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); Petroleum Resin 25; PetrResin; Bitumen; Petroleum Hydrocarbon; [Ergon MSDS] (Haz-Map. 2020).

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)

Not applicable.

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, 2020)

1.7.2. Boiling point

(°C): >482 (US EPA, 2020)

1.7.3. Solubility

<1.0x10-5 mg/L (estimated) (US EPA, 2020)

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

<1.0x10-10 to 2.6x10-7 mmHg at 25°C (estimated) (US EPA, 2020)

1.7.11. log Kow

> 10 (estimated) (US EPA, 2020)

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 NICNAS, 2018

National Occupational Exposure Survey (1981 - 1983)

Estimated Numbers of Employees Potentially Exposed to Specific Agents by Occupation*

Agent Name	PETROLEUM RESINS		
CAS #	64742-16-1		
RTECS #			
Agent Code	Y1024		
Code	Occupation Description (1980)	Total # Employees (Male & Female)	Total # Female Employees
019	MANAGERS AND ADMINISTRATORS, N.E.C.	1,830	
057	MECHANICAL ENGINEERS	42	
099	OCCUPATIONAL THERAPISTS	100	100
214	INDUSTRIAL ENGINEERING TECHNICIANS	27	27
216	ENGINEERING TECHNICIANS, N.E.C.	80	
224	CHEMICAL TECHNICIANS	2,529	359
225	SCIENCE TECHNICIANS, N.E.C.	775	55

235	TECHNICIANS, N.E.C.	1,138	57
336	RECORDS CLERKS	1,848	623
363	PRODUCTION COORDINATORS	918	143
364	TRAFFIC, SHIPPING, AND RECEIVING CLERKS	1,730	
365	STOCK AND INVENTORY CLERKS	161	46
368	WEIGHERS, MEASURERS, AND CHECKERS	408	
379	GENERAL OFFICE CLERKS	2,386	1,607
389	ADMINISTRATIVE SUPPORT OCCUPATIONS, N.E.C.	1,292	775
426	GUARDS AND POLICE, EXC. PUBLIC SERVICE	140	
453	JANITORS AND CLEANERS	12,782	741
507	BUS, TRUCK, AND STATIONARY ENGINE MECHANICS	280	
508	AIRCRAFT ENGINE MECHANICS	6,707	57
516	HEAVY EQUIPMENT MECHANICS	670	
518	INDUSTRIAL MACHINERY REPAIRERS	6,045	
519	MACHINERY MAINTENANCE OCCUPATIONS	454	33
533	MISCELLANEOUS ELECTRICAL AND ELECTRONIC EQUIPMENT REPAIRERS	12	
547	SPECIFIED MECHANICS AND REPAIRERS, N.E.C.	1,313	
549	NOT SPECIFIED MECHANICS AND REPAIRERS	3,214	302
558	SUPERVISORS, N.E.C.	1,745	

567	CARPENTERS	18,310	3
569	CARPENTER APPRENTICES	64	
575	ELECTRICIANS	230	
579	PAINTERS, CONSTRUCTION AND MAINTENANCE	92	
585	PLUMBERS, PIPEFITTERS, AND STEAMFITTERS	2,801	
587	PLUMBER, PIPEFITTER, AND STEAMFITTER APPRENTICES	460	
589	GLAZIERS	1,355	
593	INSULATION WORKERS	3,583	
595	ROOFERS	7,117	
599	CONSTRUCTION TRADES, N.E.C.	894	
633	SUPERVISORS, PRODUCTION OCCUPATIONS	1,318	170
637	MACHINISTS	1,862	
639	MACHINIST APPRENTICES	139	
647	PRECIOUS STONES AND METALS WORKERS (JEWELERS)	1,252	1,252
653	SHEET METAL WORKERS	2,758	
679	BOOKBINDERS	5,317	2,948
695	POWER PLANT OPERATORS	45	
696	STATIONARY ENGINEERS	112	
703	LATHE AND TURNING MACHINE SET-UP OPERATORS	14	

719	MOLDING AND CASTING MACHINE OPERATORS	3,932	541
723	METAL PLATING MACHINE OPERATORS	253	23
725	MISCELLANEOUS METAL AND PLASTIC PROCESSING MACHINE OPERATORS	6	
733	MISCELLANEOUS WOODWORKING MACHINE OPERATORS	631	
734	PRINTING MACHINE OPERATORS	76,723	4,836
735	PHOTOENGRAVERS AND LITHOGRAPHERS	1,140	123
736	TYPESETTERS AND COMPOSITORS	2,150	1,292
737	MISCELLANEOUS PRINTING MACHINE OPERATORS	4,136	2,077
744	TEXTILE SEWING MACHINE OPERATORS	1,446	233
748	LAUNDERING AND DRY CLEANING MACHINE OPERATORS	1,065	166
749	MISCELLANEOUS TEXTILE MACHINE OPERATORS	3,376	237
753	CEMENTING AND GLUING MACHINE OPERATORS	480	160
754	PACKAGING AND FILLING MACHINE OPERATORS	6,602	
755	EXTRUDING AND FORMING MACHINE OPERATORS	850	65
756	MIXING AND BLENDING MACHINE OPERATORS	2,376	99
757	SEPARATING, FILTERING, AND CLARIFYING MACHINE OPERATORS	285	3
759	PAINTING AND PAINT SPRAYING MACHINE OPERATORS	945	285
765	FOLDING MACHINE OPERATORS	36	18
766	FURNACE, KILN, AND OVEN OPERATORS, EXC. FOOD	44	

769	SLICING AND CUTTING MACHINE OPERATORS	3,189	1,779
774	PHOTOGRAPHIC PROCESS MACHINE OPERATORS	258	
777	MISCELLANEOUS MACHINE OPERATORS, N.E.C.	9,563	1,219
779	MACHINE OPERATORS, NOT SPECIFIED	2,083	171
783	WELDERS AND CUTTERS	490	
785	ASSEMBLERS	14,926	4,454
787	HAND MOLDING, CASTING, AND FORMING OCCUPATIONS	219	
789	HAND PAINTING, COATING, AND DECORATING OCCUPATIONS	660	587
795	MISCELLANEOUS HAND WORKING OCCUPATIONS	236	68
796	PRODUCTION INSPECTORS, CHECKERS, AND EXAMINERS	1,152	405
798	PRODUCTION SAMPLERS AND WEIGHERS	819	
804	TRUCK DRIVERS, HEAVY	874	
844	OPERATING ENGINEERS	54	
856	INDUSTRIAL TRUCK AND TRACTOR EQUIPMENT OPERATORS	90	
859	MISCELLANEOUS MATERIAL MOVING EQUIPMENT OPERATORS	1,761	630
865	HELPERS, CONSTRUCTION TRADES	549	
869	CONSTRUCTION LABORERS	12,404	
873	PRODUCTION HELPERS	1,933	363

878	MACHINE FEEDERS AND OFFBEARERS	2,534	
888	HAND PACKERS AND PACKAGERS	3,965	2,024
889	LABORERS, EXCEPT CONSTRUCTION	7,299	
TOTAL		267,878	31,152

*(1) The estimates for each occupation apply across the surveyed industries in which the agent was observed. Not all industries were surveyed, and not all agents were observed in all surveyed industries. (2) When using the estimates, standard errors associated with estimates should be considered. (3) Potential exposures to a chemical agent are categorized as actual (i.e., the surveyor observed the use of the specific agent) or tradename (i.e., the surveyor observed the use of a tradename product known to contain the specific agent). The estimates presented in the table combine both categories.

As taken from NIOSH, available at https://web.archive.org/web/20111028122746/http://www.cdc.gov/noes/noes2/y1024occ.html

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

“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).

TSCA Definition 2008: Obtained as a fraction of the extract from solvent extraction of residuum and consisting predominantly of high molecular weight compounds with high carbon-to-hydrogen ratios; [ChemIDplus] The fumes produced from heating (often necessary in applications such as paving) are considered the major occupational hazard of these complex mixtures.

As taken from Haz-Map, 2020

3. Status in legislation and other official guidance

Included on the FDA’s List of Indirect Additives Used in Food Contact Substances and covered under 21 CFR section 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, 2019, 2020

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, 2020).

There is a REACH dossier on petroleum resins (CAS RN 64742-16-1) (ECHA, 2020a).

Petroleum resins (CAS RN 64742-16-1) are not classified for packaging and labelling under Regulation (EC) No. 1272/2008 (ECHA, 2020b).

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). The Chemical Data Reporting (CDR) Rule requires companies that manufacture (including import) certain chemicals at certain volumes in the U.S. to report to EPA every four years through its CDR.

The TSCA inventory, and 2012 CDR and 2020 CDR Partial Exempt lists are available at https://iaspub.epa.gov/sor_internet/registry/substreg/searchandretrieve/searchbylist/search.do

Petroleum resins (CAS RN 64742-16-1) are included on the New Zealand Inventory of Chemicals (NZIoC) and may be used as a single component chemical under an appropriate group standard (NZ EPA, 2006).

Petroleum resins (CAS RN 64742-16-1) are listed by the US EPA Office of Pesticide Programs (2020) and were first registered as antimicrobial and “conventional chemical” pesticides on 13 November 1984.

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 NICNAS, 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 NICNAS, 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 dose, 50 percent kill	Oral	Mammal - species unspecified	7 gm/kg	Behavioral - somnolence (general depressed activity) Behavioral - excitement Behavioral - muscle contraction or spasticity	GTPZAB Gigiena Truda i Professional'nye Zabolevaniya. 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

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 NICNAS, 2018

5.2. Repeated dose toxicity

Petroleum resins
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	males for six hours/day, seven

	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).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	days/week for 28 days, and females were exposed for 35-48 days

	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 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)	6 hours/day, 3 days/week for 4 weeks
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-	6 hours/day, 3 days/week for 4 weeks

	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)	

As taken from US EPA, 2020.

“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 NICNAS, 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, 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	males for six hours/day, seven days/week for 28 days, and females were exposed for 35-48 days

	condensate (supporting chemical; derived from CASRN 64742-93-4)	

As taken from US EPA, 2020

“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 NICNAS, 2018

5.4. Mutagenicity

Petroleum resins
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, 2020

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

As taken from NICNAS, 2018

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 NICNAS, 2018

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 NICNAS, 2018

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

July 2020

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])

[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.

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

Chemical Abstract Service Registry Number (CASRN)	**Sponsored Chemicals** **See Appendix** **Supporting Chemicals** **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]

[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]	$<1\times10^{-10}$ 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 $\times 10^5$ (estimated)[2,3]	1.0 to 1.5$\times 10^5$ (estimated)[2,3]	4.3$\times 10^4$ to 6.3$\times 10^4$ (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

[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

[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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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 ^{o}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.

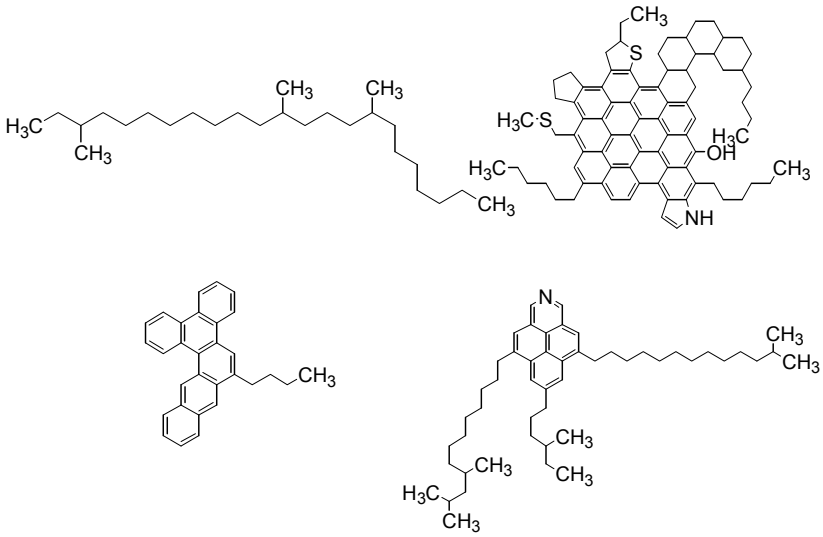
The major chemical groups in produced asphalt are:

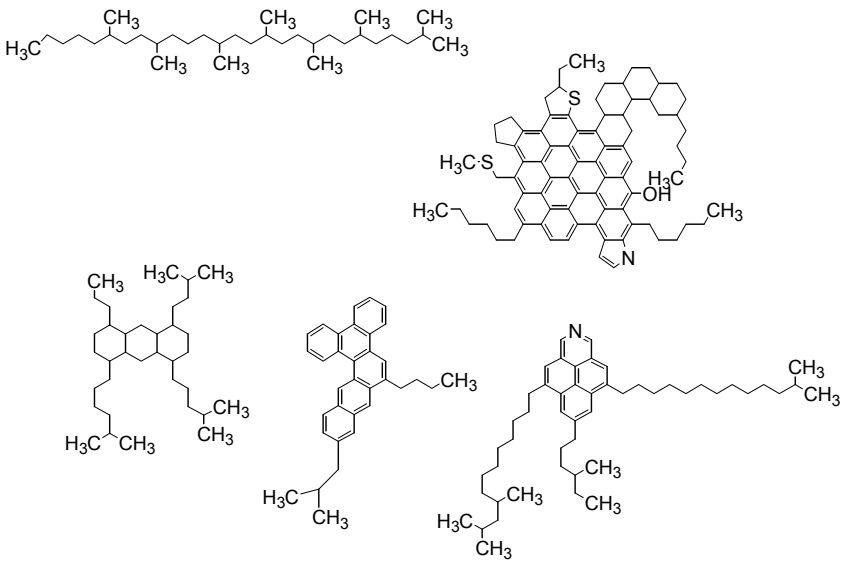
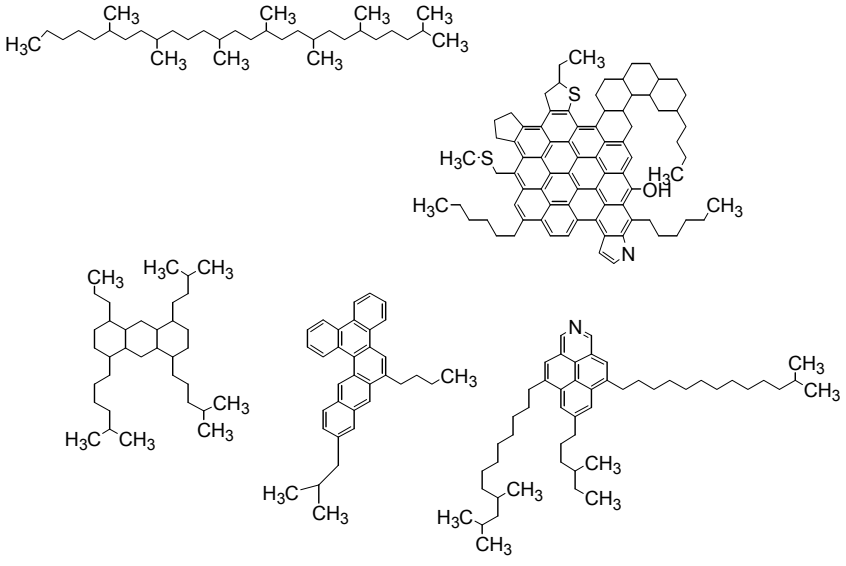
1) Asphaltenes: brittle brown-black amorphous solids, which are highly condensed aromatic compounds with molecular weight 2,000–5,000, constituting 5–25% of the weight of asphalts. They comprise one or two chromophores containing 4 to 10 fused rings each, with a significant number of alkyl substituents.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

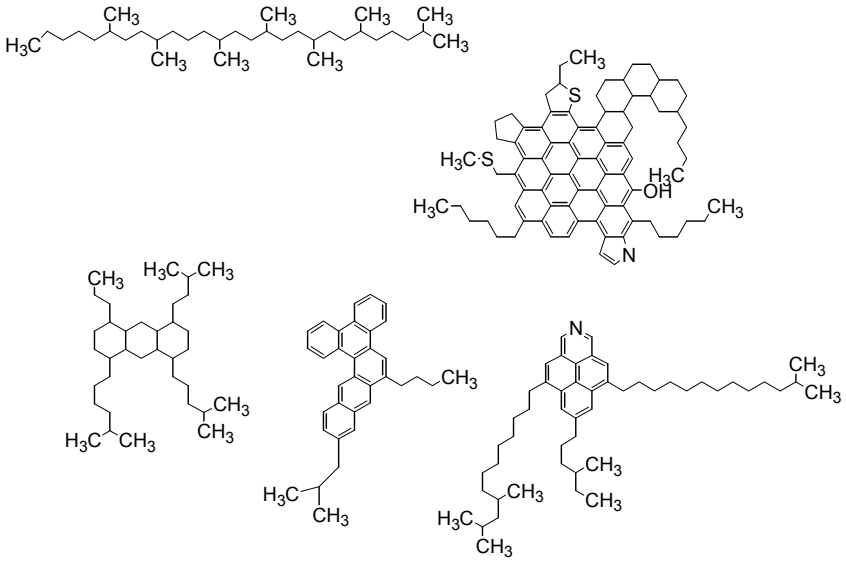
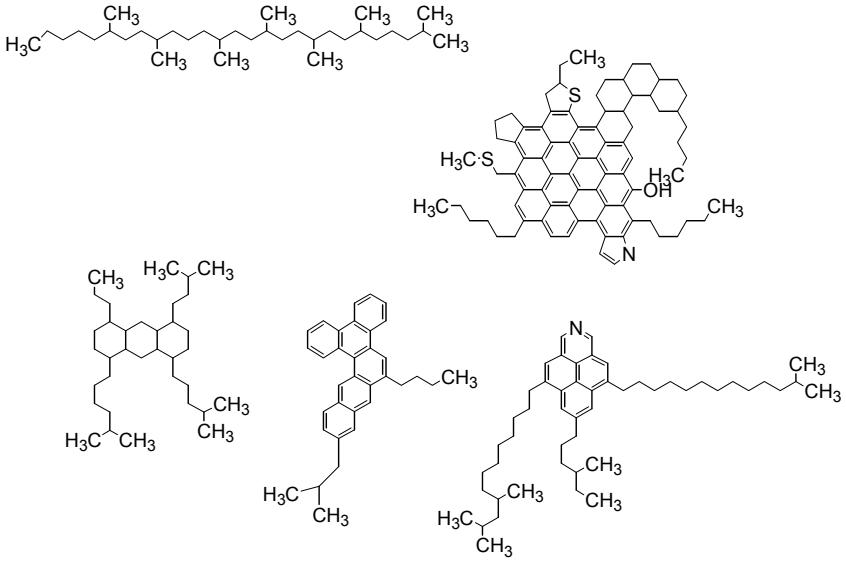
2) Resins: brown-black, adhesive, shiny solids or semi-solids. Composed of heterogeneous polar aromatic compounds with small amounts of oxygen, nitrogen, and sulfur with molecular weights of 800–2,000, constituting 15–25% of the weight of asphalts.

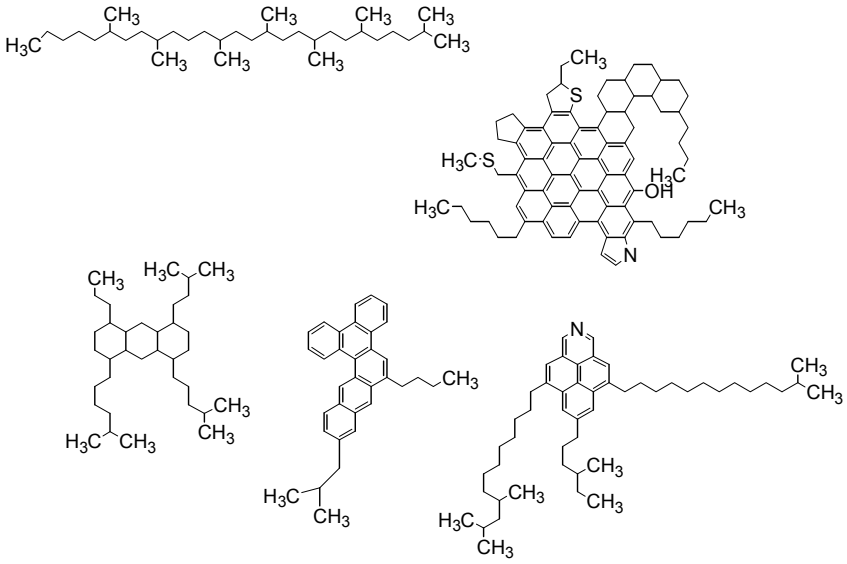
3) Aromatic oil components: viscous dark brown liquids containing mainly carbon, hydrogen, and sulfur with minor amounts of oxygen and nitrogen, with a molecular weight of 500–900, constituting 45–60% of the weight of the asphalt.

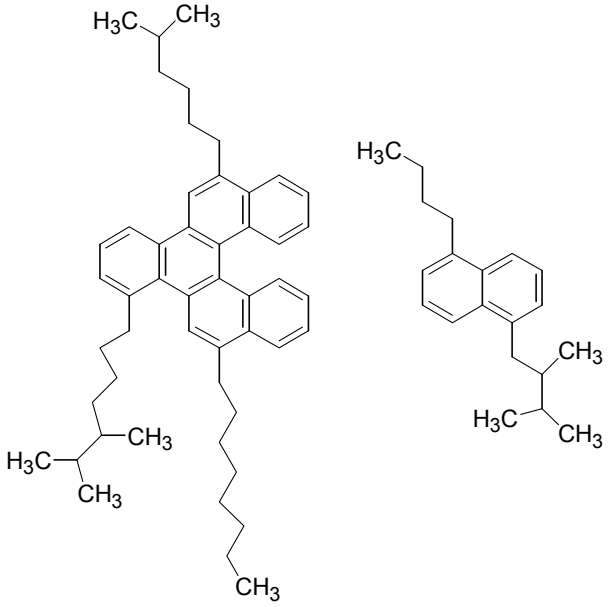
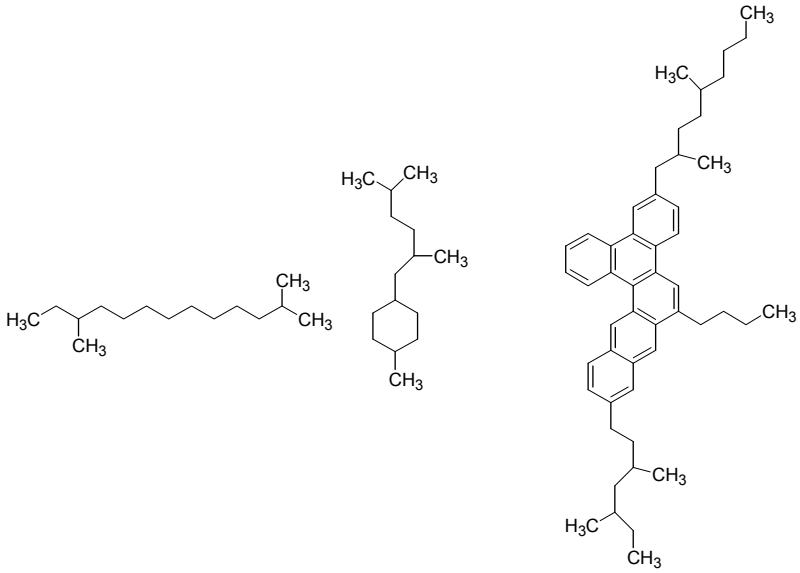
4) Saturated oil components: viscous liquids or solids ranging from straw to water-white in color, consisting mainly of long chain saturated hydrocarbons with some branched chain compounds, alkyl aromatics with long side chains, and cyclic paraffins (naphthenes), with a molecular weight of 500–1,000,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

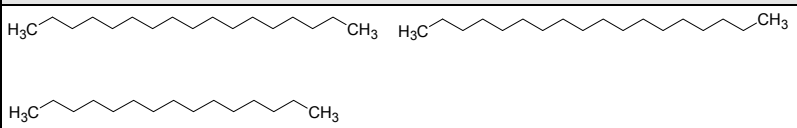
Sponsored Chemicals		
Chemical Name	**CASRN**	**Description or Chemical Structure**
Asphalt	8052-42-4	 A very complex combination of high molecular weight organic compounds containing a relatively high proportion of hydrocarbons with carbon numbers predominantly greater than C25 with high carbon-to-hydrogen 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]

[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.

1.Identification

Product name:	Petroleum resins
Catalog#:	V1528
IUPAC name:	Not available.
Product use restrictions:	Only for research and development use by, or directly under the supervision of, a technically qualified individual.
Company:	AK Scientific, Inc. 30023 Ahern Ave. Union City, CA 94587
Telephone:	(510) 429-8835
Fax:	(510) 429-8836
Website:	www.aksci.com
Emergency contact number:	1-800-633-8253 United States & Canada 1-801-629-0667 International

2.Hazard Identification:

GHS Classification

Skin irritation (Category 2)
Eye irritation (Category 2A)
Specific target organ toxicity - single exposure (Category 3), Respiratory system

Pictogram:

Signal word:

Warning

Hazard statement(s)

H315	Causes skin irritation.
H319	Causes serious eye irritation.
H335	May cause respiratory irritation.

Precautionary statement(s):

P261	Avoid breathing dust/fume/gas/mist/vapors/spray.
P264	Wash skin thoroughly after handling.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P304+P340	IF INHALED: Remove to fresh air and keep at rest in a position comfortable for breathing.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a poison center or doctor if you feel unwell.
P321	Specific treatment (see supplemental first aid instructions on this label).
P332+P313	If skin irritation occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362	Take off contaminated clothing and wash before reuse.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.
P405	Store locked up.
P501	Dispose of contents/container to an approved waste disposal plant.

Hazards not otherwise classified (HNOC) or not covered by GHS:

None

3.Composition/Information on Ingredients

Synonyms:	Not available.
CAS#:	64742-16-1
Purity:	Not available.
EC:	265-116-8

4. First Aid Measures

General Information: Immediately remove any clothing contaminated by the product. Move out of dangerous area. Consult a physician and show this safety data sheet.
Inhalation: Move person to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. Obtain medical aid.
Skin contact: Immediately flush skin with running water for at least 15 minutes while removing contaminated clothing and shoes. Wash clothing before reuse. Obtain medical aid immediately.
Eye contact: Immediately flush open eyes with running water for at least 15 minutes. Obtain medical aid immediately.
Ingestion: Do NOT induce vomiting without medical advice. Rinse mouth with water. Never administer anything by mouth to an unconscious person. Obtain medical aid immediately.
Most important symptoms and effects, both acute and delayed: No further information available. Please see sections 2 and 11.
Indication of any immediate medical attention and special treatment needed: No further information available.

5. Fire Fighting Measures

Suitable extinguishing media: Use water spray, dry chemical, carbon dioxide, or chemical foam.
Specific hazards arising from the chemical: .
Advice for firefighters: As in any fire, wear a NIOSH-approved or equivalent, pressure-demand, self-contained breathing apparatus and full protective gear. During a fire, irritating and highly toxic gases may be generated by thermal decomposition or combustion.

6. Accidental Release Measures

Personal precautions, protective equipment and emergency procedures: Wear protective equipment and keep unprotected personnel away. Ensure adequate ventilation. Remove all sources of ignition. Prevent further leak or spill if safe to do so. For personal protective equipment, please refer to section 8.
Environmental precautions: Do not let product enter drains, other waterways, or soil.
Methods and materials for containment and cleaning up: Prevent further leak or spill if safe to do so. Vacuum, sweep up, or absorb with inert material and place into a suitable disposal container. Consult local regulations for disposal. See section 13 for further disposal information.

7. Handling and Storage

Precautions for safe handling: Avoid contact with skin, eyes, and personal clothing. Wash hands thoroughly after handling. Avoid breathing fumes. Use only with adequate ventilation. Wear suitable protective clothing, gloves, and eye/face protection. Keep away from sources of ignition. Minimize dust generation and accumulation. Keep container tightly closed. Open and handle container with care. Do not eat, drink, or smoke while handling.
Conditions for safe storage, including any incompatibilities: Store in a tightly-closed

container when not in use. Store in a cool, dry, well-ventilated area away from incompatible substances. Keep away from sources of ignition.

8. Exposure Controls/Personal Protection

Exposure limits:

OSHA PEL: Not available.
NIOSH REL: Not available.
ACGIH TLV: Not available.

Appropriate engineering controls: Avoid contact with skin, eyes, and clothing. Wash hands before breaks and immediately after handling the product. Facilities storing or utilizing this material should be equipped with an eyewash fountain. Use adequate general and local exhaust ventilation to keep airborne concentrations low.

Personal protection

Eyes: Based on an evaluation of the eye or face hazards present, wear chemical splash-resistant safety glasses or goggles with side protection. A face shield may be appropriate in some workplaces. Use eyewear tested and approved under appropriate government standards such as OSHA 29 CFR 1910.133 or EU EN166.

Hands: Wear gloves selected based on an evaluation of the possible hazards to hands and skin, the duration of use, the physical conditions of the workplace, and the chemical resistance and physical properties of the glove material.

Skin and body: Protective clothing must be selected based on the hazards present in the workplace, the physical environment, the duration of exposure, and other factors. No fabric can provide protection against all potential hazards; therefore it is important to select the appropriate protective clothing for each specific hazard. At the minimum, wear a laboratory coat and close-toed footwear.

Respiratory: Respirators are not a substitute for accepted engineering control measures such as enclosure or confinement of the operation, general and local ventilation, and substitution of less toxic materials. When respiratory personal protective equipment is appropriate based on an assessment of respiratory hazards in the workplace, use a NIOSH- or CEN-certified respirator.

9. Physical and Chemical Properties

Pysical State:	Not available.
Molecular Formula:	-
Molecular Weight:	-
Odor:	Not available.
pH:	Not available.
Boiling Point Range:	Not available.
Freezing/Melting Point:	Not available.
Flash Point:	Not available.
Evaporation Rate:	Not available.
Flammability(solid,gas):	Please see section 2.
Explosive limits:	Not available.
Vapor Pressure:	Not available.
Vapor Density:	Not available.
Solubility:	Not available.
Relative Density:	Not available.
Refractive Index:	Not available.
Volatility:	Not available.
Auto-ignition Temperature:	Not available.
Decomposition Temperature:	Not available.
Partition Coefficient:	Not available.

10. Stability and Reactivity

Reactivity:	Not available.
Chemical stability:	Stable under recommended temperatures and pressures.
Possibility of hazardous reactions:	Not available.
Conditions to avoid:	Dust generation.
Incompatible materials:	Strong oxidizing agents.
Hazardous decomposition products:	.

11. Toxicological Information

RTECS#	Not available.
Acute toxicity:	Not available.
Routes of exposure:	Inhalation,eye contact,skin contact,ingestion.
Symptoms related to the physical,chemical and toxicological characteristics:	Skin contact may result in inflammation characterized by itching, scaling, reddening, blistering, pain or dryness. Eye contact may result in redness, pain or severe eye damage. Inhalation may cause irritation of the lungs and respiratory system. Overexposure may result in serious illness or death.

Carcinogenicity

IARC:	Not classified.
NTP:	Not listed.
OSHA:	Not listed.
Acute toxic effects:	Inflammation of the eye is characterized by redness, watering, and itching. Skin inflammation is characterized by itching, scaling, reddening, or, occasionally, blistering.

12. Ecological Information

Ecotoxicity:	Not available.
Persistence and degradability:	Not available.
Bioaccumulative potential:	Not available.
Mobility in soil:	Not available.
Other adverse effects:	Not available.

13. Disposal Considerations

Disposal of waste:	Chemical waste generators must determine whether a discarded chemical is classified as hazardous waste. US EPA guidelines for the classification determination are listed in 40 CFR 261.3. Additionally, waste generators must consult state and local hazardous waste regulations to ensure complete and accurate classification. Observe all federal, state and local regulations when disposing of the substance.
Disposal of packaging:	Do not reuse containers. Dispose of as unused product.

14. Transportation Information

DOT(United States)

UN number:	not hazardous material.
Proper shipping name:	Not available.
Transport hazard class:	Not available.
Packing group:	Not available.

IATA

UN Number:	not dangerous goods.
Proper shipping name:	Not available.
Transport hazard class:	Not available.
Packing group:	Not available.

15. Regulatory Information

TSCA Chemical Inventory:
This product is on the EPA Toxic Substance Control Act (TSCA) inventory. The product is supplied solely for use in research and development by or under the supervision of a technically qualified individual as defined in 40 CFR § 720 et seq. The health risks have not been fully determined. Any information that is or becomes available will be supplied on the SDS.

California Proposition 65:	Not listed.	
NFPA Rating:	Health:	Not available.
	Flammability:	Not available.
	Instability:	Not available.

16. Additional Information

Revision Date: 02/25/2019
Printed Date: 3/28/2019

The information above is believed to be accurate and represents the best information currently available to us. However, we make no warranty of merchantability or any other warranty, express or implied, with respect to such information, and we assume no liability resulting from its use. Users should make their own investigations to determine the suitability of the information for their particular purposes. In no event shall AK Scientific be liable for any claims, losses, or damages of any third party or for lost profits or any special, indirect, incidental, consequential or exemplary damages, howsoever arising, even if AK Scientific has been advised of the possibility of such damages.