Durotech Bitumen Primer

Durotech Industries

Chemwatch Hazard Alert Code: **2**

Chemwatch: **5245-80**
Version No: **2.1.1.1**
Safety Data Sheet according to WHS and ADG requirements

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SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	Durotech Bitumen Primer
Synonyms	Not Available
Proper shipping name	TARS, LIQUID, including road oils, and cutback bitumens
Other means of identification	Not Available

Relevant identified uses of the substance or mixture and uses advised against

Relevant identified uses	Use according to manufacturer's directions. Coating material.

Details of the supplier of the safety data sheet

Registered company name	Durotech Industries
Address	14 Essex Street Minto NSW 2566 Australia
Telephone	02 9603 1177
Fax	02 9475 5059
Website	www.durotechindustries.com.au
Email	accounts@durotechindustries.com.au

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	0421 670 636
Other emergency telephone numbers	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

HAZARDOUS CHEMICAL. DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

Poisons Schedule	S5
Classification [1]	Flammable Liquid Category 3, Acute Toxicity (Oral) Category 4, Acute Toxicity (Dermal) Category 4, Acute Toxicity (Inhalation) Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Skin Sensitizer Category 1, Carcinogenicity Category 2, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Specific target organ toxicity - single exposure Category 3 (narcotic effects), Aspiration Hazard Category 1, Acute Aquatic Hazard Category 2, Chronic Aquatic Hazard Category 2
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI*

Label elements

GHS label elements	
SIGNAL WORD	**DANGER**

Hazard statement(s)

H226	Flammable liquid and vapour.
H302	Harmful if swallowed.
H312	Harmful in contact with skin.
H332	Harmful if inhaled.
H315	Causes skin irritation.

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H319	Causes serious eye irritation.
H317	May cause an allergic skin reaction.
H351	Suspected of causing cancer.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.
H304	May be fatal if swallowed and enters airways.
H411	Toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
P271	Use in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P281	Use personal protective equipment as required.
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.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER or doctor/physician.
P308+P313	IF exposed or concerned: Get medical advice/attention.
P331	Do NOT induce vomiting.
P362	Take off contaminated clothing and wash before reuse.
P370+P378	In case of fire: Use alcohol resistant foam or fine spray/water fog for extinction.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell.
P303+P361+P353	IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P330	Rinse mouth.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
8052-42-4	40-60	bitumen (petroleum)
9005-90-7	40-60	gum turpentine

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.

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	▸ 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. 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 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 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.

Indication of any immediate medical attention and special treatment needed

For acute or short term repeated exposures to petroleum distillates or related hydrocarbons:

- Primary threat to life, from pure petroleum distillate ingestion and/or inhalation, is respiratory failure.
- Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO2 50 mm Hg) should be intubated.
- Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.
- Lavage is indicated in patients who require decontamination; ensure use of cuffed endotracheal tube in adult patients. [Ellenhorn and Barceloux: Medical Toxicology]

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.
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]

In acute poisonings by essential oils the stomach should be emptied by aspiration and lavage. Give a saline purgative such as sodium sulfate (30 g in 250 ml water) unless catharsis is already present. Demulcent drinks may also be given. Large volumes of fluid should be given provided renal function is adequate. [MARTINDALE: The Extra Pharmacopoeia, 28th Ed.]

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

- Water spray or fog.
- Foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.

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

Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ May be violently or explosively reactive. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ If safe, switch off electrical equipment until vapour fire hazard removed. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ Avoid spraying water onto liquid pools. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire.

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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 (CO_2) , nitrogen oxides (NOx) , sulfur oxides (SOx) , sulfur dioxide (SO_2) , other pyrolysis products typical of burning organic material. May emit clouds of acrid smoke **NOTE:** Burns with intense heat. Produces melting, flowing, burning liquid and dense acrid black smoke. **CARE**: Water in contact with hot liquid may cause foaming and a steam explosion with wide scattering of hot oil and possible severe burns. Foaming may cause overflow of containers and may result in possible fire.
HAZCHEM	2W

SECTION 6 ACCIDENTAL RELEASE MEASURES

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	**CARE**: Absorbent materials wetted with occluded oil must be moistened with water as they may auto-oxidize, become self heating and ignite. Some oils slowly oxidise when spread in a film and oil on cloths, mops, absorbents may autoxidise and generate heat, smoulder, ignite and burn. In the workplace oily rags should be collected and immersed in water. ▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ May be violently or explosively reactive. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ No smoking, naked lights or ignition sources. ▸ Increase ventilation. ▸ Stop leak if safe to do so. ▸ Water spray or fog may be used to disperse / absorb vapour. ▸ Contain spill with sand, earth or vermiculite. ▸ Use only spark-free shovels and explosion proof equipment. ▸ Collect recoverable product into labelled containers for recycling. ▸ Absorb remaining product with sand, earth or vermiculite. ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	▸ 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. ▸ **DO NOT** allow clothing wet with material to stay in contact with skin ▸ 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.

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	▸ 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.
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. Essential oil oxidation accelerates with the concentration of dissolved oxygen, which in turn depends largely on oxygen partial pressure in the head-space as well as ambient temperature. Depending on the particular essential oil and the ambient temperature, oxidation will not necessarily be prevented by avoidance of container head-space. Instead essential oils should be treated with inert gas such as argon, cautiously flushed through to displace remaining air, to prevent the formation of peroxides efficiently.

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	**HAZARD**: ▸ Although anti-oxidants may be present, in the original formulation, these may deplete over time as they come into contact with air. ▸ Rags wet / soaked with unsaturated hydrocarbons / drying oils may auto-oxidise; generate heat and, in-time, smoulder and ignite. This is especially the case where oil-soaked materials are folded, bunched, compressed, or piled together - this allows the heat to accumulate or even accelerate the reaction ▸ Oily cleaning rags should be collected regularly and immersed in water, or spread to dry in safe-place away from direct sunlight.or stored, immersed, in solvents in suitably closed containers. ▸ 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. ▸ Avoid reaction with oxidising agents

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	bitumen (petroleum)	Bitumen fumes	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	gum turpentine	Turpentine (wood)	557 mg/m3 / 100 ppm	Not Available	Not Available	Sen

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
bitumen (petroleum)	Petroleum asphalt; (Bitumen)	30 mg/m3	330 mg/m3	2,000 mg/m3
gum turpentine	Turpentine, (Alpha and beta pinene, 80-56-8)	60 ppm	120 ppm	1,500 ppm

Ingredient	Original IDLH	Revised IDLH
bitumen (petroleum)	Not Available	Not Available
gum turpentine	1,500 ppm	800 ppm

MATERIAL DATA

Exposure controls

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Appropriate engineering controls

Care: Atmospheres in bulk storages and even apparently empty tanks may be hazardous by oxygen depletion. Atmosphere must be checked before entry.

Requirements of State Authorities concerning conditions for tank entry must be met. Particularly with regard to training of crews for tank entry; work permits; sampling of atmosphere; provision of rescue harness and protective gear as needed
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
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.

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 moisturizer 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.

For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.

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	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
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.
Thermal hazards	Not Available

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 5 x ES	A-AUS / Class 1 P2	-	A-PAPR-AUS / Class 1 P2
up to 25 x ES	Air-line*	A-2 P2	A-PAPR-2 P2
up to 50 x ES	-	A-3 P2	-
50+ x ES	-	Air-line**	-

* - Continuous-flow; ** - Continuous-flow or positive pressure demand
^ - 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.

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Black viscous flammable liquid; does not mix with water.

Physical state	Liquid	**Relative density (Water = 1)**	0.92
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	>450
pH (as supplied)	Not Applicable	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	150	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	52 CC	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Flammable.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	7	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	1	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water (g/L)	Immiscible	**pH as a solution (1%)**	6-7
Vapour density (Air = 1)	4	**VOC g/L**	Not Available

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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. Inhalation hazard is increased at higher temperatures. 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 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. 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. Signs of acute turpentine intoxication in rats include ataxia, tremour, convulsions, tachypnea, decreased tidal volume, and death due to sudden apnea. Turpentine components tend to concentrate in the spleen and brain. Occupational exposure to turpentine vapours include respiratory irritation, renal injury and deaths. Inhalation exposure to oil of turpentine at 75-175 ppm produces irritation of the nose and throat; at 750-1000 ppm, exposure for several hours produces headache, dizziness, nausea and tachycardia. Heavy overexposures produce coughing, choking, dyspnea, bronchitis, chest pain, burning, sensation in the mouth, throat, oesophagus and stomach, vomiting, diarrhoea, abdominal pain, painful urination with violet-like urine odour. Bladder and kidney irritation with haematuria, albuminuria and glycosuria. Central nervous system depression may occur after initial excitement, followed by ataxia, confusion, anorexia, stupour, tinnitus, hallucinations, distorted perceptions, antipsychotic changes, delayed convulsions, coma and death due to respiratory failure 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	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. 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). Swallowing pieces of bitumen may produce pyloric obstruction due to accumulation in the stomach and the formation of a stony concretion. The mean lethal oral dose ranges between 19 and 50 ml turpentine. Aspiration may cause pathognomonic dyspnea, acute pulmonary oedema, and cyanosis. Death occurs in central arrest. Acute turpentine poisoning has been associated with glucosuria, haematuria, albuminuria, and anuria.
Skin Contact	Skin contact with the material may be harmful; systemic effects may result following absorption. 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 Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. 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. Older preparations of turpentine are said to contain high concentrations of delta-carotene, a substance said to be responsible for eczema. Workers in the

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	chemical, rubber and welding industries have developed contact dermatoses. Skin contact may produce erythema, eczema. itching, chemical burns and allergic sensitisation
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. 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 At 175 ppm turpentine vapour, eye irritation is evident. Direct liquid contact may produce blepharospasm, conjunctival hyperaemia, slight injury to the corneal epithelium, corneal burns and temporary erosion of the epithelium.
Chronic	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. 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. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. In the presence of air, a number of common flavour and fragrance chemicals can form peroxides surprisingly fast. Antioxidants can in most cases minimise the oxidation. Fragrance terpenes are generally easily oxidised in air. Non-oxidised limonene, linalool and caryophyllene turned out to be very weak sensitizers, however after oxidation limonene hydroperoxide and linalool hydroperoxide are strong sensitizers. Of the patients tested 2.6% showed positive reaction to oxidised limonene, 1.3% to oxidised linalool, 1.1% to linalool hydroperoxide, 0.5% to oxidised caryophyllene, while testing with caryophyllene oxide and oxidised myrcene resulted in few positive patch tests. 2/3 of the patients reacting positive to oxidised terpenes had fragrance related contact allergy and/or positive history for adverse reactions to fragrances. As well as the hydroperoxides produced by linalol, limonene and delta-3-carene other oxidation and resinification effects progressively causes other fairly major changes in essential oil quality over time. Autoxidation of fragrance terpenes contributes greatly to fragrance allergy, which emphasizes the need of testing with compounds that patients are actually exposed to and not only with the ingredients originally applied in commercial formulations. Some oxidised terpenoids as well as some aged essential oils have revealed skin-sensitising capacities, leading to a hypersensitivity reaction synonymous to allergic contact dermatitis. The allergenic potency in some flavouring could be mainly attributed to terpenoid hydroperoxides intermediately built-up upon autoxidation, while their non-oxidised counterparts as well as most degradation products were proven to be not or only barely irritating 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 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 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*] Essential oils and isolates derived from the Pinacea family, including Pinus and Abies genera, should only be used when the level of peroxides is kept to the

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lowest practicable level, for instance by adding antioxidants at the time of production. Such products should have a peroxide value of less than 10 millimoles peroxide per liter. Based on the published literature mentioning sensitising properties when containing peroxides (Food and Chemical Toxicology 11,1053(1973); 16,843(1978); 16,853(1978).

Hydroperoxides of d-limonene are potent contact allergens when studied in guinea pigs. They may result when d-limonene is unstabilised against oxidation, or upon prolonged standing at room temperature and/ or upon exposure to light, or when stabiliser levels diminish. The two major hydroperoxides in auto-oxidised d-limonene, are cis- and trans- limonene-2-hydroperoxide (2-hydroperoxy-p-mentha-6,8-diene). In photo-oxidised d-limonene, they represent a minor fraction. Hydroperoxides may bind to proteins of the skin to make antigens either via a radical mechanism or after reactions to give epoxides. The cross-reactivity between the epoxide limonene-1,2-oxide, a potent contact allergen, and the hydroperoxides is NOT significant, indicating different mechanisms of sensitisation.

d-Limonene was considered to be weakly carcinogenic for the mouse fore-stomach epithelium, but not tumour producing. In 13-week and 2-year gavage-studies, male rats showed a range of compound-related kidney lesions including exacerbation of age-related nephropathy, mineralisation in the renal medulla, hyperplasia of the transitional epithelium overlying the renal papilla and proliferation of the renal tubular epithelium. Neoplasms were believed to be caused by progression to tubular cell hyperplasia to tubular cell adenomas and, with increasing size, to adenocarcinomas or carcinomas. The similarity of the nephrotoxicity caused by trichloroethylene and N-(4'-fluoro-4-biphenyl)acetamide, tris(2,3-dibromopropyl)phosphate in rats and the species specific nature of the response suggests that degeneration and necrosis of convoluted tubules may be associated with the accumulation of alpha-2u-globin (a2u-G). Since a2u-G is a species and gender-specific protein that is causal for both the cytotoxic and carcinogenic response in male rats, extrapolation of d-limonene carcinogenicity data from rat studies to other species (including humans) is probably not warranted. Humans do not synthesise a2u-G; they do however produce other related low molecular weight proteins capable of binding chemicals that cause a2u-G nephropathy in rats but this does not necessarily connote human risk. The Risk Assessment Forum of the USA EPA concluded;

- Male renal rat tumours arising as a result of a process involving a2u-G accumulation do not contribute to the qualitative weight-of-evidence that the chemical poses a human carcinogenic hazard. Such tumours are included in dose-response extrapolations for the estimation of human carcinogenic risk.
- If the chemical induces a2u-G accumulation in male rats, the associated nephropathy is not to be used as an end-point for determining non-carcinogenic hazard.

Parenteral turpentine has been used as an abortifacient where it has induced peritonitis, pelvic necrosis, and inflammation, and pulmonary oedema. Repeated or prolonged exposure to oil of turpentine may produce cerebral atrophy, behavioural changes, anaemia and bone marrow injury, and glomerulonephritis.

Durotech Bitumen Primer

TOXICITY	IRRITATION
Not Available	Not Available

bitumen (petroleum)

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg[1]	Not Available
Oral (rat) LD50: >5000 mg/kg[1]	

gum turpentine

TOXICITY	IRRITATION
Oral (rat) LD50: 5760 mg/kg[2]	Eye (human): 175 ppm

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*

BITUMEN (PETROLEUM)

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. 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. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
No significant acute toxicological data identified in literature search.

WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.

GUM TURPENTINE

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.
For bicyclic terpenes:
Acute toxicity: The literature abounds with clinical reports of accidental and intentional acute poisoning with pinene-based turpentine.
Rat oral LD50 values are available for *alpha*-pinene, *beta*-pinene, camphene and turpentine oil and indicate these materials to be very low in oral acute toxicity with LD50 values in the range from 3388 mg/kg to greater than 5000 mg/kg. Rabbit dermal LD50 values similarly indicate very low toxicities with values greater than the limit doses of 2000 or 5000 mg/kg.
Acute inhalation toxicity has been measure in different animal species. The acute LC50 was reported to be 13,500 mg/m3 in rats, 13,500 mg/m3 in guinea pigs, and 9000 mg/m3 in mice . The acute inhalation LC50 of commercial grade turpentine in Wistar rats is reported to be in the range of 12,000-20,000 mg/m3 for 1 to 6 hour exposures and the LC50 for a 2-hour exposure in Swiss-Webster mice is 29,000 mg/m3 . Based on these results the acute oral, dermal, and inhalation toxicities of bicyclic terpene hydrocarbons is concluded to be low.
Repeat dose toxicity: A 28-day repeat dose study has been performed with camphene according to an OECD Guideline 407 in both sexes of Wistar rats. Animals of both sexes at the 1000 mg/kg bw/day dose exhibited vacuolization of hepatocytes and increase liver weights. Male rats also exhibited *alpha*-2-microglobulin-type nephrotoxicity at all dose levels.
Subsequent investigations have shown that the *alpha*-2-microglobulin nephropathy found in the F344/N male rat does not develop in mammals that do not express the hepatic form of *alpha*-2-microglobulin (e.g. other strains of rats, mice, dogs, humans). Therefore, the nephrotoxicity observed in the camphene study in male F344 rats is not relevant to the human health risk assessment. Based on liver toxicity, the NOAEL for this study is concluded to be 250 mg/kg bw/day
Reproductive toxicity: In the a-animal species study, no reproductive effects were observed when dose levels of up to 260 to 600 mg/kg bw of an essential oil predominantly composed of bicyclic terpene hydrocarbons (*alpha*-pinene, *beta*-pinene, and sabinene) was administered daily to mice, rats, or hamsters during gestation. When this data is combined with the fact that no adverse effects were observed to the reproductive organs in a 28-day study with camphene at dose levels up to 250 mg/kg bw/day, it is concluded that bicyclic terpene hydrocarbons including *alpha*-pinene and *beta*-pinene are not reproductive toxicants
Two ninety day inhalation studies have been performed for alpha-pinene in which a full complement of male and female sex organs and tissues were subjected

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to histopathological examination. Both studies reported no microscopic changes that could be associated with exposure to the test substance. Taking into account the lack of any effects to females in a earlier teratology study, the absence of any maternal or developmental effects in a reproductive/developmental study of a pinene-based oil and for a structurally related monoterpene hydrocarbon, myrcene, it can be concluded that the members of this category show no significant reproductive or developmental toxicity

Developmental toxicity:. Based on the NOAELs for maternal and developmental toxicity in studies with camphene (250 and 1000 mg/kg bw/day) and a terpene hydrocarbon mixture containing *alpha*- and *beta*-pinene and camphene (688 mg/kg bw/day) , and the lack of any signs of maternal or developmental toxicity in a mice,

rats, or hamsters given 260 to 600 mg/kg bw/day of a mixture composed primarily (>80%) of *alpha*- and *beta*-pinene and sabinene, it is concluded that bicyclic terpene hydrocarbons are not maternal or developmental toxicants.

Genotoxicity:

In vitro: *In vitro* genotoxicity assays available for *alpha*-pinene, *beta*-pinene and camphene demonstrate that these substances have a little, if any, genotoxic potential. In standard Ames assays of *alpha*-pinene, *beta*-pinene and camphene, *Salmonella typhimurium* strains TA97, TA98, TA100, TA1535, TA1537, and TA1538 provided no evidence of mutagenicity at any dose tested.

In vivo: Based on the lack of any evidence of genotoxicity in numerous *in vitro* assays with and without metabolic activation, it is unlikely that any of these bicyclic terpenes would exhibit a significant genotoxic potential *in vivo*.

d-Limonene is readily absorbed by inhalation and ingestion. Dermal absorption is reported to be lower than by the inhalation route. d-Limonene is rapidly distributed to different tissues in the body, readily metabolised and eliminated primarily through the urine.

Limonene exhibits low acute toxicity by all three routes in animals. Limonene is a skin irritant in both experimental animals and humans. Limited data are available on the potential to cause eye and respiratory irritation. Autooxidised products of d-limonene have the potential to be skin sensitisers. Limited data are available in humans on the potential to cause respiratory sensitisation. Autooxidation of limonene occurs readily in the presence of light and air forming a variety of oxygenated monocyclic terpenes. Risk of skin sensitisation is high in situations where contact with oxidation products of limonene occurs.

Renal tumours induced by limonene in male rats is though to be sex and species specific and are not considered relevant to humans. Repeated exposure affects the amount and activity of liver enzymes, liver weight, blood cholesterol levels and bile flow in animals. Increase in liver weight is considered a physiological adaption as no toxic effects on the liver have been reported. From available data it is not possible to identify an NOAEL for these effects. Limonene is neither genotoxic or teratogenic nor toxic to the reproductive system.

Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and connubial contact dermatitis occur.

Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes.

Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits.

Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water.

Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis.

Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a suffcient degree of fragrance contact allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the allergen. As a consequence, symptoms, i.e. allergic contact dermatitis, may occur upon re-exposure to the fragrance allergen(s) in question. Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or hands. It may affect fitness for work and the quality of life of the individual. Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure.

Hands: Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema may often be complicated by sensitisation .Fragrance allergy may be a relevant problem in patients with hand eczema; perfumes are present in consumer products to which their hands are exposed. A significant relationship between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe) chronic hand eczema may not be clear.

Axillae Bilateral axillary (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy.

Face Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of of being fragrance allergic.

Irritant reactions (including contact urticaria): Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this, Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported . The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested , but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen.

Pigmentary anomalies: The term “pigmented cosmetic dermatitis” was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified.. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil.

Photo-reactions Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon . Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. Phototoxic reactions still occur but are rare.

General/respiratory: Fragrances are volatile and therefore, in addition to skin exposure, a perfume also exposes the eyes and naso-respiratory tract. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma . Asthma-like symptoms can be provoked by sensory mechanisms. In an epidemiological investigation, a significant association

Continued...

was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.
Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A **prehapten** is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems.
In the case of prehaptens, it is possible to prevent activation outside the body to a certain extent by different measures, e.g. prevention of air exposure during handling and storage of the ingredients and the final product, and by the addition of suitable antioxidants. When antioxidants are used, care should be taken that they will not be activated themselves and thereby form new sensitisers.
Prehaptens
Most terpenes with oxidisable allylic positions can be expected to autoxidise on air exposure due to their inherent properties. Depending on the stability of the oxidation products that are formed, a difference in the sensitisation potency of the oxidised terpenes can be seen
Autoxidation is a free radical chain reaction in which hydrogen atom abstraction in combination with addition of oxygen forms peroxyl radicals. The reaction shows selectivity for positions where stable radicals can be formed. So far, all fragrance substances that have been investigated with regard to the influence of autoxidation on the allergenic potential, including identification of formed oxidation products, have oxidisable allylic positions that are able to form hydroperoxides and/or hydrogen peroxide as primary oxidation products upon air exposure. Once the hydroperoxides have been formed outside the skin they form specific antigens and act as skin sensitisers. Secondary oxidation products such as aldehydes and epoxides can also be allergenic, thus further increasing the sensitisation potency of the autoxidation mixture. The process of photoactivation may also play a role, but further research is required to establish whether this activation route is currently underestimated in importance due to insufficient knowledge of the true haptens in this context.
It should be noted that activation of substances via air oxidation results in various haptens that might be the same or cross-reacting with other haptens (allergens). The main allergens after air oxidation of linalool and linalyl acetate are the hydroperoxides. If linalyl acetate is chemically hydrolysed outside the skin it can thereafter be oxidised to the same haptens as seen for linalool. A corresponding example is citronellol and citronellyl acetate. In clincal studies, concomitant reactions to oxidised linalool and oxidised linalyl acetate have been observed. Whether these reactions depend on cross-reactivity or are due to exposure to both fragrance substances cannot be elucidated as both have an allergenic effect themselves. Linalool and linalyl acetate are the main components of lavender oil. They autoxidise on air exposure also when present in the essential oil, and form the same oxidation products found in previous studies of the pure synthetic terpenes. Experimental sensitisation studies showed that air exposure of lavender oil increased the sensitisation potency. Patch test results in dermatitis patients showed a connection between positive reactions to oxidised linalool, linalyl acetate and lavender oil.
Prohaptens
Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohaptens.
In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal.
The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin . These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohaptens can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity.
QSAR prediction: The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptens) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptens. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation.

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 available but does not fill the criteria for classification*
✔ *– Data available to make classification*
⊘ *– Data Not Available to make classification*

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
gum turpentine	LC50	96	Fish	=0.01mg/L	1
Legend:	*Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 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*				

Very toxic 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.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
	No Data available for all ingredients	No Data available for all ingredients

Continued...

Bioaccumulative potential

Ingredient	Bioaccumulation
	No Data available for all ingredients

Mobility in soil

Ingredient	Mobility
	No Data available for all ingredients

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

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	
HAZCHEM	2W

Land transport (ADG)

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	Special provisions: 223 Limited quantity: 5 L

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

Continued...

	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	
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	Marine Pollutant	
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

SECTION 15 REGULATORY INFORMATION

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

BITUMEN (PETROLEUM)(8052-42-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards | Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists

GUM TURPENTINE(9005-90-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards | Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (gum turpentine; bitumen (petroleum))
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	N (bitumen (petroleum))
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
Legend:	*Y = All ingredients are on the inventory* *N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 OTHER INFORMATION

Other information

Ingredients with multiple cas numbers

Name	CAS No
gum turpentine	9005-90-7, 8006-64-2, 8052-14-0

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.

Continued...

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
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index