Rubberized Asphalt Coating & Adhesive

Gardner-Gibson, Inc.

Version No: **1.2**
Safety Data Sheet according to OSHA HazCom Standard (2012) requirements

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SECTION 1 Identification

Product Identifier

Product name	Rubberized Asphalt Coating & Adhesive
Synonyms	APOC 139 FLEX-SHIELD Rubberized Adhesive & Coating; APOC 139 FLEX-SHIELD Thermoplastic Rubber Adhesive & Coating; Rubberized Asphalt Base Coat
Proper shipping name	Tars, liquid including road oils and cutback bitumens
Other means of identification	Not Available

Recommended use of the chemical and restrictions on use

Relevant identified uses	Roof Coating; Roof Adhesive

Name, address, and telephone number of the chemical manufacturer, importer, or other responsible party

Registered company name	**Gardner-Gibson, Inc.**
Address	4161 East 7th Avenue Tampa FL 33605 United States
Telephone	1-813-248-2101
Fax	1-813-248-6768
Website	www.icpgroup.com
Email	sds@icpgroup.com

Emergency phone number

Association / Organisation	**ChemTel**
Emergency telephone numbers	1-800-255-3924
Other emergency telephone numbers	1-813-248-0585

SECTION 2 Hazard(s) identification

Classification of the substance or mixture

NFPA 704 diamond

Note: The hazard category numbers found in GHS classification in section 2 of this SDSs are NOT to be used to fill in the NFPA 704 diamond. Blue = Health Red = Fire Yellow = Reactivity White = Special (Oxidizer or water reactive substances)

Classification	Flammable Liquids Category 3, Serious Eye Damage/Eye Irritation Category 2A, Germ Cell Mutagenicity Category 1A, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Skin Corrosion/Irritation Category 2, Carcinogenicity Category 1A, Sensitisation (Skin) Category 1, Specific Target Organ Toxicity - Repeated Exposure Category 1, Aspiration Hazard Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 3

Label elements

Hazard pictogram(s)	
Signal word	**Danger**

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Hazard statement(s)

H226	Flammable liquid and vapour.
H319	Causes serious eye irritation.
H340	May cause genetic defects.
H336	May cause drowsiness or dizziness.
H335	May cause respiratory irritation.
H315	Causes skin irritation.
H350	May cause cancer.
H317	May cause an allergic skin reaction.
H372	Causes damage to organs through prolonged or repeated exposure.
H304	May be fatal if swallowed and enters airways.
H412	Harmful to aquatic life with long lasting effects.

Hazard(s) not otherwise classified

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P233	Keep container tightly closed.
P260	Do not breathe mist/vapours/spray.
P271	Use in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P240	Ground/bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use only non-sparking tools.
P243	Take precautionary measures against static discharge.
P261	Avoid breathing mist/vapours/spray.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P202	Do not handle until all safety precautions have been read and understood.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing must not be allowed out of the workplace.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P331	Do NOT induce vomiting.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.
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/doctor/physician/first aider/if you feel unwell.
P314	Get medical advice/attention if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P302+P352	IF ON SKIN: Wash with plenty of water.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water/shower.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

P403+P235	Store in a well-ventilated place. Keep cool.
P405	Store locked up.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

Continued...

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
8052-42-4	30-60	bitumen (petroleum)
8052-41-3.	10-30	white spirit
95-63-6	1-5	1,2,4-trimethyl benzene
108-67-8	1-5	1,3,5-trimethyl benzene
12002-43-6	1-5	gilsonite
64742-95-6	1-5	naphtha petroleum, light aromatic solvent
25551-13-7	0.1-1	trimethylbenzene (mixed isomers)
14808-60-7	0.1-1	silica crystalline - quartz
13463-67-7	0.1-1	titanium dioxide

The specific chemical identity and/or exact percentage (concentration) of composition has been withheld as a trade secret.

SECTION 4 First-aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Wash out immediately with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation. ▸ Immediately drench burn area in cold running water. ▸ If hot bitumen adheres to the skin, **DO NOT** attempt to remove it (it acts as a sterile dressing). ▸ For burns to the head and neck and trunk, apply cold wet towels to the burn area, and change frequently to maintain cooling. ▸ Cooling should be maintained for no longer than thirty minutes. ▸ When hot bitumen completely encircles a limb, it may have a tourniquet effect and should be split as it cools. ▸ Transport to hospital or doctor. For thermal burns: ▸ Decontaminate area around burn. ▸ Consider the use of cold packs and topical antibiotics. For first-degree burns (affecting top layer of skin) ▸ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides. ▸ Use compresses if running water is not available. ▸ Cover with sterile non-adhesive bandage or clean cloth. ▸ Do NOT apply butter or ointments; this may cause infection. ▸ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur. For second-degree burns (affecting top two layers of skin) ▸ Cool the burn by immerse in cold running water for 10-15 minutes. ▸ Use compresses if running water is not available. ▸ Do NOT apply ice as this may lower body temperature and cause further damage. ▸ Do NOT break blisters or apply butter or ointments; this may cause infection. ▸ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape. To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort): ▸ Lay the person flat. ▸ Elevate feet about 12 inches. ▸ Elevate burn area above heart level, if possible. ▸ Cover the person with coat or blanket. ▸ Seek medical assistance. For third-degree burns Seek immediate medical or emergency assistance. In the mean time: ▸ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound. ▸ Separate burned toes and fingers with dry, sterile dressings. ▸ Do not soak burn in water or apply ointments or butter; this may cause infection. ▸ To prevent shock see above. ▸ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. ▸ Have a person with a facial burn sit up. ▸ Check pulse and breathing to monitor for shock until emergency help arrives. In case of burns: ▸ Immediately apply cold water to burn either by immersion or wrapping with saturated clean cloth. ▸ **DO NOT** remove or cut away clothing over burnt areas. **DO NOT** pull away clothing which has adhered to the skin as this can cause further injury. ▸ **DO NOT** break blister or remove solidified material. ▸ Quickly cover wound with dressing or clean cloth to help prevent infection and to ease pain. ▸ For large burns, sheets, towels or pillow slips are ideal; leave holes for eyes, nose and mouth. ▸ **DO NOT** apply ointments, oils, butter, etc. to a burn under any circumstances. ▸ Water may be given in small quantities if the person is conscious. ▸ Alcohol is not to be given under any circumstances. ▸ Reassure. ▸ Treat for shock by keeping the person warm and in a lying position. ▸ Seek medical aid and advise medical personnel in advance of the cause and extent of the injury and the estimated time of arrival of the

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	patient.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus. ▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice. ▸ Avoid giving milk or oils. ▸ Avoid giving alcohol.

Most important symptoms and effects, both acute and delayed

See Section 11

Indication of any immediate medical attention and special treatment needed

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.
For petroleum distillates
· In case of ingestion, gastric lavage with activated charcoal can be used promptly to prevent absorption - decontamination (induced emesis or lavage) is controversial and should be considered on the merits of each individual case; of course the usual precautions of an endotracheal tube should be considered prior to lavage, to prevent aspiration.
· Individuals intoxicated by petroleum distillates should be hospitalized immediately, with acute and continuing attention to neurologic and cardiopulmonary function.
· Positive pressure ventilation may be necessary.
· Acute central nervous system signs and symptoms may result from large ingestions of aspiration-induced hypoxia.
· After the initial episode,individuals should be followed for changes in blood variables and the delayed appearance of pulmonary oedema and chemical pneumonitis. Such patients should be followed for several days or weeks for delayed effects, including bone marrow toxicity, hepatic and renal impairment Individuals with chronic pulmonary disease will be more seriously impaired, and recovery from inhalation exposure may be complicated.
· Gastrointestinal symptoms are usually minor and pathological changes of the liver and kidneys are reported to be uncommon in acute intoxications.
· Chlorinated and non-chlorinated hydrocarbons may sensitize the heart to epinephrine and other circulating catecholamines so that arrhythmias may occur.Careful consideration of this potential adverse effect should precede administration of epinephrine or other cardiac stimulants and the selection of bronchodilators.
BP America Product Safety & Toxicology Department
Burns : No attempt should be made to remove the bitumen (it acts as a sterile dressing). Cover the bitumen with tulle gras and leave for two days when any detached bitumen can be removed. Re-dress and leave for a further week. If necessary refer to a burns unit. [Manufacturer]

SECTION 5 Fire-fighting measures

Extinguishing media

▸ Do NOT direct a solid stream of water or foam into burning molten material; this may cause spattering and spread the fire.

Special hazards arising from the substrate or mixture

Fire Incompatibility	▸ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

Special protective equipment and precautions for fire-fighters

Fire Fighting	▸ When silica dust is dispersed in air, firefighters should wear inhalation protection as hazardous substances from the fire may be adsorbed on the silica particles. ▸ When heated to extreme temperatures, (>1700 deg.C) amorphous silica can fuse.
Fire/Explosion Hazard	▸ Liquid and vapour are flammable. ▸ Moderate fire hazard when exposed to heat or flame. ▸ Vapour forms an explosive mixture with air. ▸ Moderate explosion hazard when exposed to heat or flame. ▸ Vapour may travel a considerable distance to source of ignition. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). Combustion products include: carbon dioxide (CO2) ▸ When silica dust is dispersed in air, firefighters should wear inhalation protection as hazardous substances from the fire may be adsorbed on the silica particles. ▸ When heated to extreme temperatures, (>1700 deg.C) amorphous silica can fuse. carbon monoxide (CO) nitrogen oxides (NOx) sulfur oxides (SOx) sulfur dioxide (SO2) silicon dioxide (SiO2) metal oxides other pyrolysis products typical of burning organic material. May emit clouds of acrid smoke

SECTION 6 Accidental release measures

Continued...

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	▸ Remove all ignition sources. ▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Control personal contact with the substance, by using protective equipment. ▸ Contain and absorb small quantities with vermiculite or other absorbent material. ▸ Wipe up. ▸ Collect residues in a flammable waste container.
Major Spills	Chemical Class: aliphatic hydrocarbons For release onto land: recommended sorbents listed in order of priority. SORBENT TYPE | RANK | APPLICATION | COLLECTION | LIMITATIONS LAND SPILL - SMALL cross-linked polymer - particulate | 1 | shovel | shovel | R, W, SS cross-linked polymer - pillow | 1 | throw | pitchfork | R, DGC, RT wood fiber - pillow | 2 | throw | pitchfork | R, P, DGC, RT treated wood fibre- pillow | 2 | throw | pitchfork | DGC, RT sorbent clay - particulate | 3 | shovel | shovel | R, I, P foamed glass - pillow | 3 | throw | pitchfork | R, P, DGC, RT LAND SPILL - MEDIUM cross-linked polymer - particulate | 1 | blower | skiploader | R,W, SS cross-linked polymer - pillow | 2 | throw | skiploader | R, DGC, RT sorbent clay - particulate | 3 | blower | skiploader | R, I, P polypropylene - particulate | 3 | blower | skiploader | W, SS, DGC expanded mineral - particulate | 4 | blower | skiploader | R, I, W, P, DGC polypropylene - mat | 4 | throw | skiploader | DGC, RT Legend DGC: Not effective where ground cover is dense R; Not reusable I: Not incinerable P: Effectiveness reduced when rainy RT:Not effective where terrain is rugged SS: Not for use within environmentally sensitive sites W: Effectiveness reduced when windy Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control; R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988

Personal Protective Equipment advice is contained in Section 8 of the SDS.

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	Hydrogen sulfide (H2S or Sour Gas) may be present when loading and unloading transport vessels. Stay upwind and away from newly opened hatches and allow to vent thoroughly before handling material. Steam may be used to vent hatches. Keep all sources of ignition away from loading area. The conductivity of this material may make it a static accumulator., A liquid is typically considered nonconductive if its conductivity is below 100 pS/m and is considered semi-conductive if its conductivity is below 10 000 pS/m., Whether a liquid is nonconductive or semi-conductive, the precautions are the same., A number of factors, for example liquid temperature, presence of contaminants, and anti-static additives can greatly influence the conductivity of a liquid. Even with proper grounding and bonding, this material can still accumulate an electrostatic charge. If sufficient charge is allowed to accumulate, electrostatic discharge and ignition of flammable air-vapour mixtures can occur. ▸ Containers, even those that have been emptied, may contain explosive vapours. ▸ Do NOT cut, drill, grind, weld or perform similar operations on or near containers. · Electrostatic discharge may be generated during pumping - this may result in fire. · Ensure electrical continuity by bonding and grounding (earthing) all equipment. · Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (<=1 m/sec until fill pipe submerged to twice its diameter, then <= 7 m/sec). · Avoid splash filling. · Do NOT use compressed air for filling discharging or handling operations. · Wait 2 minutes after tank filling (for tanks such as those on · road tanker vehicles) before opening hatches or manholes. · Wait 30 minutes after tank filling (for large storage tanks) · before opening hatches or manholes. Even with proper · grounding and bonding, this material can still accumulate an

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	· electrostatic charge. If sufficient charge is allowed to · accumulate, electrostatic discharge and ignition of flammable · air-vapour mixtures can occur. Be aware of handling · operations that may give rise to additional hazards that result · from the accumulation of static charges. These include but are · not limited to pumping (especially turbulent flow), mixing, · filtering, splash filling, cleaning and filling of tanks and · containers, sampling, switch loading, gauging, vacuum truck · operations, and mechanical movements. These activities may · lead to static discharge e.g. spark formation. Restrict line · velocity during pumping in order to avoid generation of · electrostatic discharge (= 1 m/s until fill pipe submerged to · twice its diameter, then = 7 m/s). Avoid splash filling. · Do NOT use compressed air for filling, discharging, or handling operations ▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of overexposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ Avoid smoking, naked lights or ignition sources. ▸ Avoid generation of static electricity. ▸ **DO NOT** use plastic buckets. ▸ Earth all lines and equipment. ▸ Use spark-free tools when handling. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions. ▸ **DO NOT** allow clothing wet with material to stay in contact with skin
Other information	▸ Store in original containers in approved flammable liquid storage area. ▸ Store away from incompatible materials in a cool, dry, well-ventilated area. ▸ **DO NOT** store in pits, depressions, basements or areas where vapours may be trapped. ▸ No smoking, naked lights, heat or ignition sources. ▸ Storage areas should be clearly identified, well illuminated, clear of obstruction and accessible only to trained and authorised personnel - adequate security must be provided so that unauthorised personnel do not have access. ▸ Store according to applicable regulations for flammable materials for storage tanks, containers, piping, buildings, rooms, cabinets, allowable quantities and minimum storage distances. ▸ Use non-sparking ventilation systems, approved explosion proof equipment and intrinsically safe electrical systems. ▸ Have appropriate extinguishing capability in storage area (e.g. portable fire extinguishers - dry chemical, foam or carbon dioxide) and flammable gas detectors. ▸ Keep adsorbents for leaks and spills readily available. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. In addition, for tank storages (where appropriate): ▸ Store in grounded, properly designed and approved vessels and away from incompatible materials. ▸ For bulk storages, consider use of floating roof or nitrogen blanketed vessels; where venting to atmosphere is possible, equip storage tank vents with flame arrestors; inspect tank vents during winter conditions for vapour/ ice build-up. ▸ Storage tanks should be above ground and diked to hold entire contents.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Packing as supplied by manufacturer. ▸ Plastic containers may only be used if approved for flammable liquid. ▸ Check that containers are clearly labelled and free from leaks. ▸ For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : Where a can is to be used as an inner package, the can must have a screwed enclosure. ▸ For materials with a viscosity of at least 2680 cSt. (23 deg. C) ▸ For manufactured product having a viscosity of at least 250 cSt. (23 deg. C) ▸ Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used. ▸ Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages ▸ In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	For alkyl aromatics: The alkyl side chain of aromatic rings can undergo oxidation by several mechanisms. The most common and dominant one is the attack by oxidation at benzylic carbon as the intermediate formed is stabilised by resonance structure of the ring. ▸ Following reaction with oxygen and under the influence of sunlight, a hydroperoxide at the alpha-position to the aromatic ring, is the primary oxidation product formed (provided a hydrogen atom is initially available at this position) - this product is often short-lived but may be stable dependent on the nature of the aromatic substitution; a secondary C-H bond is more easily attacked than a primary C-H bond whilst a tertiary C-H bond is even more susceptible to attack by oxygen ▸ Monoalkylbenzenes may subsequently form monocarboxylic acids; alkyl naphthalenes mainly produce the corresponding naphthalene carboxylic acids. ▸ Oxidation in the presence of transition metal salts not only accelerates but also selectively decomposes the hydroperoxides. ▸ Hock-rearrangement by the influence of strong acids converts the hydroperoxides to hemiacetals. Peresters formed from the hydroperoxides undergo Criegee rearrangement easily. ▸ Alkali metals accelerate the oxidation while CO2 as co-oxidant enhances the selectivity. ▸ Microwave conditions give improved yields of the oxidation products. ▸ Photo-oxidation products may occur following reaction with hydroxyl radicals and NOx - these may be components of photochemical smogs. Oxidation of Alkylaromatics: T.S.S Rao and Shubhra Awasthi: E-Journal of Chemistry Vol 4, No. 1, pp 1-13 January 2007

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- Vigorous reactions, sometimes amounting to explosions, can result from the contact between aromatic rings and strong oxidising agents.
- Aromatics can react exothermically with bases and with diazo compounds.

Hydrogen sulfide (H2S):

- is a highly flammable and reactive gas
- reacts violently with strong oxidisers, metal oxides, metal dusts and powders, bromine pentafluoride, chlorine trifluoride, chromium trioxide, chromyl chloride, dichlorine oxide, nitrogen trichloride, nitryl hypofluorite, oxygen difluoride, perchloryl fluoride, phospham, phosphorus persulfide, silver fulminate, soda-lime, sodium peroxide
- is incompatible with acetaldehyde, chlorine monoxide, chromic acid, chromic anhydride, copper, nitric acid, phenyldiazonium chloride, sodium
- forms explosive material with benzenediazonium salts
- attacks many metals

Flow or agitation of hydrogen sulfide may generate electrostatic charges due to low conductivity

The substance may be or contains a "metalloid"
The following elements are considered to be metalloids; boron,silicon, germanium, arsenic, antimony, tellurium and (possibly) polonium
The electronegativities and ionisation energies of the metalloids are between those of the metals and nonmetals, so the metalloids exhibit characteristics of both classes. The reactivity of the metalloids depends on the element with which they are reacting. For example, boron acts as a nonmetal when reacting with sodium yet as a metal when reacting with fluorine.
Unlike most metals, most metalloids are amphoteric- that is they can act as both an acid and a base. For instance, arsenic forms not only salts such as arsenic halides, by the reaction with certain strong acid, but it also forms arsenites by reactions with strong bases.
Most metalloids have a multiplicity of oxidation states or valences. For instance, tellurium has the oxidation states +2, -2, +4, and +6. Metalloids react like non-metals when they react with metals and act like metals when they react with non-metals.

- Sulfides are incompatible with acids, diazo and azo compounds, halocarbons, isocyanates, aldehydes, alkali metals, nitrides, hydrides, and other strong reducing agents.
- Many reactions of sulfides with these materials generate heat and in many cases hydrogen gas.
- Many sulfide compounds may liberate hydrogen sulfide upon reaction with an acid.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US OSHA Permissible Exposure Limits (PELs) Table Z-1	bitumen (petroleum)	Particulates Not Otherwise Regulated (PNOR)- Respirable fraction	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	bitumen (petroleum)	Particulates Not Otherwise Regulated (PNOR)- Total dust	15 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	bitumen (petroleum)	Inert or Nuisance Dust: Respirable fraction	5 mg/m3 / 15 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	bitumen (petroleum)	Inert or Nuisance Dust: Total Dust	15 mg/m3 / 50 mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	bitumen (petroleum)	Asphalt fumes	Not Available	Not Available	5 (15-minute) mg/m3	Ca; See Appendix A, Appendix C
US OSHA Permissible Exposure Limits (PELs) Table Z-1	white spirit	Stoddard solvent	500 ppm / 2900 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	white spirit	Stoddard solvent	350 mg/m3	Not Available	1800 (15-minute) mg/m3	Not Available
US NIOSH Recommended Exposure Limits (RELs)	1,2,4-trimethyl benzene	1,2,4-Trimethylbenzene	25 ppm / 125 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	1,3,5-trimethyl benzene	1,3,5-Trimethylbenzene	25 ppm / 125 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	gilsonite	Particulates Not Otherwise Regulated (PNOR)- Total dust	15 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	gilsonite	Particulates Not Otherwise Regulated (PNOR)- Respirable fraction	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	gilsonite	Inert or Nuisance Dust: Total Dust	15 mg/m3 / 50 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	gilsonite	Inert or Nuisance Dust: Respirable fraction	5 mg/m3 / 15 mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	gilsonite	Particulates not otherwise regulated	Not Available	Not Available	Not Available	See Appendix D
US OSHA Permissible Exposure Limits (PELs) Table Z-1	silica crystalline - quartz	Quartz - respirable	0.05 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	silica crystalline - quartz	Silica: Crystalline: Quartz (Respirable)	10 (%SiO2+2) mg/m3 / 250 (%SiO2+5) mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	silica crystalline - quartz	Silica, crystalline (as respirable dust)	0.05 mg/m3	Not Available	Not Available	Ca; See Appendix A
US OSHA Permissible Exposure Limits (PELs) Table Z-1	titanium dioxide	Titanium dioxide - Total dust	15 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	titanium dioxide	Inert or Nuisance Dust: Respirable fraction	5 mg/m3 / 15 mppcf	Not Available	Not Available	Not Available

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US OSHA Permissible Exposure Limits (PELs) Table Z-3	titanium dioxide	Inert or Nuisance Dust: Total Dust	15 mg/m3 / 50 mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	titanium dioxide	Titanium dioxide	Not Available	Not Available	Not Available	Ca; See Appendix A

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
bitumen (petroleum)	30 mg/m3	330 mg/m3	2,000 mg/m3
white spirit	300 mg/m3	1,800 mg/m3	29500** mg/m3
1,2,4-trimethyl benzene	140 mg/m3	360 mg/m3	2,200 mg/m3
1,2,4-trimethyl benzene	Not Available	Not Available	480 ppm
1,3,5-trimethyl benzene	Not Available	Not Available	480 ppm
naphtha petroleum, light aromatic solvent	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3
silica crystalline - quartz	0.075 mg/m3	33 mg/m3	200 mg/m3
titanium dioxide	30 mg/m3	330 mg/m3	2,000 mg/m3

Ingredient	Original IDLH	Revised IDLH
bitumen (petroleum)	Not Available	Not Available
white spirit	20,000 mg/m3	Not Available
1,2,4-trimethyl benzene	Not Available	Not Available
1,3,5-trimethyl benzene	Not Available	Not Available
gilsonite	Not Available	Not Available
naphtha petroleum, light aromatic solvent	Not Available	Not Available
trimethylbenzene (mixed isomers)	Not Available	Not Available
silica crystalline - quartz	25 mg/m3 / 50 mg/m3	Not Available
titanium dioxide	5,000 mg/m3	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
naphtha petroleum, light aromatic solvent	E	≤ 0.1 ppm
trimethylbenzene (mixed isomers)	E	≤ 0.1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

WARNING: For inhalation exposure **ONLY:**
This substance has been classified by the ACGIH as A2 Suspected Human Carcinogen.

WARNING: This substance is classified by the NOHSC as Category 2 Probable Human Carcinogen
WARNING: For inhalation exposure ONLY: This substance has been classified by the IARC as Group 1: **CARCINOGENIC TO HUMANS**

The International Agency for Research on Cancer (IARC) has classified occupational exposures to **respirable** (<5 um) crystalline silica as being carcinogenic to humans . This classification is based on what IARC considered sufficient evidence from epidemiological studies of humans for the carcinogenicity of inhaled silica in the forms of quartz and cristobalite. Crystalline silica is also known to cause silicosis, a non-cancerous lung disease.
Intermittent exposure produces; focal fibrosis, (pneumoconiosis), cough, dyspnoea, liver tumours.

* Millions of particles per cubic foot (based on impinger samples counted by light field techniques).
NOTE : the physical nature of quartz in the product determines whether it is likely to present a chronic health problem. To be a hazard the material must enter the breathing zone as respirable particles.

These exposure guidelines have been derived from a screening level of risk assessment and should not be construed as unequivocally safe limits. ORGS represent an 8-hour time-weighted average unless specified otherwise.
CR = Cancer Risk/10000; UF = Uncertainty factor:
TLV believed to be adequate to protect reproductive health:
LOD: Limit of detection
Toxic endpoints have also been identified as:
D = Developmental; R = Reproductive; TC = Transplacental carcinogen
Jankovic J., Drake F.: A Screening Method for Occupational Reproductive
American Industrial Hygiene Association Journal 57: 641-649 (1996)
Animals exposed by inhalation to 10 mg/m3 titanium dioxide show no significant fibrosis, possibly reversible tissue reaction. The architecture of lung air spaces remains intact.
· The label on a package containing 1% or more of titanium oxide with aerodynamic diameter equal or below 10 microns shall bear the following statement: EUH211 "Warning! Hazardous respirable droplets may be formed when sprayed. Do NOT breathe spray or mist
· The label on the packaging of solid mixtures containing 1% or more of titanium dioxide shall bear the following statement: EUH212" "Warning! Hazardous respirable dust may be formed when used. Do not breathe dust".
In addition, the label on the packaging of liquid and solid mixtures not intended for the general public and not classified as hazardous which are labelled EUH211 or EU212 shall bear statement EUH210: "Safety data sheet available on request."

Continued...

bitumen (asphalt) fumes [8052-42-4]
TLV* TWA: 0.5 mg/m3 A4 asphalt (petroleum, bitumen) fume, as benzene soluble aerosol
ES* TWA: 5 mg/m3 as fumes
OES* TWA: 5 mg/m3; STEL: 10 mg/m3 as fumes
Based on surveys of asphalt workers in oil refineries and in the roofing industry the TLV-TWA is thought to reduce the risk of possible carcinogenicity
For white spirit:
Low and high odour thresholds of 5.25 and 157.5 mg/m3, respectively, were considered to provide a rather useful index of odour as a warning property.
The TLV-TWA is calculated from data on the toxicities of the major ingredients and is intended to minimise the potential for irritative and narcotic effects, polyneuropathy and kidney damage produced by vapours.
The NIOSH (USA) REL-TWA of 60 ppm is the same for all refined petroleum solvents. NIOSH published an occupational "action level" of 350 mg/m3 for exposure to Stoddard solvent, assuming a 10-hour work shift and a 40-hour work-week. The NIOSH-REL ceiling of 1800 mg/m3 was established to protect workers from short-term effects that might produce vertigo or other adverse effects which might increase the risk of occupational accidents. Combined (gross) percutaneous absorption and inhalation exposure (at concentrations associated with nausea) are thought, by some, to be responsible for the development of frank hepatic toxicity and jaundice.
Odour Safety Factor (OSF)
OSF=0.042 (white spirit)
For trimethyl benzene as mixed isomers (of unstated proportions)
Odour Threshold Value: 2.4 ppm (detection)
Use care in interpreting effects as a single isomer or other isomer mix. Trimethylbenzene is an eye, nose and respiratory irritant. High concentrations cause central nervous system depression. Exposed workers show CNS changes, asthmatic bronchitis and blood dyscrasias at 60 ppm. The TLV-TWA is thought to be protective against the significant risk of CNS excitation, asthmatic bronchitis and blood dyscrasias associated with exposures above the limit.
Odour Safety Factor (OSF)
OSF=10 (1,2,4-TRIMETHYLBENZENE)

Exposed individuals are **NOT** reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

Class	OSF	Description
A	550	Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities
B	26-550	As "A" for 50-90% of persons being distracted
C	1-26	As "A" for less than 50% of persons being distracted
D	0.18-1	10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached
E	<0.18	As "D" for less than 10% of persons aware of being tested

Because the margin of safety of the quartz TLV is not known with certainty and given the associated link between silicosis and lung cancer it is recommended that quartz concentrations be maintained as far below the TLV as prudent practices will allow.
Exposure to respirable crystalline silicas (RCS) represents a significant hazard to workers, particularly those employed in the construction industry where respirable dusts of of cement and concrete are common. Cutting, grinding and other high speed processes, involving their finished products, may further result in dusty atmospheres. Bricks are also a potential source of RCSs under such circumstances.
It is estimated that half of the occupations, involved in construction work, are exposed to levels of RCSs, higher than the current allowable limits. Beaudry et al: Journal of Occupational and Environmental Hygiene 10: 71-77; 2013

Exposure controls

Appropriate engineering controls

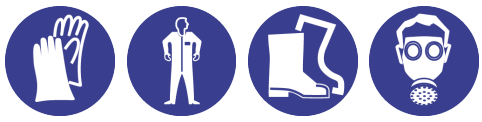
For molten materials:
Provide mechanical ventilation; in general such ventilation should be provided at compounding/ converting areas and at fabricating/ filling work stations where the material is heated. Local exhaust ventilation should be used over and in the vicinity of machinery involved in handling the molten material.
Keep dry!!
Processing temperatures may be well above boiling point of water, so wet or damp material may cause a serious steam explosion if used in unvented equipment.
Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.
For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant.
Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity

Continued...

	3: Intermittent, low production. | 3: High production, heavy use 4: Large hood or large air mass in motion | 4: Small hood-local control only Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used. · Adequate ventilation is typically taken to be that which limits the average concentration to no more than 25% of the LEL within the building, room or enclosure containing the dangerous substance. · Ventilation for plant and machinery is normally considered adequate if it limits the average concentration of any dangerous substance that might potentially be present to no more than 25% of the LEL. However, an increase up to a maximum 50% LEL can be acceptable where additional safeguards are provided to prevent the formation of a hazardous explosive atmosphere. For example, gas detectors linked to emergency shutdown of the process might be used together with maintaining or increasing the exhaust ventilation on solvent evaporating ovens and gas turbine enclosures. · Temporary exhaust ventilation systems may be provided for non-routine higher-risk activities, such as cleaning, repair or maintenance in tanks or other confined spaces or in an emergency after a release. The work procedures for such activities should be carefully considered.. The atmosphere should be continuously monitored to ensure that ventilation is adequate and the area remains safe. Where workers will enter the space, the ventilation should ensure that the concentration of the dangerous substance does not exceed 10% of the LEL (irrespective of the provision of suitable breathing apparatus)
Personal protection	
Eye and face protection	‣ Safety glasses with side shields. ‣ Chemical goggles. ‣ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	‣ Wear chemical protective gloves, e.g. PVC. ‣ Wear safety footwear or safety gumboots, e.g. Rubber **NOTE:** ‣ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ‣ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Body protection	See Other protection below

Continued...

Other protection	▸ Overalls. ▸ PVC Apron. ▸ PVC protective suit may be required if exposure severe. ▸ Eyewash unit. ▸ Ensure there is ready access to a safety shower. ▸ Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity. ▸ For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets). ▸ Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
Rubberized Asphalt Coating & Adhesive

Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
TEFLON	C
VITON	C

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

Type A-P Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required. Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

^ - Full-face
A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Bitumen (known as asphalt in the U.S.) "is the residuum produced from the non-destructive distillation of crude petroleum at atmospheric pressure and/ or under reduced pressures or absence of steam. Bitumens/ asphalts are composed mainly of high-molecular-weight alkylaryl hydrocarbons with high carbon to hydrogen ratios, with carbon numbers > C25, boiling points >400 "C, high viscosity, and negligible water solubility and vapor pressure. These bitumen/ asphalt alkylaryl hydrocarbons are a heterogeneous mixture of linear, branched and cyclic, saturated and unsaturated, and aromatic functional groups. Importantly, polycyclic aromatic hydrocarbons (PAH) such as benzo(a)pyrene, which are toxicologically significant, are only present in bitumen/ asphalt feedstock at very low concentrations. Bitumens/ asphalts contain much larger proportions of high-molecular-weight paraffinic and naphthenic hydrocarbons that are substituted with alkyl groups and ultimately sulfonated, which reduces their potential to exhibit PAH-like toxicity. In practice, the asphalt alkylaryl feedstocks are chemically characterised by a saturates, aromatics, resins, and asphaltenes. **Saturates** consist mainly of long chain saturated hydrocarbons with some Saturates branching, alkyl aromatics with long side chains, and cyclic paraffins (napthenes), with molecular weight of 500-1000. **Asphaltenes** consist mainly of substituted benzene and napthenic-aromatic nuclei with alkyl side chain constituents, with molecular weight of 500-900. **Resins** consist mainly of heterogeneous polar aromatic compounds with small amounts of oxygen, nitrogen, and sulfur, with molecular Resins weight range of 800-2000. Considered lower molecular weight asphaltenes **Asphaltenes** consist mainly of highly condensed aromatic compounds with one or two chromophores containing 4 to 10 fused rings each, with a significant number of alkyl constituents. They have a molecular weight range of 500-1000. The bitumen/ asphalt group is defined by the following six CAS Numbers: asphalt (penetration or hard) (CAS No. 8052-42-4); vacuum residues (CAS No. 64741-56-6); raffinates, residual oil decarbonization (CAS No. 64742-07-0); petroleum resins (CAS No. 64742-16-1); residues, hydrodesulfurised vacuum (CAS No. 64742-85-4); and asphalt, oxidised (CAS No. 64742-93-4). Small amounts of metals such as nickel, iron or vanadium may be present. Bitumen/ asphalt fumes must also be considered in an occupational setting and as fugitive emissions.

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Physical state	Liquid	**Relative density (Water = 1)**	1.03
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	40.5	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Flammable.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	33.83
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Not Available	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	<300

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	‣ Extremely high temperatures. ‣ Unstable in the presence of incompatible materials. ‣ Product is considered stable. ‣ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. The material has **NOT** been classified by EC Directives or other classification systems as "harmful by inhalation". This is because of the lack of corroborating animal or human evidence. In the absence of such evidence, care should be taken nevertheless to ensure exposure is kept to a minimum and that suitable control measures be used, in an occupational setting to control vapours, fumes and aerosols. High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.

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	A significant number of individuals exposed to mixed trimethylbenzenes complained of nervousness, tension, anxiety and asthmatic bronchitis. Peripheral blood showed a tendency to hypochromic anaemia and a deviation from normal in coagulability of the blood. Hydrocarbon concentrations ranged from 10 to 60 ppm. Contamination of the mixture with benzene may have been responsible for the blood dyscrasias. High concentrations of mesitylene vapour (5000 to 9000 ppm) caused central nervous system depression in mice. Similar exposures of pseudocumene also produced evidence of CNS involvement. Symptoms of hydrogen sulfide (H2S) exposure may include profuse salivation, nausea, vomiting, diarrhoea, giddiness, headache, vertigo, amnesia, palpitations, arrhythmia, weakness, muscle cramps, confusion, sudden collapse, unconsciousness and death due to respiratory paralysis (above 300 ppm). Inhalation of (H2S) at low concentrations causes headache, dizziness and upset stomach. Higher concentrations cause olfactory fatigue, irritation to the respiratory tract, excitement, confusion, and exposure for a prolonged period may cause bronchitis and pulmonary oedema. Although hydrogen sulfide is extremely odourous, the "rotten egg" odour is not a reliable indicator for warning of exposure since odour fatigue readily occurs. Odour sensation is lost immediately at concentrations exceeding 200 ppm. Case reports suggest that toxic amounts can enter the body through a punctured ear drum, even while wearing some sorts of respiratory protection. Hydrogen sulfide is primarily a respiratory toxin which inhibits the cytochrome-oxidase system and is probably more potent than hydrogen cyanide. The lifetime of hydrogen sulfide in oxygenated blood is short and sulfmethaemoglobin is rapidly detoxified by red blood cells and the liver. Most fatalities due to hydrogen sulfide intoxication occur at the scene of exposure and immediate supportive care is imperative. Ensure such contingencies are addressed as part of the site emergency plan and that operators or other employees who may become accidentally exposed, are made aware of the existence of such a plan. Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination Exposure to white spirit, in a controlled inhalation study using volunteers either at rest or during exercise, (1000 or 2500 mg/m3 for 30 minutes) produced a linear relationship between alveolar and arterial concentrations of the individual solvent components. Pulmonary absorption of the aliphatics ranged from 46-59%, whilst that of aromatic ranged from 58-70%. Although systemic absorption was greater during exercise, the proportion of circulating aliphatic to aromatic components decreased with increased activity. Exposure to 2500 - 5000 mg/m3 produces nausea and vertigo. The acute toxicity of inhaled alkylbenzene is best described by central nervous system depression. These compounds may also act as general anaesthetics. Whole body symptoms of poisoning include light-headedness, nervousness, apprehension, a feeling of well-being, confusion, dizziness, drowsiness, ringing in the ears, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness, depression of breathing, and arrest. Heart stoppage may result from cardiovascular collapse. A slow heart rate and low blood pressure may also occur. Alkylbenzenes are not generally toxic except at high levels of exposure. Their breakdown products have low toxicity and are easily eliminated from the body. Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. Acute exposure to bitumen/ asphalt vapours may cause coughing, chest tightness, headache, muscle weakness, dizziness, tiredness, poor coordination, and even nausea and vomiting. Workers exposed to hot blown bitumens show bronchitis, rhinitis, oropharyngitis and laryngitis; symptoms include cough, phlegm, burning of the throat and chest, hoarseness, headache and nasal discharge. Guinea pigs, rabbits and mice exposed to blown bitumen fumes, aerosols and smoke, developed patchy regions of emphysema, bronchiolar dilation, pneumonitis, and severe localised bronchitis. Mice, exposed to aerosols of petroleum bitumens and smoke from heated petroleum bitumens, showed congestion, acute bronchitis, pneumonitis, bronchial dilation, abscess formation, epithelial atrophy, and necrosis. In health studies in the workplace, environmental measurement showed concentrations of asphalt, ranging from "non-detectable", where there was good mechanical ventilation, to 40 mg/m3, where there was very poor natural draft. Breathing zone samples, collected during drum-filling operations, ranged from 1.0 (upwind) to 5 mg/m3 (downwind) as means of 4-hour exposures. In the opinion of industrial hygienists conducting these studies, work conditions were satisfactory where asphalt fumes were kept below 10 mg/m3
Ingestion	Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result. Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis). The material has **NOT** been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern. Swallowing pieces of bitumen may produce pyloric obstruction due to accumulation in the stomach and the formation of a stony concretion. Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosal ulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea and vomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injury may produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Light aromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration into the lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage. Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. The liquid may be miscible with fats or oils and may degrease the skin, producing a skin reaction described as non-allergic contact dermatitis. The material is unlikely to produce an irritant dermatitis as described in EC Directives . Aromatic hydrocarbons may produce skin irritation, vasodilation with erythema and changes in endothelial cell permeability. Systemic intoxication, resulting from contact with the light aromatics, is unlikely due to the slow rate of permeation. Branching of the side chain appears to increase percutaneous absorption.
Eye	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.

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	Workers exposed to fumes of blown bitumens developed keratoconjunctivitis. Exposure to H2S may produce pain, blurred vision, and irritation. These symptoms are temporary in all but severe cases. Eye irritation may produce conjunctivitis, photophobia, pain, and at higher concentrations blurred vision and corneal blistering Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may also result. The aromatic fraction may produce irritation and lachrymation.
Chronic	On the basis of epidemiological data, it has been concluded that prolonged inhalation of the material, in an occupational setting, may produce cancer in humans. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. There is sufficient evidence to provide a strong presumption that human exposure to the material may produce heritable genetic damage. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in the development of heritable genetic damage, generally on the basis of - appropriate animal studies, - other relevant information Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses. Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding. Hydrocarbon solvents are liquid hydrocarbon fractions derived from petroleum processing streams, containing only carbon and hydrogen atoms, with carbon numbers ranging from approximately C5-C20 and boiling between approximately 35-370 deg C. Many of the hydrocarbon solvents have complex and variable compositions with constituents of 4 types, alkanes (normal paraffins, isoparaffins, and cycloparaffins) and aromatics (primarily alkylated one- and two-ring species). Despite the compositional complexity, most hydrocarbon solvent constituents have similar toxicological properties, and the overall toxicological hazards can be characterized in generic terms. Hydrocarbon solvents can cause chemical pneumonitis if aspirated into the lung, and those that are volatile can cause acute CNS effects and/or ocular and respiratory irritation at exposure levels exceeding occupational recommendations. Otherwise, there are few toxicologically important effects. The exceptions, n-hexane and naphthalene, have unique toxicological properties Animal studies: No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) was observed in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of a alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human. The synthetic, amorphous silicas are believed to represent a very greatly reduced silicosis hazard compared to crystalline silicas and are considered to be nuisance dusts. When heated to high temperature and a long time, amorphous silica can produce crystalline silica on cooling. Inhalation of dusts containing crystalline silicas may lead to silicosis, a disabling pulmonary fibrosis that may take years to develop. Discrepancies between various studies showing that fibrosis associated with chronic exposure to amorphous silica and those that do not may be explained by assuming that diatomaceous earth (a non-synthetic silica commonly used in industry) is either weakly fibrogenic or nonfibrogenic and that fibrosis is due to contamination by crystalline silica content Repeated exposure to synthetic amorphous silicas may produce skin dryness and cracking. Available data confirm the absence of significant toxicity by oral and dermal routes of exposure. Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted in a number of species, at airborne concentrations ranging from 0.5 mg/m3 to 150 mg/m3. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m3. When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m3. Differences in values may be due to particle size, and therefore the number of particles administered per unit dose. Generally, as particle size diminishes so does the NOAEL/ LOAEL. Exposure produced transient increases in lung inflammation, markers of cell injury and lung collagen content. There was no evidence of interstitial pulmonary fibrosis. Long term exposure to coal tar dusts may produce chronic bronchitis or lung cancer. Dust, liquid or fume contact with skin may result in photosensitisation of skin areas and sunburn on frequent exposure to sunlight or ultra-violet radiation. Workers exposed to hot tar and pitch showed abnormal serum protein levels due to liver dysfunction. Chronic exposure of mice to 0.3 mg/l of tar aerosols, for three 2 hour periods, produced necrotising tracheobronchitis and hyperplasia of the epithelium; these were occasionally accompanied by papillary infolding. Exposed body surfaces and the scrotum of long-term coal-tar pitch workers may show kerato-acanthoma ("tar mollusca"), pitch warts or tar

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warts, even after exposure has ceased; the head, neck and other extremities are particularly prone. Pitch keratosis and acanthomas (cancerous or precancerous skin lesions) may also develop. Hyperpigmentation of the body surfaces and scrotum may be localised or diffuse.
Corneal ulcers, conjunctivitis and papillomata of the lids have also been described in workers chronically exposed to coal tar pitches. Workers exposed to petroleum, tar or pitch appear to show an elevated risk of cancer of the renal pelvis. Millwrights and welders in a stamping plant, occupationally exposed to coal-tars and coal-tar pitch showed a greater incidence of leukaemia and cancers of the lung and digestive organs.
Coal tar fumes or dusts have been implicated in the development of occupational cancers. A minimal time of exposure (1-5 years) has been cited. Similarly occupational cancers may develop many years after exposure ceases. Deaths from cancer of the lungs and pleura of retired gas workers was approximately twice the expected rate. Pot-room workers in the aluminium smelting industries showed an increased rate of lung-cancer mortality. One report from the former Soviet Union associated such an increase with concentrations of tarry substances between 27 and 210 mg/m3 (B[a]P levels of 0.6 to 56 ug/m3). High respiratory mortality has been reported among coke oven workers in Great Britain whilst kidney and lung cancers were prevalent among American coke-oven workers predominantly exposed for more than 5 years.
A UK mortality analysis (in 1946) showed an increase in scrotal cancers in patent-fuel workers. Reports of skin and scrotal cancers are frequent amongst workers exposed to coal-tar fumes in coal gasification and coke production. A small excess
of bladder cancer is described in tar distillers and patent-fuel workers.
Benzene extracts of atmospheric samples from a coal tar plant, painted on the intrascapular area of black mice, three times weekly, caused tumours to appear (some occurred within 465 days). Animal studies indicate that lung and kidney tumours were induced following exposure to coal tar aerosols. The degree of lung change of rats breathing air-contaminated with polycyclic aromatic hydrocarbons (PAHs) is dose-related.
Coal-tar containing ointments have been implicated in a number of human skin cancers. Evidence exists for mutagenic action (as seen in urine samples) after application of these ointments
Follicular dermatitis may develop rapidly on repeated immersion of the hands and forearms in white spirits. A Belgian report, produced in 1958, described sub-chronic toxicity amongst workers exposed to white spirit (83% paraffins, 17% aromatics) over a 4 month period. These workers complained of nausea and vomiting and one developed aplastic anaemia; bone marrow depression was confirmed. This employee died several months later as a result of septicaemia. Bone marrow depression, associated with human exposure, might be explained by the presence of myelotoxic compounds, the most notable being benzene.
Chronic exposure to bitumen/ asphalt fumes, over extended periods, may cause central nervous system depression, and liver and kidney changes. Chronic bitumen/ asphalt poisoning may result in a decrease in the number of white and red blood cells. [*ILO Encyclopedia*]
Prolonged contact with bitumens may produce irritation, inflammation, dermatitis, acne-like lesions, keratoses, melanosis and photosensitisation.
Animal inhalation studies do NOT yield sufficient evidence of bitumen/ asphalt-induced lung cancer. It is generally accepted that oxidation of polycyclic aromatic hydrocarbons (PAHs) destroys their carcinogenic potential and the differing character of the polycyclic aromatic fraction of petroleum asphalt fume and those of coal tar pitch volatiles suggested a lessened potential for carcinogenicity.
Inhalation of fumes of heated bitumens by guinea pigs and rats produced chronic fibrosing pneumonitis with peribronchial adenomatosis; rats developed squamous cell metaplasias.
Various extracts of steam-refined and air-refined bitumens and their mixtures, undiluted steam-refined bitumens and cracking residue bitumens, produced skin tumours following application to mouse skin. Subcutaneous injection in mice and rats, of steam- and air- reined bitumens, produced sarcomas at the sites of injection. Application of air-refined bitumens, in toluene, to the skin of mice, produced skin tumours. No tumours were produced by the undiluted bitumen. A pooled mixture of steam- and air-blown petroleum bitumen in benzene, produced tumours at the site of application to mouse skin.
No significant difference was found in the health of asphalt workers and of groups of controls in a study conducted in 25 oil refineries. Other studies have not demonstrated health defects in paving and roofing operations (using asphalt-based products) and interstate trucking over asphalt highways.
NOTE: The term bitumen and asphalt are often used interchangeably and have been used to describe products derived from petroleum and/ or coal. Asphalt is a native mixture of hydrocarbons which occurs as an amorphous, brownish-black solid or semisolid and results from the evaporation of the lighter hydrocarbons from petroleum and partial oxidation of the residue. Petroleum asphalts (bitumens) should therefore be differentiated from coal pitch bitumens which result from the destructive distillation of coal.
The term "asphalt" originally applied to "Trinidad asphalt" which is a mined solid and is closely related to gilsonite.
On occasion there are reports of epidemiological studies which have found an increased cancer mortality in workers exposed to heated bitumens and bitumen fumes. There are reports of significantly increased incidence of cancers of the mouth, oesophagus, rectum and lung. The bitumens, used by this cohort, are likely to have their origin in coal and should be distinguished from materials derived from the petroleum industry (the asphalts).
Hardened bitumens/ asphalts do not normally constitute a health hazard. Mined sources of bitumens/ asphalts may present an additional hazard related to their naturally occurring content of quartz. Chronic inhalation of high levels of quartz dusts may produce silicosis, a disabling form of pneumoconiosis which may lead to scarring of the lining of the air-sacs of the lung.

Chronic low level exposures to hydrogen sulfide may produce headache, fatigue, dizziness, irritability and loss of libido. These symptoms may also result from damage produced by isolated or repeated unmeasured peak high level exposures in healthy persons or those suffering from pre-existing neurological diseases. A study on long term effects showed that H2S apparently can cause continuing, sometimes unrecognised olfactory deficits. [*Hirsch, A.R. - Occ. Env. Med.,1999, Vol 5, Iss 4, pp 284-287*]
On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

Rubberized Asphalt Coating & Adhesive	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
bitumen (petroleum)	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; >5000 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
white spirit	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >3000 mg/kg[1]	Eye (human): 470 ppm/15m
	Inhalation(Rat) LC50: >5.5 mg/l4h[1]	Eye (rabbit): 500 mg/24h moderate
	Oral (Rat) LD50; >5000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
		Skin: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
1,2,4-trimethyl benzene	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >3160 mg/kg[2]	Not Available
	Inhalation(Rat) LC50: 18 mg/L4h[2]	
	Oral (Rat) LD50; 6000 mg/kg[1]	

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1,3,5-trimethyl benzene	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >3460 mg/kg[1]	Eye (rabbit): 500 mg/24h mild
	Inhalation(Rat) LC50: 24 mg/L4h[2]	Eye: adverse effect observed (irritating)[1]
	Oral (Rat) LD50; 6000 mg/kg[1]	Skin (rabbit): 20 mg/24h moderate
		Skin: adverse effect observed (irritating)[1]
gilsonite	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
naphtha petroleum, light aromatic solvent	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >1900 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50: >4.42 mg/L4h[1]	Skin: adverse effect observed (irritating)[1]
	Oral (Rat) LD50; >4500 mg/kg[1]	
trimethylbenzene (mixed isomers)	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; 8970 mg/kg[2]	Eye (rabbit): 500 mg/24h - mild
		Skin (rabbit): 500 mg/24h-moderate
silica crystalline - quartz	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; 500 mg/kg[2]	Not Available
titanium dioxide	**TOXICITY**	**IRRITATION**
	dermal (hamster) LD50: >=10000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50: >2.28 mg/l4h[1]	Skin (human): 0.3 mg /3D (int)-mild *
	Oral (Rat) LD50; >=2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*	

Rubberized Asphalt Coating & Adhesive

For silica amorphous:
Derived No Adverse Effects Level (NOAEL) in the range of 1000 mg/kg/d.
In humans, synthetic amorphous silica (SAS) is essentially non-toxic by mouth, skin or eyes, and by inhalation. Epidemiology studies show little evidence of adverse health effects due to SAS. Repeated exposure (without personal protection) may cause mechanical irritation of the eye and drying/cracking of the skin.
When experimental animals inhale synthetic amorphous silica (SAS) dust, it dissolves in the lung fluid and is rapidly eliminated. If swallowed, the vast majority of SAS is excreted in the faeces and there is little accumulation in the body. Following absorption across the gut, SAS is eliminated via urine without modification in animals and humans. SAS is not expected to be broken down (metabolised) in mammals.
After ingestion, there is limited accumulation of SAS in body tissues and rapid elimination occurs. Intestinal absorption has not been calculated, but appears to be insignificant in animals and humans. SASs injected subcutaneously are subjected to rapid dissolution and removal. There is no indication of metabolism of SAS in animals or humans based on chemical structure and available data. In contrast to crystalline silica, SAS is soluble in physiological media and the soluble chemical species that are formed are eliminated via the urinary tract without modification.
Both the mammalian and environmental toxicology of SASs are significantly influenced by the physical and chemical properties, particularly those of solubility and particle size. SAS has no acute intrinsic toxicity by inhalation. Adverse effects, including suffocation, that have been reported were caused by the presence of high numbers of respirable particles generated to meet the required test atmosphere. These results are not representative of exposure to commercial SASs and should not be used for human risk assessment. Though repeated exposure of the skin may cause dryness and cracking, SAS is not a skin or eye irritant, and it is not a sensitiser.
Repeated-dose and chronic toxicity studies confirm the absence of toxicity when SAS is swallowed or upon skin contact.
Long-term inhalation of SAS caused some adverse effects in animals (increases in lung inflammation, cell injury and lung collagen content), all of which subsided after exposure.
Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted with SAS in a number of species, at airborne concentrations ranging from 0.5 mg/m3 to 150 mg/m3. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m3. When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m3. The difference in values may be explained by different particle size, and therefore the number of particles administered per unit dose. In general, as particle size decreases so does the NOAEL/LOAEL.
Neither inhalation nor oral administration caused neoplasms (tumours). SAS is not mutagenic in vitro. No genotoxicity was detected in in vivo assays. SAS does not impair development of the foetus. Fertility was not specifically studied, but the reproductive organs in long-term studies were not affected.
For Synthetic Amorphous Silica (SAS)
Repeated dose toxicity
Oral (rat), 2 weeks to 6 months, no significant treatment-related adverse effects at doses of up to 8% silica in the diet.
Inhalation (rat), 13 weeks, Lowest Observed Effect Level (LOEL) =1.3 mg/m3 based on mild reversible effects in the lungs. Inhalation (rat), 90 days, LOEL = 1 mg/m3 based on reversible effects in the lungs and effects in the nasal cavity.
For silane treated synthetic amorphous silica:
Repeated dose toxicity: oral (rat), 28-d, diet, no significant treatment-related adverse effects at the doses tested.
There is no evidence of cancer or other long-term respiratory health effects (for example, silicosis) in workers employed in the manufacture of SAS. Respiratory symptoms in SAS workers have been shown to correlate with smoking but not with SAS exposure, while serial pulmonary function values and chest radiographs are not adversely affected by long-term exposure to SAS.
Data demonstrate that during inhalation exposure,aromatic hydrocarbons undergo substantial partitioning into adipose tissues. Following cessation of exposure, the level of aromatic hydrocarbons in body fats rapidly declines. Thus, the aromatic hydrocarbons are unlikely to bioaccumulate in the body. Selective partitioning of the aromatic hydrocarbons into the non-adipose tissues is unlikely. No data is available regarding distribution following dermal absorption. However, distribution following this route of exposure is likely to resemble the pattern occurring with inhalation exposure.
Aromatics hydrocarbons may undergo several different Phase I dealkylation, hydroxylation and oxidation reactions which may or may not be followed by Phase II conjugation to glycine, sulfation or glucuronidation. However, the major predominant biotransformation pathway is typical of that of the alkylbenzenes and consists of: (1) oxidation of one of the alkyl groups to an alcohol moiety; (2) oxidation of the hydroxyl group to a

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carboxylic acid; (3) the carboxylic acid is then conjugated with glycine to form a hippuric acid. The minor metabolites can be expected to consist of a complex mixture of isomeric triphenols, the sulfate and glucuronide conjugates of dimethylbenzyl alcohols, dimethylbenzoic acids and dimethylhippuric acids. Consistent with the low propensity for bioaccumulation of aromatic hydrocarbons, these substances are likely to be significant inducers of their own metabolism.
The predominant route of excretion of aromatic hydrocarbons following inhalation exposure involves either exhalation of the unmetabolized parent compound, or urinary excretion of its metabolites. When oral administration occurs, there is little exhalation of unmetabolized these hydrocarbons, presumably due to the first pass effect in the liver. Under these circumstances, urinary excretion of metabolites is the dominant route of excretion.
Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length,with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo-paraffins.
The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption,is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.
The production of wood creosote, coal tar creosote, coal tar, coal tar pitch, and coal tar pitch volatiles, stems from the incomplete combustion or pyrolysis of carbon-containing materials. Creosotes, coal tar, coal tar pitch, and coal tar pitch volatiles are composed of many individual compounds of varying physical and chemical characteristics. In addition, the composition of each, although referred to by specific name (e.g., wood creosote or coal tar creosote) is not consistent. Coal tars are by-products of the carbonization of coal to produce coke or natural gas. Physically, they are usually viscous liquids or semisolids that are black or dark brown with a naphthalene-like odor. The coal tars are complex combinations of polycyclic aromatic hydrocarbons (PAHs), phenols, heterocyclic oxygen, sulfur, and nitrogen compounds. By comparison, coal tar creosotes are distillation products of coal tar. They have an oily liquid consistency and range in color from yellowish-dark green to brown. At least 75% of the coal tar creosote mixture is PAHs. Unlike the coal tars and coal tar creosotes, coal tar pitch is a residue produced during the distillation of coal tar. (Beech)wood creosote consists mainly of phenol, cresols, guaiacol, xylenol, and creosol. Creosote bush resin consists of phenolic (e.g., flavonoids and nordihydroguaiaretic acid), neutral (e.g., waxes), basic (e.g., alkaloids), and acidic (e.g., phenolic acids) compounds. The phenolic portion comprises 83-91% of the total resin. Nordihydroguaiaretic acid accounts for 5-10% of the dry weight of the leaves.
It is likely that the toxicity of wood creosote, coal tar creosote, coal tar, coal tar pitch, and coal tar pitch volatiles is due largely to the major individual components, phenols, PAHs and others.

For "distillates of coal tar" or 'creosotes.
Critical Health Effects
The critical health effects for risk characterisation are systemic long-term effects including carcinogenicity, mutagenicity, reproductive toxicity and developmental toxicity. The chemicals are also considered to be phototoxic and have the potential to cause skin irritation and sensitisation and mild respiratory irritation.
Toxicokinetics
Limited data are available. Toxicological data indicate that the chemicals are absorbed via all routes of exposure (WHO, 2004). The PAHs can be absorbed through the respiratory tract, the gastrointestinal tract and the skin. Following absorption, PAHs are widely distributed throughout the body to all internal organs. During metabolism, the parent compounds are converted via intermediate epoxides to phenols, diols, and tetrols, which then conjugate with sulfate or glucuronic acids or with glutathione (IPCS, 1998).
Observation in humans
Evidence of skin, eye and respiratory irritation in humans following exposure to creosote have been reported (ATSDR, 2002).
Skin irritation, eczema and folliculitis were noted when an industrial health survey was conducted of workers exposed to coal tar creosote (ATSDR, 2002). In these workers, the effects of dermal irritation were reported as being exacerbated by exposure to ultraviolet (UV) light.
The phototoxic effects of several PAHs were compared by treating human fibroblasts with these PAHs and then irradiating them with ultraviolet light (<400 nm). A good correlation was found between the phototoxic effects and known carcinogenic potential (IPCS, 1998).
Studies involving workers included reported instances of irritation to superficial ocular tissues after being exposed to coal tar creosote; this was exacerbated after exposure to the sun (ATSDR, 2002).
Skin Sensitisation
Limited data are available. Distillates, coal tar, naphthalene oils (CAS No. 84650-04-4), gave positive results in a single local lymph node assay (LLNA). Creosote (CAS No. 8001-58-9) was found to induce dermal sensitisation when tested according to OECD TG 406 in a guinea pig maximisation test (GPMT) using Dunkin-Hartley guinea pigs (REACH). Overall, the available data support classification for all the chemicals in this group .
An LLNA study (OECD TG 429) was conducted in female BALB/c mice (n = 5/concentration) with coal tar distillates, naphthalene oils (CAS No. 84650-04-4), using a 40 % dimethylacetamide, 30 % acetone and 30 % ethanol (DAE 433) mixture as a vehicle. The test concentrations of 0.3, 3 and 30 % had a simulation index (SI) of 1.36, 1.41 and 5.88 respectively. The positive control, dinitrochlorobenzene at a 0.5 % concentration, gave an SI of 11.55. The three-fold increase in lymphocyte proliferation (EC3 value) could not be calculated (REACHc).
In a GPMT (OECD TG 406) with creosote (CAS No. 8001-58-9), positive skin reactions were reported in 17/19 animals after 24 hours (average Draize score = 1.2) and 6/19 animals after 48 hours (average Draize score = 0.4) (REACHb).
Repeated Dose Toxicity
Oral
Limited data are available regarding the non-cancer effects of the chemicals.
The chemicals in this group are not considered to cause serious damage to health through repeated oral exposure based on the no observed adverse effect levels (NOAELs) (generally >100 mg/kg bw/day) reported for the following 2–4-ring PAHs:
-naphthalene;
-acenaphthene;
-fluorene;
-fluoranthene; and
- pyrene.
Effects on the liver, kidney and blood were observed at higher doses (IPCS, 1998).
Dermal
Limited data are available regarding the non-cancer effects of the chemicals.
Inhalation
Limited data are available regarding the non-cancer effect of the chemicals.
Male Fischer 344 rats were exposed to high-boiling coal liquid (heavy distillate) via inhalation (700 mg/m3) for six hours/day, five days/week for six weeks. A 20 % increase in arterial blood pressure and heart rate was reported, although it was not determined if the response was exposure-related. The growth rate of the rats was reported as suppressed during the time of the study (ATSDR, 2002).
Repeated dose toxicity (inhalation) was determined by exposing 20 (sex/dose) Charles River (CD) rats to CAS No. 90640-86-1 (as distilled coal tar) (5.4, 49 and 106 mg/m3) for six hours/day, five days/week for 13 weeks. A decrease in body weight was recorded as significant in both sexes in the mid- and high-range dose groups during the sixth week of exposure. A treatment related increase in weight was reported in the lung/trachea/body weight ratio and was consistent with macroscopic observation of grey discolouration of the lungs and microscopic observation of macrophages in the lungs. Increases in liver weight (mid-dose group) and liver/body weight ratio (mid- and high-dose group) were recorded in male animals. Increases in the liver weight (high-dose group), liver/body weight ratio and liver/brain weight ratio (mid- and high-dose group) were recorded in the female animals. Reversible hypertrophy of the thyroid follicular cells reported as related to a reduction of colloid was reported at

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all dose levels. A NOAEL of 5.4 mg/m3 was reported for this study (REACHb).
Observation in humans
Mild respiratory effects, including reduced lung function, have been reported in workers using coal tar creosote in wood preservative plants.
Genotoxicity
Several of the chemicals (CAS No. 73665-18-6, CAS No. 84650-03-3 and CAS No. 84650-04-4) are classified as hazardous—Category 2 mutagenic substance—with the risk phrase 'May cause heritable genetic damage (T; R46) in the HSIS (Safe Work Australia). The available data support this classification for all the chemicals in this group, although the associated annotations will differ for each chemical (refer Recommendation section).
For the chemicals CAS No. 84650-03-3 and CAS No. 84650-04-4, in vitro data using the reverse mutation assays with various strains of Salmonella typhimurium were negative for genotoxicity (REACH). No compositional information was available but these chemicals are lower boiling point distillate fractions that are likely to contain aromatics, tar bases and acids (see Grouping rationale). The classification of these chemicals is dependent on benzene concentration (refer to Existing Worker Health and Safety Controls: Hazard Classification section). Benzene is classified as hazardous—Category 2 mutagenic substance—with the risk phrase 'May cause heritable genetic damage (T; R46) in the HSIS (Safe Work Australia).
The chemical, CAS No. 90640-86-1 was positive in a reverse mutation assay in Salmonella typhimurium strains TA98 and TA1537 in the presence of metabolic activation. Weakly positive responses were also observed in strains TA100 and TA102. The sample was reported to contain >50 ppm B[a]P.
Various creosotes have been reported to produce a positive response in vitro. Almost all creosotes tested showed mutagenic activity after metabolic activation (S9 mix) in the conventional Ames assay with S. typhimurium TA98. Positive results were also obtained with several other S. typhimurium TA or YG strains, or with the mouse lymphoma cell assay and the sister chromatid exchange test with Chinese hamster ovary cells. A common feature in the tests with Salmonella strains TA98 and TA100 (plus S9 mix) was that the mutagenicity appeared in the distillation fractions having the highest boiling point ranges (>290 °C) and high concentrations of known mutagenic PAHs (WHO, 2004). A creosote reported to contain <50 ppm B[a]P was tested according to OECD 476 (in vitro mouse lymphoma gene mutation assay). The chemical showed a weak positive mutagenic activity in the presence of metabolic activation. A creosote containing <50 ppm B[a]P did not induce chromosome aberrations in human lymphocytes cultures in the presence and absence of metabolic activation (REACHb).
DNA adduct formation in mammalian systems has been observed following exposure to creosote, with adducts in rats (liver) and mice (lungs, forestomach and spleen) (ATSDR, 2002). A commercially available coal tar creosote was positive in an in vivo mouse micronucleus assay. The CD-1 male mice received two intraperitoneal (i.p.)injections (with an interval of 24 hours) of creosote (in olive oil) at concentrations of 92.5, 185, or 370 mg/kg bw. Dose-dependent increases in the frequency of micronucleated polychromatic erythrocytes in bone marrow were observed. A single intraperitoneal treatment of 370 mg/kg body weight also induced micronuclei (WHO, 2004). A creosote reported to contain <50 ppm B[a]P was reported to be negative in an in vivo mouse micronucleus test (REACHb).
Genotoxicity of PAHs
The chemicals have the potential to contain fluoranthene and chrysene as well as higher molecular weight PAHs that are genotoxic, including benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene (IARC, 2010; IARC, 2012; NICNAS). Positive effects were seen in most assays for the mutagenicity of B[a]P, including induced sperm abnormalities in mice (IPCS, 1998). Data for B[a]P are considered sufficient to indicate that the chemicals could induce mutations in germ cells.
Carcinogenicity
The chemicals are classified as hazardous—Category 2 carcinogenic substances—with the risk phrase 'May cause cancer (T; R45) in the HSIS (Safe Work Australia). The available data support this classification for all the chemicals in this group, although the associated notes will differ for each chemical (refer Recommendation section).
Several creosote or cresosote oils produced skin tumours in mice following dermal application. Lung tumours were also reported in one study. Worker exposure to creosotes has been associated with an increased risk of testicular cancer. The only available cohort study was considered limited by its small size (IARC, 1985; IARC, 2010).
The International Agency for Research on Cancer (IARC) concluded that creosotes are probably carcinogenic to humans (Group 2A). This was based on limited evidence of carcinogenicity in humans and sufficient evidence in experimental animals (IARC, 2010).
There are a number of potential carcinogenic components of the chemicals. There is sufficient evidence in experimental animals for the carcinogenicity of four membered PAHs such as chrysene and pyrene and also several higher molecular weight PAHs (IARC, 2010; IARC 2012).
The classification of a number of chemicals in this group is subject to note M (refer to Existing Worker Health and Safety Controls: Hazard Classification section), which exempts classification if it can be shown that the substance contains <0.005 % w/w B[a]P (50 ppm). No data have been identified regarding the rationale for note M. However, in the absence of detailed composition details, this is considered reasonable as, whilst several carcinogenic PAHs might be present as constituents in these chemicals at levels similar or higher than B[a]P, the cut-off concentration for mixtures containing category 1 carcinogens is 0.1 % (several orders of magnitude higher than 0.005 %).
The classification of some of the lower boiling point distillate fractions are subject to note J (refer to Existing Worker Health and Safety Controls: Hazard Classification section), which exempts classification if it can be shown that the substance contains <0.1% w/w benzene. Benzene is classified as hazardous, a Category 1 carcinogenic substance, with the risk phrase 'May cause cancer (T; R45) in the HSIS (Safe Work Australia).
Reproductive and Developmental Toxicity
Overall, the reproductive and developmental data are limited for chemicals in the group, although the data for higher molecular weight PAHs are considered sufficient for classification for all chemicals except the lower boiling point distillate fractions (CAS Nos. 84650-03-3 and 84650-04-4). The associated notes will differ for each chemical.
In a two-generation study, the chemical, distillates, coal tar, heavy oils (CAS No. 90640-86-1), was administered via oral gavage (25, 75 and 150 mg/kg bw/day) to male and female CD rats (26/sex/dose). At all dose levels, decrease in body weight during the pre-mating period was observed and recorded as dose-related. Decreased fertility and pregnancy indices in the F1 female parental rats were recorded at all dose levels (25, 75, 150 mg/kg bw/d). There was a significant dose-related reduction in the number of live F1 offspring at doses ³75 mg/kg bw/d. A dose-related decrease in growth of the F1 offspring was reported, starting at 25 mg/kg bw/d. Although the NOAEL is reported as 25 mg/kg bw/d (REACHb), reproductive effects were indicated at all doses.
In a developmental toxicity study, the chemical, distillates, coal tar, heavy oils (CAS No. 90640-86-1), was administered via oral gavage (25, 50 and 175 mg/kg bw/day) to 30 (per dose) mated female CD rats, during gestation day(GD) 6–15. Increases in post implantation loss, resorptions and a reduction in live foetuses were observed in 175 mg/kg bw/day group. Developmental toxicity was not observed at doses of 50 mg/kg bw/day or lower. Malformations were observed in all dose groups, although the incidences were significantly higher in the mid- and high-dose groups. These were historically common malformations and not considered by the study authors to be treatment related. There were no adverse effects observed for late intrauterine development of live foetuses in any dose group. The NOAEL for maternal toxicity was reported as 50 mg/kg bw/d and for teratogenicity 175 mg/kg bw/d (REACHb).
Coal tar creosote was tested for oestrogenic activity using an assay in ovariectomised (OVX) ICR and DBA/2 mice. The animals received oral doses (by gavage) once every 24 hours for four days and were euthanised on day five. No increase in absolute or relative uterine wet weight or vaginal cornification was observed.
A decrease in mean foetal body weight was observed in the offspring of female ICR mice dosed by gavage with 400 mg/kg petroleum creosote in DMSO on GD 5–9. Moderate maternal toxicity in the form of reduced body weight gain was observed for both creosote-treated and vehicle-control mice compared with untreated controls. (ATSDR, 2002; WHO, 2004).
Embryotoxicity of petroleum creosote has been studied in a mouse preimplantation embryo culture system. The ICR mice embryos (n = 15) collected on day 3.5 of gestation (blastocyst stage) were exposed for 1 hour to different concentrations of creosote in a serum-supplemented culture medium with and without rodent hepatic S9 microsomal fractions, and subsequently cultured in a control medium for 24–72 hours. Embryonic viability was inversely related to petroleum creosote concentration (WHO, 2004).
An experiment with pregnant pigs, held on wooden platforms treated with coal tar creosote, resulted in adverse developmental effects. A significant number (24/41) of piglets died at birth and 11 piglets died by day three post farrowing.
The chemicals may contain several higher molecular weight PAHs that are embryotoxic. B[a]P also had adverse effects on female fertility, reproduction and postnatal development (IPCS, 1998).
The chemicals are recommended for classification and labelling under the current approved criteria and adopted GHS as below. This assessment

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	does not consider classification of physical and environmental hazards. The classification criteria for mixtures should be applied to known components based on their concentrations in these UVCB substances. In the absence of detailed composition data the following notes should be applied. Information on notes A note should be added for the acute toxicity classification. The acute toxicity R23 classification need not apply if it can be shown that the chemical contains <8 % pyrene; however, R20 classification applies if the chemicals contains >1 % pyrene. The current HSIS classification for carcinogenicity of the chemicals indicated Note H. Note H is no longer considered relevant for these chemicals as the acute, systemic and local effects of the chemicals have been evaluated. The classification for CAS Nos. 61789-28-4, 65996-91-0, 65996-92-1, 68188-48-7, 73665-18-6, 84650-04-4 and 91995-51-6 are subject to Note M (refer to Existing Worker Health and Safety Controls: Hazard Classification section), which exempts classification if it can be shown that the substance contains <0.005 % w/w B[a]P (50 ppm). Given that Note M for carcinogenicity is considered appropriate for these chemicals and the cut-off concentration for mixtures is similar for the mutagenicity, reproductive/developmental and carcinogenicity classifications, a similar note for the proposed genotoxicity and reproductive/developmental classification is considered appropriate. Therefore, Note M should be slightly modified as follows: 'Note M: The classification (with the exception of classification for acute toxicity and sensitisation) need not apply if it can be shown that the substance contains less than 0.005% w/w benzo[a]pyrene (EINECS no. 200-028-5). This note only applies to certain complex coal-derived substances in Annex I.' The classification for CAS Nos. 84650-03-3, 84650-04-4 and 73665-18-6 are subject to Note J (refer to Existing Worker Health and Safety Controls: Hazard Classification section), which exempts classification if it can be shown that the substance contains <0.1% w/w benzene. These chemicals are described as including lower boiling point distillation fractions and therefore Note J is considered appropriate. Based on the description of CAS No. 65996-92-1 ('The distillate from coal tar having an approximate distillation range of 100 deg C to 450 deg C (212 deg F to 842 deg F). Composed primarily of two to four membered condensed ring aromatic hydrocarbons, phenolic compounds, and aromatic nitrogen bases.' (NCI)). Note J is also considered applicable to this chemical. The classification for CAS Nos. 8001-58-9 and 90640-86-1 are not subject to any notes. The lack of a note may be because the chemicals under these CAS Nos. might not be available in sufficiently purified forms. In the absence of further information, the addition of note M is not recommended. NICNAS HUMAN HEALTH TIER II ASSESSMENT FOR Coal Tar Distillates http://www.nicnas.gov.au/chemical-information/imap-assessments/imap-group-assessment-report?assessment_id=1442
WHITE SPIRIT	white spirit, as CAS RN 8052-41-3 For petroleum: This product contains benzene, which can cause acute myeloid leukaemia, and n-hexane, which can be metabolized to compounds which are toxic to the nervous system. This product contains toluene, and animal studies suggest high concentrations of toluene lead to hearing loss. This product contains ethyl benzene and naphthalene, from which animal testing shows evidence of tumour formation. Cancer-causing potential: Animal testing shows inhaling petroleum causes tumours of the liver and kidney; these are however not considered to be relevant in humans. Mutation-causing potential: Most studies involving gasoline have returned negative results regarding the potential to cause mutations, including all recent studies in living human subjects (such as in petrol service station attendants). Reproductive toxicity: Animal studies show that high concentrations of toluene (>0.1%) can cause developmental effects such as lower birth weight and developmental toxicity to the nervous system of the foetus. Other studies show no adverse effects on the foetus. Human effects: Prolonged or repeated contact may cause defatting of the skin which can lead to skin inflammation and may make the skin more susceptible to irritation and penetration by other materials. Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable.
1,2,4-TRIMETHYL BENZENE	CHEMWATCH 2325 1,3,5-trimethylbenzene
1,3,5-TRIMETHYL BENZENE	CHEMWATCH 12171 1,2,4-trimethylbenzene
NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT	For C9 aromatics (typically trimethylbenzenes - TMBs) Acute Toxicity Acute toxicity studies (oral, dermal and inhalation routes of exposure) have been conducted in rats using various solvent products containing predominantly mixed C9 aromatic hydrocarbons (CAS RN 64742-95-6). Inhalation LC50 s range from 6,000 to 10,000 mg/m 3 for C9 aromatic naphtha and 18,000 to 24,000 mg/m3 for 1,2,4 and 1,3,5-TMB, respectively. A rat oral LD50 reported for 1,2,4-TMB is 5 grams/kg bw and a rat dermal LD50 for the C9 aromatic naphtha is >4 ml/kg bw. These data indicate that C9 aromatic solvents show that LD50/LC50 values are greater than limit doses for acute toxicity studies established under OECD test guidelines. Irritation and Sensitization Several irritation studies, including skin, eye, and lung/respiratory system, have been conducted on members of the category. The results indicate that C9 aromatic hydrocarbon solvents are mildly to moderately irritating to the skin, minimally irritating to the eye, and have the potential to irritate the respiratory tract and cause depression of respiratory rates in mice. Respiratory irritation is a key endpoint in the current occupational exposure limits established for C9 aromatic hydrocarbon solvents and trimethylbenzenes. No evidence of skin sensitization was identified. Repeated Dose Toxicity Inhalation: The results from a subchronic (3 month) neurotoxicity study and a one-year chronic study (6 hr/day, 5 days/week) indicate that effects from inhalation exposure to C9 Aromatic Hydrocarbon Solvents on systemic toxicity are slight. A battery of neurotoxicity and neurobehavioral endpoints were evaluated in the 3-month inhalation study on C9 aromatic naphtha tested at concentrations of 0, 101, 452, or 1320 ppm (0, 500, 2,220, or 6,500 mg/m3). In this study, other than a transient weight reduction in the high exposure group (not statistically significant at termination of exposures), no effects were reported on neuropathology or neuro/behavioral parameters. The NOAEL for systemic and/or neurotoxicity was 6,500 mg/m3, the highest concentration tested. In an inhalation study of a commercial blend, rats were exposed to C9 aromatic naphtha concentrations of 0, 96, 198, or 373 ppm (0, 470, 970, 1830 mg/m3) for 6 hr/day, 5 days/week, for 12 months. Liver and kidney weights were increased in the high exposure group but no accompanying histopathology was observed in these organs. The NOAEL was considered to be the high exposure level of 373 ppm, or 1830 mg/m3. In two subchronic rat inhalation studies, both of three months duration, rats were exposed to the individual TMB isomers (1,2,4-and 1,3,5-) to nominal concentrations of 0, 25, 100, or 250 ppm (0, 123, 492, or 1230 mg/m3). Respiratory irritation was observed at 492 (100 ppm) and 1230 mg/m3 (250 ppm) and no systemic toxicity was observed in either study. For both pure isomers, the NOELs are 25 ppm or 123 mg/m3 for respiratory irritation and 250 ppm or 1230 mg/m3 for systemic effects. Oral: The C9 aromatic naphtha has not been tested via the oral route of exposure. Individual TMB isomers have been evaluated in a series of repeated-dose oral studies ranging from 14 days to 3 months over a wide range of doses. The effects observed in these studies included increased liver and kidney weights, changes in blood chemistry, increased salivation, and decreased weight gain at higher doses. Organ weight changes appeared to be adaptive as they were not accompanied by histopathological effects. Blood changes appeared sporadic and without pattern. One study reported hyaline droplet nephropathy in male rats at the highest dose (1000 mg/kg bw-day), an effect that is often associated with alpha-2mu-globulin-induced nephropathy and not considered relevant to humans. The doses at which effects were detected were 100 mg/kg-bw day or above (an exception was the pilot 14 day oral study - LOAEL 150 mg/kg bw-day - but the follow up three month study had a LOAEL of 600 mg/kg/bw-day with a NOAEL of 200 mg/kg bw-day). Since effects generally were not severe and could be considered adaptive or spurious, oral exposure does not appear to pose a high toxicity hazard for pure trimethylbenzene isomers. Mutagenicity In vitro genotoxicity testing of a variety of C9 aromatics has been conducted in both bacterial and mammalian cells. In vitro point mutation tests were conducted with Salmonella typhimurium and Escherichia coli bacterial strains, as well as with cultured mammalian cells such as the Chinese hamster cell ovary cells (HGPRT assay) with and without metabolic activation. In addition, several types of in vitro chromosomal aberration tests have been performed (chromosome aberration frequency in Chinese hamster ovary and lung cells, sister chromatid exchange in CHO cells). Results were negative both with and without metabolic activation for all category members. For the supporting chemical 1,2,3-TMB, a single in vitro chromosome aberration test was weakly positive. In in vivo bone marrow cytogenetics test, rats were exposed to C9 aromatic naphtha at concentrations of 0, 153, 471, or 1540 ppm (0, 750, 2,310, or 7,560 mg/m3) 6 hr/day, for 5 days. No evidence of in vivo somatic cell

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	genotoxicity was detected. Based on the cumulative results of these assays, genetic toxicity is unlikely for substances in the C9 Aromatic Hydrocarbon Solvents Category Reproductive and Developmental Toxicity Results from the three-generation reproduction inhalation study in rats indicate limited effects from C9 aromatic naphtha. In each of three generations (F0, F1 and F2), rats were exposed to High Flash Aromatic Naphtha (CAS RN 64742-95-6) via whole body inhalation at target concentrations of 0, 100, 500, or 1500 ppm (actual mean concentrations throughout the full study period were 0, 103, 495, or 1480 ppm, equivalent to 0, 505, 2430, or 7265 mg/m3 , respectively). In each generation, both sexes were exposed for 10 weeks prior to and two weeks during mating for 6 hrs/day, 5 days/wks. Female rats in the F0, F1, and F2 generation were then exposed during gestation days 0-20 and lactation days 5-21 for 6 hrs/day, 7 days/wk. The age at exposure initiation differed among generations; F0 rats were exposed starting at 9 weeks of age, F1 exposure began at 5-7 weeks, and F2 exposure began at postnatal day (PND) 22. In the F0 and F1 parental generations, 30 rats/sex /group were exposed and mated. However, in the F2 generation, 40/sex/group were initially exposed due to concerns for toxicity, and 30/sex /group were randomly selected for mating, except that all survivors were used at 1480 ppm. F3 litters were not exposed directly and were sacrificed on lactation day 21. Systemic Effects on Parental Generations: The F0 males showed statistically and biologically significantly decreased mean body weight by ~15% at 1480 ppm when compared with controls. Seven females died or were sacrificed in extremis at 1480 ppm. The F0 female rats in the 495 ppm exposed group had a 13% decrease in body weight gain when adjusted for initial body weight when compared to controls. The F1 parents at 1480 ppm had statistically significantly decreased mean body weights (by ~13% (females) and 22% (males)), and locomotor activity. F1 parents at 1480 ppm had increased ataxia and mortality (six females). Most F2 parents (70/80) exposed to 1480 ppm died within the first week. The remaining animals survived throughout the rest of the exposure period. At week 4 and continuing through the study, F2 parents at 1480 ppm had statistically significant mean body weights much lower than controls (~33% for males; ~28% for females); body weights at 495 ppm were also reduced significantly (by 13% in males and 15% in females). The male rats in the 495 ppm exposed group had a 12% decrease in body weight gain when adjusted for initial body weight when compared to controls. Based on reduced body weight observed, the overall systemic toxicity LOAEC is 495 ppm (2430 mg/m3). Reproductive Toxicity-Effects on Parental Generations: There were no pathological changes noted in the reproductive organs of any animal of the F0, F1, or F2 generation. No effects were reported on sperm morphology, gestational period, number of implantation sites, or post-implantation loss in any generation. Also, there were no statistically or biologically significant differences in any of the reproductive parameters, including: number of mated females, copulatory index, copulatory interval, number of females delivering a litter, number of females delivering a live litter, or male fertility in the F0 or in the F2 generation. Male fertility was statistically significantly reduced at 1480 ppm in the F1 rats. However, male fertility was not affected in the F0 or in the F2 generations; therefore, the biological significance of this change is unknown and may or may not be attributed to the test substance. No reproductive effects were observed in the F0 or F1 dams exposed to 1480 ppm (7265 mg/m3). Due to excessive mortality at the highest concentration (1480 ppm, only six dams available) in the F2 generation,, a complete evaluation is precluded. However, no clear signs of reproductive toxicity were observed in the F2 generation. Therefore, the reproductive NOAEC is considered 495 ppm (2430 mg/m3), which excludes analysis of the highest concentration due to excessive mortality. Developmental Toxicity - Effects on Pups: Because of significant maternal toxicity (including mortality) in dams in all generations at the highest concentration (1480 ppm), effects in offspring at 1480 ppm are not reported here. No significant effects were observed in the F1 and F2 generation offspring at 103 or 495 ppm. However, in F3 offspring, body weights and body weight gain were reduced by ~ 10-11% compared with controls at 495 ppm for approximately a week (PND 14 through 21). Maternal body weight was also depressed by ~ 12% throughout the gestational period compared with controls. The overall developmental LOAEC from this study is 495 ppm (2430 mg/m3) based on the body weights reductions observed in the F3 offspring. Conclusion: No effects on reproductive parameters were observed at any exposure concentration, although a confident assessment of the group exposed at the highest concentration was not possible. A potential developmental effect (reduction in mean pup weight and weight gain) was observed at a concentration that was also associated with maternal toxicity. * [Devoe] .
TRIMETHYLBENZENE (MIXED ISOMERS)	NOTE: This data is for mixed isomers of unstated proportions.
SILICA CRYSTALLINE - QUARTZ	**WARNING:** For inhalation exposure ONLY: This substance has been classified by the IARC as Group 1: **CARCINOGENIC TO HUMANS** The International Agency for Research on Cancer (IARC) has classified occupational exposures to **respirable** (<5 um) crystalline silica as being carcinogenic to humans . This classification is based on what IARC considered sufficient evidence from epidemiological studies of humans for the carcinogenicity of inhaled silica in the forms of quartz and cristobalite. Crystalline silica is also known to cause silicosis, a non-cancerous lung disease. Intermittent exposure produces; focal fibrosis, (pneumoconiosis), cough, dyspnoea, liver tumours. * Millions of particles per cubic foot (based on impinger samples counted by light field techniques). NOTE : the physical nature of quartz in the product determines whether it is likely to present a chronic health problem. To be a hazard the material must enter the breathing zone as respirable particles.
TITANIUM DIOXIDE	* IUCLID Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies. The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
Rubberized Asphalt Coating & Adhesive & BITUMEN (PETROLEUM) & 1,2,4-TRIMETHYL BENZENE & 1,3,5-TRIMETHYL BENZENE & GILSONITE & NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT & TRIMETHYLBENZENE (MIXED ISOMERS) & TITANIUM DIOXIDE	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
Rubberized Asphalt Coating & Adhesive & GILSONITE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.

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Rubberized Asphalt Coating & Adhesive & TITANIUM DIOXIDE	For titanium dioxide: Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin. Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica. No data were available on genotoxic effects in titanium dioxide-exposed humans. Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts. Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium. Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light. **Animal carcinogenicity data** Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats. In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative. Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice. In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative.
Rubberized Asphalt Coating & Adhesive & 1,2,4-TRIMETHYL BENZENE & 1,3,5-TRIMETHYL BENZENE & NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT & TRIMETHYLBENZENE (MIXED ISOMERS)	For trimethylbenzenes: Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure. Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal. Following oral administration of the chemical to rats, 62.6% of the dose was recovered as urinary metabolites indicating substantial absorption . 1,2,4-Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. In the blood stream, approximately 85% of the chemical is bound to red blood cells Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion . After a single oral dose to rats of 1200 mg/kg, urinary metabolites consisted of approximately 43.2% glycine, 6.6% glucuronic, and 12.9% sulfuric acid conjugates . The two principle metabolites excreted by rabbits after oral administration of 438 mg/kg/day for 5 days were 2,4-dimethylbenzoic acid and 3,4-dimethylhippuric acid . The major routes of excretion of 1,2,4-trimethyl- benzene are exhalation of parent compound and elimination of urinary metabolites. Half-times for urinary metabolites were reported as 9.5 hours for glycine, 22.9 hours for glucuronide, and 37.6 hours for sulfuric acid conjugates. **Acute Toxicity** Direct contact with liquid 1,2,4-trimethylbenzene is irritating to the skin and breathing the vapor is irritating to the respiratory tract causing pneumonitis. Breathing high concentrations of the chemical vapor causes headache, fatigue, and drowsiness. In humans liquid 1,2,4-trimethylbenzene is irritating to the skin and inhalation of vapor causes chemical pneumonitis . High concentrations of vapor (5000-9000 ppm) cause headache, fatigue, and drowsiness . The concentration of 5000 ppm is roughly equivalent to a total of 221 mg/kg assuming a 30 minute exposure period (see end note 1). 2. Animals - Mice exposed to 8130-9140 ppm 1,2,4-trimethylbenzene (no duration given) had loss of righting response and loss of reflexes Direct dermal contact with the chemical (no species given) causes vasodilation, erythema, and irritation (U.S. EPA). Seven of 10 rats died after an oral dose of 2.5 mL of a mixture of trimethylbenzenes in olive oil (average dose approximately 4.4 g/kg) . Rats and mice were exposed by inhalation to a coal tar distillate containing about 70% 1,3,5- and 1,2,4-trimethylbenzene; no pathological changes were noted in either species after exposure to 1800-2000 ppm for up to 48 continuous hours, or in rats after 14 exposures of 8 hours each at the same exposure levels . No effects were reported for rats exposed to a mixture of trimethyl- benzenes at 1700 ppm for 10 to 21 days **Neurotoxicity** 1,2,4-Trimethylbenzene depresses the central nervous system. Exposure to solvent mixtures containing the chemical causes headache, fatigue, nervousness, and drowsiness. Occupationally, workers exposed to a solvent containing 50% 1,2,4-trimethylbenzene had nervousness, headaches, drowsiness, and vertigo (U.S. EPA). Headache, fatigue, and drowsiness were reported for workers exposed (no dose given) to paint thinner containing 80% 1,2,4- and 1,3,5-trimethylbenzenes Results of the developmental toxicity study indicate that the C9 fraction caused adverse neurological effects at the highest dose (1500 ppm) tested. **Subchronic/Chronic Toxicity** Long-term exposure to solvents containing 1,2,4-trimethylbenzene may cause nervousness, tension, and bronchitis. Painters that worked for several years with a solvent containing 50% 1,2,4- and 30% 1,3,5-trimethylbenzene showed nervousness, tension and anxiety, asthmatic bronchitis, anemia, and alterations in blood clotting; haematological effects may have been due to trace amounts of benzene Rats given 1,2,4-trimethylbenzene orally at doses of 0.5 or 2.0 g/kg/day, 5 days/week for 4 weeks. All rats exposed to the high dose died and 1 rat in the low dose died (no times given); no other effects were reported. Rats exposed by inhalation to 1700 ppm of a trimethylbenzene isomeric mixture for 4 months had decreased weight gain, lymphopenia and neutrophilia . **Genotoxicity:** Results of mutagenicity testing, indicate that the C9 fraction does not induce gene mutations in prokaryotes (Salmonella tymphimurium/mammalian microsome assay); or in mammalian cells in culture (in Chinese hamster ovary cells with and without activation). The C9 fraction does not does not induce chromosome mutations in Chinese hamster ovary cells with and without activation; does not induce chromosome aberrations in the bone marrow of Sprague-Dawley rats exposed by inhalation (6 hours/day for 5 days); and does not induce sister chromatid exchange in Chinese hamster ovary cells with and without activation. **Developmental/Reproductive Toxicity:** A three-generation reproductive study on the C9 fraction was conducted CD rats (30/sex/group) were exposed by inhalation to the C9 fraction at concentrations of 0, 100, 500, or 1500 ppm (0, 100, 500, or 1500 mg/kg/day) for 6 hours/day, 5 days/week. There was evidence of parental and reproductive toxicity at all dose levels. Indicators of parental toxicity included reduced body

Continued...

	weights, increased salivation, hunched posture, aggressive behavior, and death. Indicators of adverse reproductive system effects included reduced litter size and reduced pup body weight. The LOEL was 100 ppm; a no-observed-effect level was not established Developmental toxicity, including possible develop- mental neurotoxicity, was evident in rats in a 3-generation reproductive study No effects on fecundity or fertility occurred in rats treated dermally with up to 0.3 mL/rat/day of a mixture of trimethyl- benzenes, 4-6 hours/day, 5 days/week over one generation
BITUMEN (PETROLEUM) & GILSONITE & TITANIUM DIOXIDE	No significant acute toxicological data identified in literature search.
BITUMEN (PETROLEUM) & TITANIUM DIOXIDE	**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
1,2,4-TRIMETHYL BENZENE & 1,3,5-TRIMETHYL BENZENE	Other Toxicity data is available for CHEMWATCH 12172 1,2,3-trimethylbenzene
1,3,5-TRIMETHYL BENZENE & TRIMETHYLBENZENE (MIXED ISOMERS)	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

Acute Toxicity	✗	**Carcinogenicity**	✓
Skin Irritation/Corrosion	✓	**Reproductivity**	✗
Serious Eye Damage/Irritation	✓	**STOT - Single Exposure**	✓
Respiratory or Skin sensitisation	✓	**STOT - Repeated Exposure**	✓
Mutagenicity	✓	**Aspiration Hazard**	✓

Legend: ✗ *– Data either not available or does not fill the criteria for classification*
✓ *– Data available to make classification*

SECTION 12 Ecological information

Toxicity

Rubberized Asphalt Coating & Adhesive

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

bitumen (petroleum)

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

white spirit

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	720h	Crustacea	0.024mg/l	2
LC50	96h	Fish	0.14mg/l	2
EC50	96h	Algae or other aquatic plants	0.277mg/l	2

1,2,4-trimethyl benzene

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1344h	Fish	31-207	7
EC50(ECx)	96h	Algae or other aquatic plants	2.356mg/l	2
EC50	48h	Crustacea	ca.6.14mg/l	1
LC50	96h	Fish	3.41mg/l	2
EC50	96h	Algae or other aquatic plants	2.356mg/l	2

1,3,5-trimethyl benzene

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1680h	Fish	23-342	7
EC50	48h	Crustacea	13mg/L	5
NOEC(ECx)	384h	Crustacea	0.257mg/l	2
LC50	96h	Fish	5.216mg/l	2
EC50	96h	Algae or other aquatic plants	3.084mg/l	2

gilsonite

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

naphtha petroleum, light aromatic solvent

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	96h	Algae or other aquatic plants	64mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	1mg/l	1
EC50	72h	Algae or other aquatic plants	19mg/l	1
EC50	48h	Crustacea	6.14mg/l	1

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trimethylbenzene (mixed isomers)	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
silica crystalline - quartz	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available

titanium dioxide	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	BCF	1008h	Fish	<1.1-9.6	7
	EC50	72h	Algae or other aquatic plants	3.75-7.58mg/l	4
	EC50	48h	Crustacea	1.9mg/l	2
	NOEC(ECx)	504h	Crustacea	0.02mg/l	4
	LC50	96h	Fish	1.85-3.06mg/l	4
	EC50	96h	Algae or other aquatic plants	179.05mg/l	2

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
For 1,2,4 - Trimethylbenzene:
Half-life (hr) air: 0.48-16;
Half-life (hr) H2O surface water: 0.24 -672;
Half-life (hr) H2O ground: 336-1344;
Half-life (hr) soil: 168-672;
Henry's Pa m3 /mol: 385 -627;
Bioaccumulation: not significant. 1,2,4-Trimethylbenzene is a volatile organic compound (VOC) substance.
Atmospheric Fate: 1,2,4-trimethylbenzene can contribute to the formation of photochemical smog in the presence of other VOCs. Degradation of 1,2,4-trimethylbenzene in the atmosphere occurs by reaction with hydroxyl radicals. Reaction also occurs with ozone but very slowly (half life 8820 days).
Aquatic Fate: 1,2,4-Trimethylbenzene volatilizes rapidly from surface waters with volatilization half-life from a model river calculated to be 3.4 hours. Biodegradation of 1,2,4-trimethylbenzene has been noted in both seawater and ground water. Various strains of Pseudomonas can biodegrade 1,2,4-trimethylbenzene.
Terrestrial Fate: 1,2,4-Trimethylbenzene also volatilizes from soils however; moderate adsorption to soils and sediments may occur. Volatilization is the major route of removal of 1,2,4-trimethylbenzene from soils; although, biodegradation may also occur. Due to the high volatility of the chemical it is unlikely to accumulate in soil or surface water to toxic concentrations.
Ecotoxicity: No significant bioaccumulation has been noted. 1,2,4-Trimethylbenzene is moderately toxic to fathead minnow and slightly toxic to dungeness crab. 1,2,4-Trimethylbenzene has moderate acute toxicity to aquatic organisms. No stress was observed in rainbow trout, sea lamprey and Daphnia magna water fleas. The high concentrations required to induce toxicity in laboratory animals are not likely to be reached in the environment.
For Aromatic Substances Series:
Environmental Fate: Large, molecularly complex polycyclic aromatic hydrocarbons, or PAHs, are persistent in the environment longer than smaller PAHs.
Atmospheric Fate: PAHs are 'semi-volatile substances" which can move between the atmosphere and the Earth's surface in repeated, temperature-driven cycles of deposition and volatilization. Terrestrial Fate: BTEX compounds have the potential to move through soil and contaminate ground water, and their vapors are highly flammable and explosive.
Ecotoxicity - Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. The order of most toxic to least in a study using grass shrimp and brown shrimp was dimethylnaphthalenes > methylnaphthalenes >naphthalenes. Anthrcene is a phototoxic PAH. UV light greatly increases the toxicity of anthracene to bluegill sunfish. Biological resources in strong sunlight are at more risk than those that are not. PAHs in general are more frequently associated with chronic risks.
For petroleum distillates:
Environmental fate:
When petroleum substances are released into the environment, four major fate processes will take place: dissolution in water, volatilization, biodegradation and adsorption. These processes will cause changes in the composition of these UVCB substances. In the case of spills on land or water surfaces, photodegradation-another fate process-can also be significant.
As noted previously, the solubility and vapour pressure of components within a mixture will differ from those of the component alone. These interactions are complex for complex UVCBs such as petroleum hydrocarbons.
Each of the fate processes affects hydrocarbon families differently. Aromatics tend to be more water-soluble than aliphatics of the same carbon number, whereas aliphatics tend to be more volatile. Thus,when a petroleum mixture is released into the environment, the principal water contaminants are likely to be aromatics, whereas aliphatics will be the principal air contaminants . The trend in volatility by component class is as follows: alkenes = alkanes > aromatics = cycloalkanes.
The most soluble and volatile components have the lowest molecular weight; thus there is a general shift to higher molecular weight components in residual materials.
Biodegradation:
Biodegradation is almost always operative when petroleum mixtures are released into the environment. It has been widely demonstrated that nearly all soils and sediments have populations of bacteria and other organisms capable of degrading petroleum hydrocarbons Degradation occurs both in the presence and absence of oxygen. Two key factors that determine degradation rates are oxygen supply and molecular structure. In general, degradation is more rapid under aerobic conditions. Decreasing trends in degradation rates according to structure are as follows:
(1) n-alkanes, especially in the C10–C25 range, which are degraded readily;
(2) isoalkanes;
(3) alkenes;
(4) benzene, toluene, ethylbenzene, xylenes (BTEX) (when present in concentrations that are not toxic to microorganisms);
(5) monoaromatics;
(6) polynuclear (polycyclic) aromatic hydrocarbons (PAHs); and
(7) higher molecular weight cycloalkanes (which may degrade very slowly.
Three weathering processes-dissolution in water, volatilization and biodegradation-typically result in the depletion of the more readily soluble,volatile and degradable compounds and the accumulation of those most resistant to these processes in residues.
When large quantities of a hydrocarbon mixture enter the soil compartment, soil organic matter and other sorption sites in soil are fully saturated and the hydrocarbons will begin to form a separate phase (a non-aqueous phase liquid, or NAPL) in the soil. At concentrations below the retention capacity for the hydrocarbon in the soil, the NAPL will be immobile this is referred to as residual NAPL . Above the retention capacity, the NAPL becomes mobile and will move within the soil
Bioaccumulation:
Bioaccumulation potential was characterized based on empirical and/or modelled data for a suite of petroleum hydrocarbons expected to occur in petroleum substances.
Bioaccumulation factors (BAFs) are the preferred metric for assessing the bioaccumulation potential of substances, as the bioconcentration factor (BCF) may not adequately account for the bioaccumulation potential of substances via the diet, which predominates for substances with log Kow > ~4.5
In addition to fish BCF and BAF data, bioaccumulation data for aquatic invertebrate species were also considered. Biota-sediment/soil accumulation factors (BSAFs), trophic magnification factors and biomagnification factors were also considered in characterizing bioaccumulation potential.
Overall, there is consistent empirical and predicted evidence to suggest that the following components have the potential for high bioaccumulation, with BAF/BCF values greater than

Continued...

5000: C13–C15 isoalkanes, C12 alkenes, C12–C15 one-ring cycloalkanes, C12 and C15 two-ring cycloalkanes, C14 polycycloalkanes, C15 one-ring aromatics, C15 and C20 cycloalkane monoaromatics, C12–C13 diaromatics, C20 cycloalkane diaromatics, and C14 and C20 three-ring PAHs
These components are associated with a slow rate of metabolism and are highly lipophilic. Exposures from water and diet, when combined, suggest that the rate of uptake would exceed that of the total elimination rate. Most of these components are not expected to biomagnify in aquatic or terrestrial foodwebs, largely because a combination of metabolism, low dietary assimilation efficiency and growth dilution allows the elimination rate to exceed the uptake rate from the diet; however,
one study suggests that some alkyl-PAHs may biomagnify.While only BSAFs were found for some PAHs, it is possible that BSAFs will be > 1 for invertebrates, given that they do not have the same metabolic competency as fish.
In general, fish can efficiently metabolize aromatic compounds. There is some evidence that alkylation increases bioaccumulation of naphthalene but it is not known if this can be generalized to larger PAHs or if any potential increase in bioaccumulation due to alkylation will be sufficient to exceed a BAF/BCF of 5000.
Some lower trophic level organisms (i.e., invertebrates) appear to lack the capacity to efficiently metabolize aromatic compounds, resulting in high bioaccumulation potential for some aromatic components as compared to fish.
This is the case for the C14 three-ring PAH, which was bioconcentrated to a high level (BCF > 5000) by invertebrates but not by fish. There is potential for such bioaccumulative components to reach toxic levels in organisms if exposure is continuous and of sufficient magnitude, though this is unlikely in the water column following a spill scenario due to relatively rapid dispersal
Bioaccumulation of aromatic compounds might be lower in natural environments than what is observed in the laboratory. PAHs may sorb to organic material suspended in the water column (dissolved humic material), which decreases their overall bioavailability primarily due to an increase in size. This has been observed with fish
Ecotoxicity:
Diesel fuel studies in salt water are available. The values varied greatly for aquatic species such as rainbow trout and Daphnia magna, demonstrating the inherent variability of diesel fuel compositions and its effects on toxicity. Most experimental acute toxicity values are above 1 mg/L. The lowest 48-hour LC50 for salmonids was 2.4 mg/L. Daphnia magna had a 24-hour LC50 of 1.8 mg/.The values varied greatly for aquatic species such as rainbow trout and Daphnia magna, demonstrating the inherent variability of diesel fuel compositions and its effects on toxicity. Most experimental acute toxicity values are above 1 mg/L. The lowest 48-hour LC50 for salmonids was 2.4 mg/L. Daphnia magna had a 24-hour LC50 of 1.8 mg/L
The tropical mysid Metamysidopsis insularis was shown to be very sensitive to diesel fuel, with a 96-hour LC50 value of 0.22 mg/L this species has been shown to be as sensitive as temperate mysids to toxicants. However,However this study used nominal concentrations,and therefore was not considered acceptable. In another study involving diesel fuel, the effect on brown or common shrimp (Crangon crangon) a 96-hour LC50 of 22 mg/L was determined. A “gas oil”was also tested and a 96-hour LC50 of 12 mg/L.was determined
The steady state cell density of marine phytoplankton decreased with increasing concentrations of diesel fuel, with different sensitivities between species . The diatom Phaeodactylum tricornutum showed a 20% decrease in cell density in 24 hours following a 3 mg/L exposure with a 24-hour no-observed effect concentration (NOEC) of 2.5 mg/L. The microalga Isochrysis galbana was more tolerant to diesel fuel, with a 24-hour lowest-observed-effect concentration (LOEC) of 26 mg/L (14% decrease in cell density), and a NOEC of 25 mg/L. Finally, the green algae Chlorella salina was relatively insensitive to diesel fuel contamination, with a 24-hour LOEC of 170 mg/L (27% decrease in cell density), and a NOEC of 160 mg/L . All populations of phytoplankton returned to a steady state within 5 days of exposure
In sandy soils, earthworm (Eisenia fetida) mortality only occurred at diesel fuel concentrations greater than 10 000 mg/kg, which was also the concentration at which sub-lethal weight loss was recorded
Nephrotoxic effects of diesel fuel have been documented in several animal and human studies.Some species of birds (mallard ducks in particular) are generally resistant to the toxic effects of petrochemical ingestion, and large amounts of petrochemicals are needed in order to cause direct mortality

Sulfide ion is very toxic to aquatic life, threshold concentration for fresh or saltwater fish is 0.5ppm. The product therefore is very toxic to aquatic life. The major decomposition product, hydrogen sulfide, is damaging to vegetation at 5ppm for 24 hours
for bitumens/ asphalts:
This family of hydrocarbon is expected to have similar boiling points, vapor pressures, log Kow values (>10), and water solubilities. Limited environmental fate data also support the grouping of bitumens/ asphalts under one category. Bitumen/ asphalts contain complex hydrocarbon mixtures with molecular weights ranging from 500-2000 and carbon numbers predominantly higher than C25, vapor pressures are negligible. The high molecular weights and similar hydrocarbon distributions among the bitumens/ asphalts support the conclusion that the toxicity of this group, in general, is not expected to vary significantly across members.
Environmental fate:
Upon release to the environment, bitumens/ asphalts are expected to distribute similarly because of their low volatility and limited water solubility. Bitumen/ asphalts are expected to be resistant to biodegradation, and those components that are soluble in water are expected to be resistant to hydrolysis. When bitumen/ asphalts are heated to facilitate paving or roofing applications, the lighter, more volatile components are distilled into the atmosphere. They condense as they cool, forming small droplets of liquid known as bitumen or asphalt fume condensate. The majority of hydrocarbons in bitumen/ asphalts are not susceptible to direct photolysis, since they do not have functional groups that absorb sunlight greater than 290 nm. However, certain aromatic and unsaturated compound members have the potential to undergo photolysis because they absorb light in the environmental UV region. Since bitumens/ asphalts contain high molecular weight hydrocarbons, partitioning to the atmosphere is not considered to be important.
When compositionally analysing bitumens/ asphalts for certain toxicity endpoints the percentage of 3- to 7-ring polyaromatic hydrocarbons (PAHs) is important. The levels of 3- to 7-ring PAHs are expected to be low considering the processes used to manufacture these substances.. Fumes generated experimentally at high temperatures are more likely to contain carcinogenic PAHs than fumes generated at the lower temperatures usually seen in field samples. Therefore, generating conditions are expected to significantly affect toxicity.
.
Ecotoxicity:
Bitumens/ asphalts by analogy with other high molecular weight hydrocarbons are not likely to show adverse acute or chronic ecological effects in aquatic species.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
1,2,4-trimethyl benzene	LOW (Half-life = 56 days)	LOW (Half-life = 0.67 days)
1,3,5-trimethyl benzene	HIGH	HIGH
titanium dioxide	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
1,2,4-trimethyl benzene	LOW (BCF = 275)
1,3,5-trimethyl benzene	LOW (BCF = 342)
titanium dioxide	LOW (BCF = 10)

Mobility in soil

Ingredient	Mobility
1,2,4-trimethyl benzene	LOW (KOC = 717.6)
1,3,5-trimethyl benzene	LOW (KOC = 703)
titanium dioxide	LOW (KOC = 23.74)

SECTION 13 Disposal considerations

Waste treatment methods

Continued...

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible. ▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▸ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material). ▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	NO

Land transport (DOT)

UN number	1999
UN proper shipping name	Tars, liquid including road oils and cutback bitumens
Transport hazard class(es)	Class: 3 Subrisk: Not Applicable
Packing group	III
Environmental hazard	Not Applicable
Special precautions for user	Hazard Label: 3 Special provisions: B1, B13, IB3, T1, TP3

Air transport (ICAO-IATA / DGR)

UN number	1999
UN proper shipping name	Tars, liquid including road asphalt and oils, bitumen and cut backs
Transport hazard class(es)	ICAO/IATA Class: 3 ICAO / IATA Subrisk: Not Applicable ERG Code: 3L
Packing group	III
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: A3 Cargo Only Packing Instructions: 366 Cargo Only Maximum Qty / Pack: 220 L Passenger and Cargo Packing Instructions: 355 Passenger and Cargo Maximum Qty / Pack: 60 L Passenger and Cargo Limited Quantity Packing Instructions: Y344 Passenger and Cargo Limited Maximum Qty / Pack: 10 L

Sea transport (IMDG-Code / GGVSee)

UN number	1999

Continued...

UN proper shipping name	TARS, LIQUID including road oils, and cutback bitumens
Transport hazard class(es)	IMDG Class: 3 IMDG Subrisk: Not Applicable
Packing group	III
Environmental hazard	Not Applicable
Special precautions for user	EMS Number: F-E, S-E Special provisions: 955 Limited Quantities: 5 L

Transport in bulk according to Annex II of MARPOL and the IBC code

Not Applicable

Transport in bulk in accordance with MARPOL Annex V and the IMSBC Code

Product name	Group
bitumen (petroleum)	Not Available
white spirit	Not Available
1,2,4-trimethyl benzene	Not Available
1,3,5-trimethyl benzene	Not Available
gilsonite	Not Available
naphtha petroleum, light aromatic solvent	Not Available
trimethylbenzene (mixed isomers)	Not Available
silica crystalline - quartz	Not Available
titanium dioxide	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
bitumen (petroleum)	Not Available
white spirit	Not Available
1,2,4-trimethyl benzene	Not Available
1,3,5-trimethyl benzene	Not Available
gilsonite	Not Available
naphtha petroleum, light aromatic solvent	Not Available
trimethylbenzene (mixed isomers)	Not Available
silica crystalline - quartz	Not Available
titanium dioxide	Not Available

SECTION 15 Regulatory information

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

bitumen (petroleum) is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
US - Alaska Air Quality Control - Concentrations Triggering an Air Quality Episode for Air Pollutants Other Than PM-2.5
US - Massachusetts - Right To Know Listed Chemicals
US DOE Temporary Emergency Exposure Limits (TEELs)
US NIOSH Carcinogen List
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Limits (PELs) Table Z-1
US OSHA Permissible Exposure Limits (PELs) Table Z-3
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

white spirit is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
US - Massachusetts - Right To Know Listed Chemicals
US DOE Temporary Emergency Exposure Limits (TEELs)
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Limits (PELs) Table Z-1
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

1,2,4-trimethyl benzene is found on the following regulatory lists

Continued...

US - Massachusetts - Right To Know Listed Chemicals
US DOE Temporary Emergency Exposure Limits (TEELs)
US EPA Integrated Risk Information System (IRIS)
US EPCRA Section 313 Chemical List
US NIOSH Recommended Exposure Limits (RELs)
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

1,3,5-trimethyl benzene is found on the following regulatory lists

US - Massachusetts - Right To Know Listed Chemicals
US DOE Temporary Emergency Exposure Limits (TEELs)
US EPA Integrated Risk Information System (IRIS)
US NIOSH Recommended Exposure Limits (RELs)
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances
US TSCA Section 4/12 (b) - Sunset Dates/Status

gilsonite is found on the following regulatory lists

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
US - Alaska Air Quality Control - Concentrations Triggering an Air Quality Episode for Air Pollutants Other Than PM-2.5
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Limits (PELs) Table Z-1
US OSHA Permissible Exposure Limits (PELs) Table Z-3

naphtha petroleum, light aromatic solvent is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
US DOE Temporary Emergency Exposure Limits (TEELs)
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

trimethylbenzene (mixed isomers) is found on the following regulatory lists

US - Massachusetts - Right To Know Listed Chemicals
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

silica crystalline - quartz is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans
US - California Proposition 65 - Carcinogens
US - California Safe Drinking Water and Toxic Enforcement Act of 1986 - Proposition 65 List
US - Massachusetts - Right To Know Listed Chemicals
US DOE Temporary Emergency Exposure Limits (TEELs)
US National Toxicology Program (NTP) 15th Report Part A Known to be Human Carcinogens
US NIOSH Carcinogen List
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Carcinogens Listing
US OSHA Permissible Exposure Limits (PELs) Table Z-1
US OSHA Permissible Exposure Limits (PELs) Table Z-3
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

titanium dioxide is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
US - Alaska Air Quality Control - Concentrations Triggering an Air Quality Episode for Air Pollutants Other Than PM-2.5
US - California Proposition 65 - Carcinogens
US - California Safe Drinking Water and Toxic Enforcement Act of 1986 - Proposition 65 List
US - Massachusetts - Right To Know Listed Chemicals
US DOE Temporary Emergency Exposure Limits (TEELs)
US List of Active Substances Exempt from the TSCA Inventory Notifications (Active-Inactive) Rule
US NIOSH Carcinogen List
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Limits (PELs) Table Z-1
US OSHA Permissible Exposure Limits (PELs) Table Z-3
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

Federal Regulations

Superfund Amendments and Reauthorization Act of 1986 (SARA)

Section 311/312 hazard categories

Flammable (Gases, Aerosols, Liquids, or Solids)	Yes
Gas under pressure	No
Explosive	No
Self-heating	No
Pyrophoric (Liquid or Solid)	No
Pyrophoric Gas	No
Corrosive to metal	No
Oxidizer (Liquid, Solid or Gas)	No
Organic Peroxide	No
Self-reactive	No
In contact with water emits flammable gas	No
Combustible Dust	No
Carcinogenicity	Yes
Acute toxicity (any route of exposure)	No

Continued...

Reproductive toxicity	No
Skin Corrosion or Irritation	Yes
Respiratory or Skin Sensitization	Yes
Serious eye damage or eye irritation	Yes
Specific target organ toxicity (single or repeated exposure)	Yes
Aspiration Hazard	Yes
Germ cell mutagenicity	Yes
Simple Asphyxiant	No
Hazards Not Otherwise Classified	No

US. EPA CERCLA Hazardous Substances and Reportable Quantities (40 CFR 302.4)

None Reported

State Regulations

US. California Proposition 65

⚠ **WARNING:** This product can expose you to chemicals including **silica crystalline - quartz, titanium dioxide**, which are known to the State of California to cause cancer. For more information, go to www.P65Warnings.ca.gov.

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (bitumen (petroleum); white spirit; 1,2,4-trimethyl benzene; 1,3,5-trimethyl benzene; gilsonite; naphtha petroleum, light aromatic solvent; trimethylbenzene (mixed isomers); silica crystalline - quartz)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (gilsonite)
Japan - ENCS	No (bitumen (petroleum); gilsonite)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	No (gilsonite)
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	12/09/2022
Initial Date	12/10/2022

CONTACT POINT

PLEASE NOTE THAT TITANIUM DIOXIDE IS NOT PRESENT IN CLEAR OR NEUTRAL BASES

SDS Version Summary

Version	Date of Update	Sections Updated
0.2	12/09/2022	Ingredients, Physical Properties, Synonyms, Name

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.
The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
ES: Exposure Standard
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value

Continued...

LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index
AIIC: Australian Inventory of Industrial Chemicals
DSL: Domestic Substances List
NDSL: Non-Domestic Substances List
IECSC: Inventory of Existing Chemical Substance in China
EINECS: European INventory of Existing Commercial chemical Substances
ELINCS: European List of Notified Chemical Substances
NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory
KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals
PICCS: Philippine Inventory of Chemicals and Chemical Substances
TSCA: Toxic Substances Control Act
TCSI: Taiwan Chemical Substance Inventory
INSQ: Inventario Nacional de Sustancias Químicas
NCI: National Chemical Inventory
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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end of SDS