include coal tar pitches, have been previously assessed under the Coal Tars and their Distillates Assessment (ECCC, HC 2021a). Pitch (CAS RN 61789-60-4) is considered to fit the general description of the substance named high-temperature coal tar pitch (CAS RN 65996-93-2). No additional general population and environmental media exposures to pitch beyond those already captured in the Coal Tars and their Distillates screening assessment are expected.

The substances in the Coal Tars and their Distillates screening assessment (ECCC, HC 2021a) met the criteria under sections 64 (*a*) and (*c*) of CEPA and risk management measures for these substances are outlined in the Risk Management Approach for Coal Tars and their Distillates (ECCC, HC 2021b), wherein they have been recommended to be added to the List of Toxic Substances in Schedule 1 of CEPA.

Pitch is considered to fall within the scope of the Coal Tars and their Distillates screening assessment and will, therefore, not be subject to further risk assessment at this time. It will be addressed by any subsequent risk management measures that are being or have been developed for the Coal Tars and their Distillates Group.

7.4 Light oil (coal) coke-oven (CAS RN 65996-78-3)

Light oil (coal) coke-oven is a stream of the coal tar production process. It is a volatile organic liquid extracted from the gas evolved in the high temperature (greater than 700° C) destructive distillation of coal. It is composed primarily of benzene, toluene, and xylenes. It may contain other minor hydrocarbon components. This substance is also called benzole (benzol) (ECHA 2018b). This substance considered to fall within the scope of the group assessment of Coal tars and their Distillates (ECCC, HC 2021a).

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, with the resulting coal tars often delineated by the pyrolysis temperature (low or high). Coal tar distillates are various boiling point fractions derived from the distillation of coal tars at a coal tar refiner, and include both the fractions obtained from the distillation tower as well as the residue remaining following distillation. The Coal Tars and their Distillates screening assessment concluded that these substances have the potential to cause harm to the environment and the general population, and met the criteria under paragraphs 64(*a*) and (*c*) of CEPA (ECCC, HC 2021a), due to releases of hazardous substances such as polycyclic aromatic hydrocarbons (PAHs) and benzene. The production of Light oil (coal) coke-oven in integrated steel mills is addressed through existing regulations (Environment Canada 2001).

Light oil (coal) coke-oven is recognized to be of high benzene content (typically 60% or greater) and is used as a feedstock in the chemical industry for the production of aromatic chemicals. The uses of benzene in chemical production and as a solvent are considered in the Benzene Priority Substance List Assessment Report (Canada 1993).

The manufacture and use of light oil (coal) coke-oven are considered to have been assessed as part of the Coal Tars and their Distillates Screening Assessment (ECCC, HC 2021a) and the Benzene Priority Substance List Assessment Report (Canada 1993). As no general population and environmental media exposures beyond those already considered in these two assessments are expected, this substance will not be subject to further risk assessment at this time and will be addressed by risk management measures that are being or have been developed for the coal tars and their distillates group or for benzene.

8. Conclusion

Considering all available lines of evidence presented in the draft screening assessment, there is low risk of harm to the environment from the eight hydrocarbon-based substances in this assessment. It is proposed to conclude that the eight hydrocarbon-based substances in this assessment do not meet the criteria under paragraphs 64(*a*) or (*b*) of CEPA as they are not entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity or that constitute or may constitute a danger to the environment on which life depends.

On the basis of the information presented in this draft screening assessment, it is proposed to conclude that the eight hydrocarbon-based substances in this assessment do not meet the criteria under paragraph 64(*c*) of CEPA as they are not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

It is therefore proposed to conclude that the eight hydrocarbon-based substances assessed in this draft screening assessment do not meet any of the criteria set out in section 64 of CEPA.

Based on their composition, physical-chemical properties and reported uses, 35 substances (31 low boiling point naphthas; two natural gases; pitch and light oil (coal) coke-oven) are considered to fall within the scope of hydrocarbon groups which have previously been assessed under CEPA. Identified uses of these 35 substances are not expected to result in exposures beyond those already considered in previous assessments. Since these substances are not expected to contribute additional concern to human health or the environment beyond those that have already been identified in past assessments of similar substances, they will not undergo further assessment at this time. In addition, existing or future risk management actions related to the previous assessments, when applicable, are expected to address the risks from these substances.

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Appendix A. Hydrocarbon-based substances for which risk assessment activities have already taken place under CEPA

Table A-1 Thirty-six hydrocarbon-based substances which fall under previous risk assessments

CAS RN	DSL name
64741-46-4[a,c]	Naphtha (petroleum), light straight run
64741-63-5[a,c]	SIDS Naphtha (petroleum), light catalytic reformed
64741-72-6[a,c]	Naphtha (petroleum), polymn.
64741-83-9[a,c]	Naphtha (petroleum), heavy thermal cracked
64741-99-7[a,c]	Extracts (petroleum), light naphtha solvent
67891-79-6[a,c]	Distillates (petroleum), heavy arom.
68131-49-7[a,c]	Aromatic hydrocarbons, C6-10, acid-treated, neutralized
68410-98-0[a,c]	Distillates (petroleum), hydrotreated heavy naphtha, deisohexanizer overheads
68425-35-4[a,c]	Raffinates (petroleum), reformer, Lurgi unit-sepd.
368475-70-7[a,c]	Aromatic hydrocarbons, C6-8, naphtha-raffinate pyrolyzate-derived
68475-79-6[a,c]	Distillates (petroleum), catalytic reformed depentanizer
68476-47-1[a,c]	Hydrocarbons, C2-6, C6-8 catalytic reformer
68476-55-1[a,c]	Hydrocarbons, C5-rich
68477-63-4[a,c]	Extracts (petroleum), reformer recycle
68478-15-9[a,c]	Residues (petroleum), C6-8 catalytic reformer
68513-63-3[a,c]	Distillates (petroleum), catalytic reformed straight-run naphtha overheads
68516-20-1[a,c]	Naphtha (petroleum), steam-cracked middle arom.
68527-21-9[a,c]	Naphtha (petroleum), clay-treated full-range straight-run
68527-22-0[a,c]	Naphtha (petroleum), clay-treated light straight-run
68603-00-9 [a,c]	Distillates (petroleum), thermal cracked naphtha and gas oil
68783-11-9[a,c]	Naphtha (petroleum), light polymn.
68783-66-4[a,c]	Naphtha (petroleum), light, sweetened
68919-15-3[a,c]	Hydrocarbons, C6-12, benzene-recovery
68921-08-4 [a,c]	Distillates (petroleum), light straight-run gasoline fractionation stabilizer overheads
92045-52-8[a,c]	Naphtha (petroleum), hydrodesulfurized full-range
92045-60-8[a,c]	Naphtha (petroleum), light, C5-rich, sweetened
128683-32-9[a,c]	Naphtha (oil sand)
128683-33-0[a,c]	Naphtha (oil sand), hydrotreated
128683-34-1[a,c]	Naphtha (oil sand), light straight-run

CAS RN	DSL name
129893-11-4[a,c]	Residues (petroleum), vacuum, hydrocracked, naphtha fraction
139730-55-5[a,c]	Naphtha (petroleum), hydrotreated light, catalytic reformed
8006-14-2[b,d]	Natural gas
68410-63-9 [b,d]	Natural gas, dried
61789-60-4 [a,e]	Pitch
65996-78-3 [b,e]	Light oil (coal), coke-oven

[a] This substance was categorized under subsection 73(1) of CEPA.

[b] This substance was not identified under subsection 73(1) of CEPA but was considered a priority on the basis of other human health concerns.

[c] This substance is considered to have been previously assessed under the Site-Restricted LBPNs Screening Assessment and/or the Industry-Restricted LBPNs Screening Assessment (ECCC, HC 2011, 2013).

[d] This substance is considered to have been previously assessed under the Liquefied Petroleum Gases assessment (ECCC, HC 2016a).

[e] This substance is considered to have been previously assessed under the Coal Tars and their Distillates screening assessments (ECCC, HC 2021a).

GRENHAL

- SAFETY DATA SHEET -

SECTION 1: IDENTIFICATION

Product Name: **PETRORESIN GC-300L**
Chemical Name: Petroleum Hydrocarbon Resin
Recommended use: Tackifier used in the manufacture of Adhesives, Inks & Coatings
Restrictions on use: None

MANUFACTURER / SUPPLIER:
Grenhall Industries Inc.
1 Imperial Court
Brampton, Ontario, Canada L6T 4X4
Tel: 905-458-8549
Fax: 905-458-8363
www.grenhall.com

EMERGENCY CONTACT:
Chemtrec: 1-800-424-9300
Canutec: 613-996-6666

SECTION 2: HAZARDS IDENTIFICATION

Hazard Classification: This product is considered non-hazardous.
This product is not classified according to OSHA regulations (CFR 29 1910.1200)

OSHA Specified Hazards:
Combustible dust: If converted to small particles during further processing, handling or by other means may form combustible dust concentrations in air.

Warning label items including precautionary statement:

Signal Word(s): **WARNING!**

Hazard Statement(s): If converted to small particles during further processing, handling or by other means, product may form combustible dust concentrations in air.

Precautionary Statement:
Disposal: Dispose of contents/container to an appropriate treatment and disposal facility in accordance with applicable laws and regulations, and product characteristics at time of disposal.

Hazard(s) not otherwise classified (HNOC): None known

SECTION 3: COMPOSITION / INFORMATION ON INGREDIENTS

Substances / Mixtures
General Information:

Chemical Name	CAS No.	Concentration
Petroleum Resins	64742-16-1	100%

SECTION 4: FIRST-AID MEASURES

INHALATION:	In pellet form, this product is not expected to be a problem. If resin dust is inhaled, move victim to fresh air. Treat symptomatically. Get medical attention if symptoms persist.
EYE CONTACT:	Any material that contacts the eye should be washed out immediately with water. Get medical attention if symptoms persist.
SKIN CONTACT:	Wash contact areas with soap and water. Get medical attention if symptoms persist.
INGESTION:	Seek medical advice. Material is not expected to be absorbed from the gastrointestinal tract so that induction of vomiting should not be necessary.

SECTION 5: FIRE-FIGHTING MEASURES

General Fire Hazards:	Material can accumulate static charges which may cause an electrical spark (ignition source). Use proper bonding and/or grounding procedures.

Extinguishing media

Suitable extinguishing media:	Water spray. Carbon Dioxide. Dry chemical.
Unsuitable extinguishing media:	None known
Special hazards arising from the substance: or mixture	Powdered material may form explosive dust-air mixture

Advice for firefighters

Special fire fighting procedures:	Minimize dust generation and accumulation.
Special protective equipment for fire-fighters:	Self-contained breathing apparatus and full protective clothing must be worn in case of fire.

SECTION 6: ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures:	Wear appropriate personal protective equipment.
Environmental Precautions:	Not regarded as dangerous for the environment.
Methods and material for containment and cleaning up:	Sweep up and place in a clearly labeled container for chemical waste. Large Spillages: Flush spill area with water spray.

	Prevent runoff from entering drains, sewers, or streams. Dike for later disposal.
Notification Procedures:	In the event of a spill or accidental release, notify relevant authorities in accordance with all applicable regulations.

SECTION 7: HANDLING AND STORAGE

Precautions for safe handling:	Minimize dust generation and accumulation.
Conditions for safe storage, including any incompatibilities:	Keep container closed.
Specific end use(s):	Chemical Intermediate

SECTION 8: EXPOSURE CONTROLS / PERSONAL PROTECTION

Control Parameters	
Occupational Exposure Limits	
	Country specific exposure limits have not been established or are not applicable unless listed below.
Exposure controls	
Appropriate engineering controls:	Good general ventilation (typically 10 air changes per hour) should be used. Ventilation rates should be matched to conditions. If applicable, use process enclosures, local exhaust ventilation, or other engineering controls to maintain airborne levels below recommended exposure limits. If exposure limits have not been established, maintain airborne levels to an acceptable level.
Individual protection measures, such as personal protective equipment	
General information:	Eye bath. Washing facilities.
Eye/face protection:	It is a good industrial hygiene practice to minimize eye contact.
Skin protection	
Hand Protection:	It is a good industrial hygiene practice to minimize skin contact.
Other:	No data available.
Respiratory Protection:	If engineering controls do not maintain airborne concentrations below recommended exposure limits (where applicable) or to an acceptable level (in countries where exposure limits have not been established), an approved respirator must be worn. In the United States of America, if respirators are used, a program should be instituted to assure compliance with OSHA Standard 63 FR 1152, January 8, 1998. Respirator type: Air-purifying respirator with an appropriate, government approved (where applicable), air-purifying filter, cartridge or canister. Contact health and safety professional or manufacturer for specific information.
Hygiene measures:	Observe good industrial hygiene practices.
Environmental Controls:	No data available.

SECTION 9: PHYSICAL AND CHEMICAL PROPERTIES

Appearance
  Physical State: Solid
  Form: Flakes or Prills
  Color: light yellow
Odour: weak, characteristic hydrocarbon
Odour Threshold: Not determined.
pH: No data available.
Softening Point: 120-130°C (Ring and Ball Method)
Boiling Point: No data available.
Flash Point: 260°C (Cleveland open cup)
Evaporation Rate: Not applicable.
Flammability (solid, gas): Product is not flammable.
Flammability Limit - Upper (%)–: Not applicable.
Flammability Limit - Lower (%)–: Not applicable.
Vapour pressure: Not determined.
Vapour density (air=1): No data available.
Specific Gravity: 1.06 (25°C)
Solubility(ies)
  Solubility in Water: Negligible
  Solubility (other): No data available.
Partition coefficient (n-octanol/water): No data available.
Autoignition Temperature: Product is not self-igniting.
Decomposition Temperature: Not determined.
Dynamic Viscosity: Not determined.
Kinematic viscosity: Not determined.
Explosive properties: Product is not explosive.
Oxidizing properties: Not oxidizing.

SECTION 10: STABILITY AND REACTIVITY

Reactivity: None known
Chemical Stability: Product is stable under ordinary conditions.
Possibility of Hazardous Reactions: None known
Conditions to avoid: None at ambient temperature
Incompatible Materials: Strong oxidizing agents
Hazardous Decomposition Products: Carbon Monoxide. Carbon Dioxide.

SECTION 11: TOXICOLOGICAL INFORMATION

Information on likely routes of exposure
  Inhalation: None known.

  Ingestion: None known.

  Skin Contact: None known.

  Eye contact: None known.

Information on toxicological effects

  Oral
    Product: No data available.

Dermal
Product: No data available.

Inhalation
Product: No data available.

Repeated Dose Toxicity
Product: No data available.

Skin Corrosion/Irritation
Product: No data available.
Serious Eye Damage/Eye Irritation
Product: No data available.

Respiratory or Skin Sensitization
Product: No data available.

Carcinogenicity
Product: No data available.

Specified substance(s):
additive(s)/colorant(s) IARC Not Listed. NTP Not Listed. OSHA Not Listed.

Toxicity to reproduction
Product: No data available.

Developmental Toxicity
Product: No data available.

Germ Cell Mutagenicity
In vitro Product: No data available.

In vivo Product: No data available.

Specific Target Organ Toxicity - Single Exposure
Product: No data available.

Specific Target Organ Toxicity - Repeated Exposure
Product: No data available.

Aspiration Hazard
Product: No data available.

Other Effects: No data available.

SECTION 12: ECOLOGICAL INFORMATION

Ecotoxicity:

Acute hazards to the aquatic environment:

Fish
Product: No data available.

Aquatic Invertebrates
Product: No data available.

Chronic hazards to the aquatic environment:

Fish

Product:	No data available.

Aquatic Invertebrates

Product:	No data available.

Toxicity to Aquatic Plants

Product:	No data available.

Persistence and Degradability

Biodegradation

Product:	Not easily biodegradable. Easily eliminable from water.

BOD/COD Ratio

Product:	No data available.

Bioaccumulative Potential

Bioconcentration Factor (BCF)

Product:	No data available.

Partition Coefficient n-octanol / water (log Kow)

Product:	No data available.

Mobility in Soil:	Adsorption to solid phase is possible.

Known or predicted distribution to environmental compartments

hydrocarbon resin	No data available.
additive(s)/colorant(s)	No data available.

Other Adverse Effects:	No data available.

SECTION 13: DISPOSAL CONSIDERATIONS

Waste treatment methods

General information:	No data available.
Disposal methods:	Dispose of waste and residues in accordance with local authority requirements. Incinerate.

SECTION 14: TRANSPORT INFORMATION

TDG:	Class not regulated
DOT:	Class not regulated
IMDG:	Class not regulated
IATA:	Class not regulated

SECTION 15: REGULATORY INFORMATION

Safety, health and environmental regulations/legislation specific for the substance or mixture:

WHMIS (Canada) Status: non-controlled

SARA 311-312 Hazard Classification(s):
fire hazard

US EPCRA (SARA Title III) Section 313 - Toxic Chemical List
NONE
NONE

OSHA: non-hazardous
DSL (Canada - Domestic Substances List) Inventory: Product is listed

TSCA (US Toxic Substances Control Act): All components of this product are listed on the TSCA inventory. Any impurities present in this product are exempt from listing.

MITI (Japanese Handbook of Existing and New Chemical Substances): All components of this product are listed in the Handbook or have been approved in Japan by new substance notification.

Philippines Inventory (PICCS) : All components of this product are listed on the Philippine inventory or otherwise comply with PICCS.

Inventory of Existing Chemical Substances in China: All components of this product are listed on the Inventory of Existing Chemical Substances in China (IECSC).

Australian Inventory of Chemical Substances: Product is listed.

New Zealand Inventory of Chemical Substances: Product is listed.

SECTION 16: OTHER INFORMATION

According to REACH, this product does not legally require a SDS. However, it is used to communicate information according to Article 32 of REACH.

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