



Fibred Aluminum Roof Coating

Gardner-Gibson, Inc.

Version No: 1.1
Safety Data Sheet according to OSHA HazCom Standard (2012) requirements

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SECTION 1 Identification

Product Identifier

Product name	Fibred Aluminum Roof Coating
Synonyms	APOC 208 Silver Guard-F Fibred Aluminum Roof Coating
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	Asphalt Based Aluminum Reflective Roof Coating; Reflective Roof Coating; Fibred Aluminum Roof Coating
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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

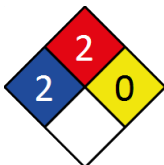
Emergency phone number

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
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Label elements

Hazard pictogram(s)	
Signal word	Danger

Hazard statement(s)

Fibred Aluminum Roof Coating

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.
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SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Continued...

Fibred Aluminum Roof Coating

Mixtures

CAS No	%[weight]	Name
111-84-2	0.1-5	<u>n-nonane</u>
25551-13-7	0.1-1.5	<u>trimethylbenzene (mixed isomers)</u>
8052-41-3.	30-60	<u>white spirit</u>
7429-90-5	10-30	<u>aluminium powder coated</u>
12002-43-6	1-5	<u>gilsonite</u>
8052-42-4	15-40	<u>bitumen (petroleum)</u>
95-63-6	0.1-1	<u>1,2,4-trimethyl benzene</u>
108-67-8	0.1-1	<u>1,3,5-trimethyl benzene</u>
93763-70-3	1-5	<u>perlite</u>
9004-34-6	1-5	<u>cellulose</u>
64742-95-6	0.1-1	<u>naphtha petroleum, light aromatic solvent</u>

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	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ 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	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ 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. <p>For thermal burns:</p> <ul style="list-style-type: none"> ▶ Decontaminate area around burn. ▶ Consider the use of cold packs and topical antibiotics. <p>For first-degree burns (affecting top layer of skin)</p> <ul style="list-style-type: none"> ▶ 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. <p>For second-degree burns (affecting top two layers of skin)</p> <ul style="list-style-type: none"> ▶ 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. <p>To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort):</p> <ul style="list-style-type: none"> ▶ 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. <p>For third-degree burns</p> <p>Seek immediate medical or emergency assistance.</p> <p>In the mean time:</p> <ul style="list-style-type: none"> ▶ 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.
Inhalation	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

Fibred Aluminum Roof Coating

- ▶ 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.

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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

Metal dust fires need to be smothered with sand, inert dry powders.

DO NOT USE WATER, CO₂ or FOAM.

- ▶ Use DRY sand, graphite powder, dry sodium chloride based extinguishers, G-1 or Met L-X to smother fire.
- ▶ Confining or smothering material is preferable to applying water as chemical reaction may produce flammable and explosive hydrogen gas.
- ▶ Chemical reaction with CO₂ may produce flammable and explosive methane.
- ▶ If impossible to extinguish, withdraw, protect surroundings and allow fire to burn itself out.
- ▶ **DO NOT** use halogenated fire extinguishing agents.

Special hazards arising from the substrate or mixture

Fire Incompatibility	<ul style="list-style-type: none"> ▶ Reacts with acids producing flammable / explosive hydrogen (H₂) gas ▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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Special protective equipment and precautions for fire-fighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves in the event of a fire. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Use fire fighting procedures suitable for surrounding area. ▶ 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. ▶ Equipment should be thoroughly decontaminated after use.
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Fire/Explosion Hazard	<p>Combustion products include: carbon dioxide (CO₂)</p> <ul style="list-style-type: none"> ▶ DO NOT disturb burning dust. Explosion may result if dust is stirred into a cloud, by providing oxygen to a large surface of hot metal. ▶ DO NOT use water or foam as generation of explosive hydrogen may result. <p>With the exception of the metals that burn in contact with air or water (for example, sodium), masses of combustible metals do not represent unusual fire risks because they have the ability to conduct heat away from hot spots so efficiently that the heat of combustion cannot be maintained - this means that it will require a lot of heat to ignite a mass of combustible metal. Generally, metal fire risks exist when sawdust, machine shavings and other metal 'fines' are present.</p> <p>Metal powders, while generally regarded as non-combustible:</p> <ul style="list-style-type: none"> ▶ May burn when metal is finely divided and energy input is high. ▶ May react explosively with water. ▶ May be ignited by friction, heat, sparks or flame. ▶ May REIGNITE after fire is extinguished. ▶ Will burn with intense heat. <p>Note:</p> <ul style="list-style-type: none"> ▶ Metal dust fires are slow moving but intense and difficult to extinguish. ▶ Containers may explode on heating. ▶ Dusts or fumes may form explosive mixtures with air. ▶ Gases generated in fire may be poisonous, corrosive or irritating. ▶ Hot or burning metals may react violently upon contact with other materials, such as oxidising agents and extinguishing agents used on fires involving ordinary combustibles or flammable liquids. ▶ Temperatures produced by burning metals can be higher than temperatures generated by burning flammable liquids ▶ Some metals can continue to burn in carbon dioxide, nitrogen, water, or steam atmospheres in which ordinary combustibles or flammable liquids would be incapable of burning.
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Fibred Aluminum Roof Coating

- ▶ 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.
- When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles.

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	<ul style="list-style-type: none"> ▶ 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	<p>Chemical Class: aromatic hydrocarbons For release onto land: recommended sorbents listed in order of priority.</p> <table border="1"> <thead> <tr> <th>SORBENT TYPE</th> <th>RANK</th> <th>APPLICATION</th> <th>COLLECTION</th> <th>LIMITATIONS</th> </tr> </thead> </table> <p>LAND SPILL - SMALL</p> <table border="1"> <tbody> <tr> <td>Feathers - pillow</td> <td>1</td> <td>throw</td> <td>pitchfork</td> <td>DGC, RT</td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>2</td> <td>shovel</td> <td>shovel</td> <td>R,W,SS</td> </tr> <tr> <td>cross-linked polymer- pillow</td> <td>2</td> <td>throw</td> <td>pitchfork</td> <td>R, DGC, RT</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>3</td> <td>shovel</td> <td>shovel</td> <td>R, I, P,</td> </tr> <tr> <td>treated clay/ treated natural organic - particulate</td> <td>3</td> <td>shovel</td> <td>shovel</td> <td>R, I</td> </tr> <tr> <td>wood fibre - pillow</td> <td>4</td> <td>throw</td> <td>pitchfork</td> <td>R, P, DGC, RT</td> </tr> </tbody> </table> <p>LAND SPILL - MEDIUM</p> <table border="1"> <tbody> <tr> <td>cross-linked polymer -particulate</td> <td>1</td> <td>blower</td> <td>skiploader</td> <td>R, W, SS</td> </tr> <tr> <td>treated clay/ treated natural organic - particulate</td> <td>2</td> <td>blower</td> <td>skiploader</td> <td>R, I</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>3</td> <td>blower</td> <td>skiploader</td> <td>R, I, P</td> </tr> <tr> <td>polypropylene - particulate</td> <td>3</td> <td>blower</td> <td>skiploader</td> <td>W, SS, DGC</td> </tr> <tr> <td>feathers - pillow</td> <td>3</td> <td>throw</td> <td>skiploader</td> <td>DGC, RT</td> </tr> <tr> <td>expanded mineral - particulate</td> <td>4</td> <td>blower</td> <td>skiploader</td> <td>R, I, W, P, DGC</td> </tr> </tbody> </table> <p>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</p>	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS	Feathers - pillow	1	throw	pitchfork	DGC, RT	cross-linked polymer - particulate	2	shovel	shovel	R,W,SS	cross-linked polymer- pillow	2	throw	pitchfork	R, DGC, RT	sorbent clay - particulate	3	shovel	shovel	R, I, P,	treated clay/ treated natural organic - particulate	3	shovel	shovel	R, I	wood fibre - pillow	4	throw	pitchfork	R, P, DGC, RT	cross-linked polymer -particulate	1	blower	skiploader	R, W, SS	treated clay/ treated natural organic - particulate	2	blower	skiploader	R, I	sorbent clay - particulate	3	blower	skiploader	R, I, P	polypropylene - particulate	3	blower	skiploader	W, SS, DGC	feathers - pillow	3	throw	skiploader	DGC, RT	expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC
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Personal Protective Equipment advice is contained in Section 8 of the SDS.

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	<p>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.</p> <p>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.</p>
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Fibred Aluminum Roof Coating

	<ul style="list-style-type: none"> ▶ 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. ▶ 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
<p style="text-align: center;">Other information</p>	<ul style="list-style-type: none"> ▶ 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. <p>In addition, for tank storages (where appropriate):</p> <ul style="list-style-type: none"> ▶ 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

<p style="text-align: center;">Suitable container</p>	<ul style="list-style-type: none"> ▶ 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.
<p style="text-align: center;">Storage incompatibility</p>	<p>For aluminas (aluminium oxide): Incompatible with hot chlorinated rubber. In the presence of chlorine trifluoride may react violently and ignite. -May initiate explosive polymerisation of olefin oxides including ethylene oxide. -Produces exothermic reaction above 200°C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals. -Produces exothermic reaction with oxygen difluoride. -May form explosive mixture with oxygen difluoride. -Forms explosive mixtures with sodium nitrate. -Reacts vigorously with vinyl acetate.</p> <p>Aluminium oxide is an amphoteric substance, meaning it can react with both acids and bases, such as hydrofluoric acid and sodium hydroxide, acting as an acid with a base and a base with an acid, neutralising the other and producing a salt.</p> <p>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.</p> <ul style="list-style-type: none"> ▶ 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 NO_x - these may be components of photochemical smogs. <p>Oxidation of Alkylaromatics: T.S.S Rao and Shubhra Awasthi: E-Journal of Chemistry Vol 4, No. 1, pp 1-13 January 2007</p>

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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.

- ▶ Many metals may incandesce, react violently, ignite or react explosively upon addition of concentrated nitric acid.
- ▶ 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.

Cellulose and its derivatives may react vigorously with calcium oxide, bleaching powder, perchlorates, perchloric acid, sodium chlorate, fluorine, nitric acid, sodium nitrate and sodium nitrite.

May be incompatible with aminacrine hydrochloride, chlorocresol, mercuric chloride, phenol, resorcinol, tannic acid and silver nitrate.

Metals exhibit varying degrees of activity. Reaction is reduced in the massive form (sheet, rod, or drop), compared with finely divided forms. The less active metals will not burn in air but:

- ▶ can react exothermically with oxidising acids to form noxious gases.
- ▶ catalyse polymerisation and other reactions, particularly when finely divided
- ▶ react with halogenated hydrocarbons (for example, copper dissolves when heated in carbon tetrachloride), sometimes forming explosive compounds.

- ▶ Finely divided metal powders develop pyrophoricity when a critical specific surface area is exceeded; this is ascribed to high heat of oxide formation on exposure to air.

- ▶ Safe handling is possible in relatively low concentrations of oxygen in an inert gas.

- ▶ Several pyrophoric metals, stored in glass bottles have ignited when the container is broken on impact. Storage of these materials moist and in metal containers is recommended.

- ▶ The reaction residues from various metal syntheses (involving vacuum evaporation and co-deposition with a ligand) are often pyrophoric.

Factors influencing the pyrophoricity of metals are particle size, presence of moisture, nature of the surface of the particle, heat of formation of the oxide, or nitride, mass, hydrogen content, stress, purity and presence of oxide, among others.

- ▶ Many metals in elemental form react exothermically with compounds having active hydrogen atoms (such as acids and water) to form flammable hydrogen gas and caustic products.
- ▶ Elemental metals may react with azo/diazo compounds to form explosive products.
- ▶ Some elemental metals form explosive products with halogenated hydrocarbons.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US NIOSH Recommended Exposure Limits (RELs)	n-nonane	Nonane	200 ppm / 1050 mg/m ³	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	white spirit	Stoddard solvent	500 ppm / 2900 mg/m ³	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	white spirit	Stoddard solvent	350 mg/m ³	Not Available	1800 (15-minute) mg/m ³	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	aluminium powder coated	Aluminum Metal (as Al)- Total dust	15 mg/m ³	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	aluminium powder coated	Aluminum Metal (as Al)- Respirable fraction	5 mg/m ³	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	aluminium powder coated	Inert or Nuisance Dust: Respirable fraction	5 mg/m ³ / 15 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	aluminium powder coated	Inert or Nuisance Dust: Total Dust	15 mg/m ³ / 50 mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	aluminium powder coated	Aluminum (pyro powders and welding fumes, as Al)	5 mg/m ³	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	aluminium powder coated	Aluminum - respirable	5 mg/m ³	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	aluminium powder coated	Aluminum - total	10 mg/m ³	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/m ³	Not Available	Not Available	Not Available

Continued...

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
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	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-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-3	bitumen (petroleum)	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	bitumen (petroleum)	Inert or Nuisance Dust: Respirable fraction	5 mg/m3 / 15 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 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	perlite	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	perlite	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	perlite	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	perlite	Inert or Nuisance Dust: Total Dust	15 mg/m3 / 50 mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	perlite	Perlite - total	10 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	perlite	Perlite - respirable	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	cellulose	Cellulose- Total dust	15 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	cellulose	Cellulose- Respirable fraction	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	cellulose	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	cellulose	Inert or Nuisance Dust: Total Dust	15 mg/m3 / 50 mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	cellulose	Cellulose - respirable	5 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	cellulose	Cellulose - total	10 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
n-nonane	600 ppm	830 ppm	5,000 ppm
white spirit	300 mg/m3	1,800 mg/m3	29500** mg/m3
bitumen (petroleum)	30 mg/m3	330 mg/m3	2,000 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
perlite	15 mg/m3	230 mg/m3	1,400 mg/m3
naphtha petroleum, light aromatic solvent	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3

Ingredient	Original IDLH	Revised IDLH
n-nonane	Not Available	Not Available
trimethylbenzene (mixed isomers)	Not Available	Not Available
white spirit	20,000 mg/m3	Not Available
aluminium powder coated	Not Available	Not Available
gilsonite	Not Available	Not Available
bitumen (petroleum)	Not Available	Not Available

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Ingredient	Original IDLH	Revised IDLH
1,2,4-trimethyl benzene	Not Available	Not Available
1,3,5-trimethyl benzene	Not Available	Not Available
perlite	Not Available	Not Available
cellulose	Not Available	Not Available
naphtha petroleum, light aromatic solvent	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
trimethylbenzene (mixed isomers)	E	≤ 0.1 ppm
naphtha petroleum, light aromatic solvent	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: This substance is classified by the NOHSC as Category 2 Probable Human Carcinogen for perlite:

Because perlite appears to have little adverse effect on the human lung and industrial experience indicates that it does not produce organic disease or toxic effects under controlled conditions of exposure, the recommended TLV-TWA is equivalent to that recommended for exposures to nuisance dust and is thought to protect workers from any significant risk of eye, skin and physical irritation.

For aluminium oxide and pyrophoric grades of aluminium:

Twenty seven year experience with aluminium oxide dust (particle size 96% 1.2 um) without adverse effects either systemically or on the lung, and at a calculated concentration equivalent to 2 mg/m³ over an 8-hour shift has lead to the current recommendation of the TLV-TWA.

The limit should also apply to aluminium pyro powders whose toxicity is reportedly greater than aluminium dusts and should be protective against lung changes.

For aluminium oxide:

The experimental and clinical data indicate that aluminium oxide acts as an "inert" material when inhaled and seems to have little effect on the lungs nor does it produce significant organic disease or toxic effects when exposures are kept under reasonable control.

[Documentation of the Threshold Limit Values], ACGIH, Sixth Edition

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)

For n-nonane and isomers:

Odour Threshold: 47 ppm

Inhalation of high concentrations of aliphatic hydrocarbons produces central nervous system depression leading to coma with inhibition of deep tendon reflexes. The TLV-TWA is protective against narcotic effects

produced at higher concentrations.

Odour Safety Factor(OSF)

OSF=4.3 (n-NONANE)

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

Cellulose is considered a nuisance dust which has little adverse effect on lung and does not produce significant organic disease or toxic effects when appropriate controls are applied.

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)

Odour threshold: 0.25 ppm.

The TLV-TWA is protective against ocular and upper respiratory tract irritation and is recommended for bulk handling of gasoline based on calculations of hydrocarbon content of gasoline vapour. A STEL is recommended to prevent mucous membrane and ocular irritation and prevention of acute depression of the central nervous system. Because of the wide variation in molecular weights of its components, the conversion of ppm to mg/m³ is approximate. Sweden recommends hexane type limits of 100 ppm and heptane and octane type limits of 300 ppm. Germany does not assign a value because of the widely differing compositions and resultant differences in toxic properties.

Odour Safety Factor (OSF)

OSF=0.042 (gasoline)

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)

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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:

ClassOSF 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 |

Exposure controls

Appropriate engineering controls	<p>Metal dusts must be collected at the source of generation as they are potentially explosive.</p> <ul style="list-style-type: none"> ▶ Avoid ignition sources. ▶ Good housekeeping practices must be maintained. ▶ Dust accumulation on the floor, ledges and beams can present a risk of ignition, flame propagation and secondary explosions. ▶ Do not use compressed air to remove settled materials from floors, beams or equipment ▶ Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation. ▶ Use non-sparking handling equipment, tools and natural bristle brushes. Cover and reseal partially empty containers. Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations. ▶ Do not allow chips, fines or dusts to contact water, particularly in enclosed areas. ▶ Metal spraying and blasting should, where possible, be conducted in separate rooms. This minimises the risk of supplying oxygen, in the form of metal oxides, to potentially reactive finely divided metals such as aluminium, zinc, magnesium or titanium. ▶ Work-shops designed for metal spraying should possess smooth walls and a minimum of obstructions, such as ledges, on which dust accumulation is possible. ▶ Wet scrubbers are preferable to dry dust collectors. ▶ Bag or filter-type collectors should be sited outside the workrooms and be fitted with explosion relief doors. ▶ Cyclones should be protected against entry of moisture as reactive metal dusts are capable of spontaneous combustion in humid or partially wetted states. ▶ Local exhaust systems must be designed to provide a minimum capture velocity at the fume source, away from the worker, of 0.5 metre/sec. ▶ Local ventilation and vacuum systems must be designed to handle explosive dusts. Dry vacuum and electrostatic precipitators must not be used, unless specifically approved for use with flammable/ explosive dusts. <p>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.</p> <table border="1" style="width: 100%;"> <tr> <td>Type of Contaminant:</td> <td>Air Speed:</td> </tr> <tr> <td>welding, brazing fumes (released at relatively low velocity into moderately still air)</td> <td>0.5-1.0 m/s (100-200 f/min.)</td> </tr> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" style="width: 100%;"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>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 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 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.</p>	Type of Contaminant:	Air Speed:	welding, brazing fumes (released at relatively low velocity into moderately still air)	0.5-1.0 m/s (100-200 f/min.)	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
Type of Contaminant:	Air Speed:														
welding, brazing fumes (released at relatively low velocity into moderately still air)	0.5-1.0 m/s (100-200 f/min.)														
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3: Intermittent, low production.	3: High production, heavy use														
4: Large hood or large air mass in motion	4: Small hood-local control only														
Personal protection															
Eye and face protection	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber 														

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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

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 and 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:

Fibred Aluminum Roof Coating

Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NAT+NEOPR+NITRILE	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

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

Continued...

Fibred Aluminum Roof Coating

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.

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	<p>Modified cellulose polymers formed by the reaction with the free hydroxyl groups in cellulose. The number of hydroxyl groups reacting, as well as the nature of the substituent, largely determine the physical properties, particularly solubility, of the product.</p> <p>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.</p> <p>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.</p> <p>Saturates consist mainly of long chain saturated hydrocarbons with some Saturates branching, alkyl aromatics with long side chains, and cyclic paraffins (naphthenes), with molecular weight of 500-1000.</p> <p>Asphaltenes consist mainly of substituted benzene and naphthenic-aromatic nuclei with alkyl side chain constituents, with molecular weight of 500-900.</p> <p>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</p> <p>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.</p> <p>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.</p>
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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)	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)	>38	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)	49.08
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	<450

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.

Fibred Aluminum Roof Coating

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

<p>Inhaled</p>	<p>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.</p> <p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Cellulose, after a single intratracheal dose (15 mg per animal) brought about fibrosing granulomatous bronchioloalveolitis and an increase of IgA production in the bronchioalveolar lavage. Fibrosing alveolitis showed moderate progression as a function of time. Injury of Type I pneumocytes and incomplete repair of Type II pneumocytes were detected. The damage of alveolar epithelium initiated and activated a series of processes that led to definite pulmonary alterations and pulmonary fibrosis leading to disintegration of the alveolo-capillary morphological functional unit. Tatrai, E. et al: Journal of Applied Toxicology; 16(2) 129-135 (1996)</p> <p>Some health effects associated with wood, cotton, flax, jute and hemp particles or fibres are not attributable to cellulose content but to other substances and/or impurities.</p> <p>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.</p> <p>Not normally a hazard due to non-volatile nature of product</p> <p>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.</p> <p>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.</p> <p>Concentrated nonane vapours may cause irritation of the nose and throat, headache, drowsiness, dizziness, confusion, nausea, tremors, incoordination and difficulty in breathing. Very high concentrations may cause unconsciousness and death.</p> <p>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</p> <p>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.</p> <p>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.</p> <p>Alkylbenzenes are not generally toxic except at high levels of exposure. Their breakdown products have low toxicity and are easily eliminated from the body.</p> <p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p>
<p>Ingestion</p>	<p>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.</p> <p>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).</p> <p>Large doses of cellulose may be administered orally as non-nutritive bulk. Doses of up to 30 g/day can be tolerated as bulk laxative. Extremely large oral doses may produce gastrointestinal disturbances.</p> <p>Acute toxic responses to aluminium are confined to the more soluble forms.</p> <p>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.</p> <p>Swallowing pieces of bitumen may produce pyloric obstruction due to accumulation in the stomach and the formation of a stony concretion.</p>

Fibred Aluminum Roof Coating

	<p>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.</p> <p>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.</p>
<p style="text-align: center;">Skin Contact</p>	<p>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.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.</p> <p>Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus.</p> <p>Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>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.</p> <p>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.</p>
<p style="text-align: center;">Eye</p>	<p>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.</p> <p>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.</p> <p>Workers exposed to fumes of blown bitumens developed keratoconjunctivitis.</p> <p>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</p> <p>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.</p>
<p style="text-align: center;">Chronic</p>	<p>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.</p> <p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>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.</p> <p>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.</p> <p>Substances that can cause 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</p> <p>Wherever it is reasonably practicable, exposure to substances that can cause 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.</p> <p>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.</p> <p>There is sufficient evidence to provide a strong presumption that human exposure to the material may produce heritable genetic damage.</p> <p>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</p> <ul style="list-style-type: none"> - appropriate animal studies, - other relevant information <p>Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.</p> <p>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.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminium oxide during abrasives production.</p> <p>Very fine Al₂O₃ powder was not fibrogenic in rats, guinea pigs, or hamsters when inhaled for 6 to 12 months and sacrificed at periods up to 12 months following the last exposure.</p> <p>When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C.</p> <p>The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis (aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity.</p> <p>Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction.</p> <p>There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious</p>

Fibred Aluminum Roof Coating

disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveoli (sub 5 µm) are able to produce pathogenic effects in the lungs.

A single 50-mg suspension of raw perlite or expanded perlite was administered intratracheally to albino rats. The animals were sacrificed after 9 months. Expanded perlite was found to produce more lung fibrosis than the raw ore. This was attributed to the cristobalite and tridymite content. In a study in which rats and guinea pigs inhaled perlite over a period of 18 months (average concentration 226 mg/m³), no significant pulmonary reaction, including fibrosis, was observed.

Chest X-ray evaluation of men in the perlite industry showed that workers with greater than 15 years service had a higher rate of abnormalities but this could not be attributed unambiguously to perlite exposure.

Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic.

Because aluminium competes with calcium for absorption, increased amounts of dietary aluminium may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies, aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseous tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory. These salts' estrogen-like effects have led to their classification as a metalloestrogen. Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.

After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus. Aluminium may persist for a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans.

At high levels of exposure, some aluminium compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminium compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminium potassium sulphate at high levels in the diet.

Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer's disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produce lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, testes, embryotoxicity, and the developing nervous system of 52, 75, 100, and 50 mg aluminium/kg bw/day, respectively. Similarly, the lowest no-observed-adverse-effect levels (NOAELs) for effects on these endpoints were reported at 30, 27, 100, and for effects on the developing nervous system, between 10 and 42 mg aluminium/kg bw per day, respectively.

Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 mg/l aluminium compared with communities where the aluminium level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminium exposure to brain disease. Aluminium concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramid-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations.

Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of "tau" a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminium hyperphosphorylates tau in vitro. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade. Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA).

Aluminium enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminium include baking powder, antacids and aluminium products used for general food preparation and storage (over 12 months, aluminium levels in soft drink packed in aluminium cans rose from 0.05 to 0.9 mg/l). [Walton, J and Bryson-Taylor, D. - *Chemistry in Australia*, August 1995] 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

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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 cellulose derivatives pass essentially unchanged through the gastrointestinal tract following oral administration to rats, dogs and man. Acute, subchronic, chronic toxicity, reproductive and developmental toxicity, genotoxicity and carcinogenicity studies of cellulose derivatives indicated that they are practically non-toxic when administered by oral, intraperitoneal, subcutaneous or dermal routes. While no clinical inhalation studies have been conducted, long term exposure to the dusts of cellulose ethers in manufacturing operations has not lead to any significant adverse effects. Ocular and dermal irritation studies indicate that the cellulose derivatives are, at most, minimally irritating and are not dermal sensitizers. Clinical studies confirm these results.

Amended Safety Assessment of Cellulose and Related Polymers as used in Cosmetics: Final Report of the Cosmetic Ingredient Review (CIR) Expert Panel: March 2009

Inhalation studies indicate that cellulose fibres may be fibrogenic; this finding continues to be the subject of extensive research. Cellulose is not considered an inert substance because :

- in rats, it causes granulomatous fibrosing alveolitis at the end of the third month after exposure,
- in rats there was an increase in the secretion of plasminogen activator and interleukin 1 as well as the release of lactate dehydrogenase from macrophages, in a manner similar to asbestos,
- there were increases in the incidence of obstructive lung diseases and bronchial asthma in humans at work and in the residential environment where exposure to cellulose was common,
- the substance may induce free radical production in human leucocytes.

Byssinosis is an occupational disease of the lungs caused by inhalation of cotton dust or dusts from other vegetable fibres such as flax, hemp, or sisal. Byssinosis is a chronic, asthma-like narrowing of the airways. Also called brown lung disease, byssinosis occurs almost exclusively in people who work with unprocessed cotton.

Cotton dust disease, "byssinosis", is well known among cotton mill workers. Cotton dust consists largely of cellulose fibre. Exposure to two components of the total dust, the "respirable" and "medium" fraction correlated significantly with the prevalence of respiratory symptoms.

Inhalation exposure to a concentration of 0.3 to 0.4 mg/m³ of "fly-free" dust results in a 20% occurrence of byssinosis. "Fly-free" dust is the sum of respirable and medium-length fibres. At 0.46 mg/m³, Grade II byssinosis occurs. A byssinosis (all grades) prevalence of 20%, at 0.3 mg/m³ occurs when the fibre length is less than 15 µm (aerodynamic equivalent diameter). Byssinosis is not caused by mechanical irritation but by reactions caused by pharmacologically active substances producing oedema or contraction of the smooth musculature of the airways. The causative agent is suspected to be an endotoxin, in turn, thought to be a cell wall component of bacteria found in cotton. Symptoms of byssinosis include chest tightness, wheezing and dyspnoea. Symptoms usually appear after an absence from work and may subside after 2-days of exposure. As the disease progresses, symptoms may persist for longer periods until they are constant. The individual may eventually exhibit chronic bronchitis and emphysema. Increased physical exertion may produce shortness of breath.

Studies indicate that diets containing large amounts of non-absorbable polysaccharides, such as cellulose, might decrease absorption of calcium, magnesium, zinc and phosphorus. Polysaccharides are polymeric carbohydrates that consist of monosaccharide units, which are connected together with glycosidic bonds. Due to the structural variation of different monosaccharides as well as the innumerable ways that these building blocks link with each other, polysaccharides can be considered as structurally complex biomacromolecules. Polysaccharides originating from plants (e.g., starch and guar gum), microbes (e.g., xanthan), algae (e.g., alginates and carrageenans) and animals (e.g., glycogen and chitin) are frequently used in food. Starch, a high molar mass compound consisting of (1->4)-linked alpha-D-glucopyranosyl units, is an important energy nutrient that is abundant in common foods, such as cereals and root crops. Although many other food polysaccharides are not digested in the upper gastrointestinal tract of humans, they often serve functions other than being components giving nutritional value. For example, plant cell-wall polysaccharides, such as arabinoxylans and beta-glucan, exist in cereal-based foods, and "plant gums" are used as thickeners, emulsifiers, emulsion stabilizers, gelling agents and encapsulating agents. These non-digestible polysaccharides are important for health because they are considered as dietary fibre, which promote colon health, regulate post-prandial blood glucose levels and reduce serum cholesterol levels.

Despite the fact that nature provides various sources of polysaccharides, and that scientific research on their exploitation as food materials is increasingly active, a relatively low number of polysaccharides are authorized for use as food ingredients. For example, in the European Union (EU) and in Switzerland, among the permitted food additives (identified by an E number) only a small percentage are polysaccharide-based (native or structurally modified). The difference between other food ingredients and food additives is mainly the quantity used in any given product. Food ingredients can be consumed alone as food (e.g., starch), whereas food additives (e.g., carboxymethyl cellulose) are used in small quantities (usually less than 2%) relative to the total food composition but they, nonetheless, play an important role in the food products. Regarding food additive use in Europe, the European Food Safety Authority (EFSA) has an expert Panel on Food Additives and Nutrient Sources Added to Food (ANS), which evaluates the safety of food additives. Similarly, if new ingredients are released into the market, EFSA's Panel on Dietetic Products, Nutrition and Allergies (NDA) has the responsibility of evaluating the safety of Novel Food ingredients.

The vast majority of polysaccharides used as food ingredients are plant-based. In addition to the cellulosic polysaccharides, other types of food-grade ingredients or additives, such as, vanillin aroma, glycerol esters of wood rosins (E445), xylitol (E967) and steryls/stanol, are derived from wood. The main components of wood are polysaccharides: cellulose (40–50 wt%) and hemicelluloses (20–35%), while lignin comprises 15–30% of wood mass.

The material contains a substantial proportion of a polymer considered to be of low concern (PLC). The trend towards production of lower molecular weight polymers (thus reducing the required level of solvent use and creating a more "environmentally-friendly" material) has brought with it the need to define PLCs as those

having molecular weights of between 1000 and 10000 and containing less than 10% of the molecules with molecular weight below 500 and less than 25% of the molecules with a molecular weight below 1000. These may contain unlimited low concern functional groups or moderate concern reactive functional groups with a combined functional group equivalent weight (FGEW, a concept developed by the US EPA describing whether the reactive functional group is sufficiently diluted by polymeric material) of a 1000 or more (provided no high concern groups are present) or high concern reactive functional groups with a FGEW of 5000 or more (FGEW includes moderate concern groups if present).

having molecular weights exceeding 10000 (without restriction on reactive groups). Inhalation of polymers with molecular weights > 70,000 Da has been linked with irreversible lung damage due to lung overloading and impaired clearance of particles from the lung, particularly following repeated exposure. If the polymer is inhaled at low levels and/or infrequently, it is assumed that it will be cleared from the lungs.

Reactive functional groups are in turn classified as being of low, moderate or high concern. Classification of the polymer as a PLC, in accordance with established criteria, does not mean that hazards will not be associated with the polymer (during its import, manufacture, use, storage, handling or disposal). The polymer may, for example, contain a large number of particles in the respirable range, a hazard which may need to be assessed in the health and safety risk assessment. Similarly a polymer with low concern reactive may be released into the environment in large quantities and produce an environmental hazard.

Whilst it is generally accepted that polymers with a molecular weight exceeding 1000 are unlikely to pass through biological membranes, oligomers with lower molecular weight and specifically, those with a molecular weight below 500, may. Estimations based on a "highly" dispersed polymer population (polydispersity = 10) suggests that the molecular weight of the polymer carrying a reactive group of high concern must be 5000 to be considered a PLC; similarly a polymer of approximate molecular weight 1000 could contain no more than one reactive group of moderate concern (for two moderate concern groups, the molecular weight would be about 2500).

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

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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 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- refined 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.

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.

Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]

Fibred Aluminum Roof Coating	TOXICITY	IRRITATION
	Not Available	Not Available
n-nonane	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation(Rat) LC50: 3200 ppm4h ^[2]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >5000 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

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white spirit	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >3000 mg/kg ^[1]	Eye (human): 470 ppm/15m
	Inhalation(Rat) LC50: >5.5 mg/4h ^[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]
aluminium powder coated	TOXICITY	IRRITATION
	Inhalation(Rat) LC50: >2.3 mg/4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50; >2000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
gilsonite	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]
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]	
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]
perlite	TOXICITY	IRRITATION
	Oral (Mouse) LD50; 12960 mg/kg ^[2]	Not Available
cellulose	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Not Available
	Inhalation(Rat) LC50: >5.8 mg/L4h ^[2]	
	Oral (Rat) LD50; >5000 mg/kg ^[2]	
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]	
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	

Fibred Aluminum Roof Coating	<p>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</p>
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Fibred Aluminum Roof Coating

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 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.

For aluminium compounds:

Aluminium present in food and drinking water is poorly absorbed through the gastrointestinal tract. The bioavailability of aluminium is dependent on the form in which it is ingested and the presence of dietary constituents with which the metal cation can complex. Ligands in food can have a marked effect on absorption of aluminium, as they can either enhance uptake by forming absorbable (usually water soluble) complexes (e.g., with carboxylic acids such as citric and lactic), or reduce it by forming insoluble compounds (e.g., with phosphate or dissolved silicate).

Considering the available human and animal data it is likely that the oral absorption of aluminium can vary 10-fold based on chemical form alone. Although bioavailability appears to generally parallel water solubility, insufficient data are available to directly extrapolate from solubility in water to bioavailability.

For oral intake from food, the European Food Safety Authority (EFSA) has derived a tolerable weekly intake (TWI) of 1 milligram (mg) of aluminium per kilogram of bodyweight. In its health assessment, the EFSA states a medium bioavailability of 0.1 % for all aluminium compounds which are ingested with food. This corresponds to a systemically available tolerable daily dose of 0.143 microgrammes (µg) per kilogramme (kg) of body weight. This means that for an adult weighing 60 kg, a systemically available dose of 8.6 µg per day is considered safe.

Based on a neuro-developmental toxicity study of aluminium citrate administered via drinking water to rats, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) established a Provisional Tolerable Weekly Intake (PTWI) of 2 mg/kg bw (expressed as aluminium) for all aluminium compounds in food, including food additives. The Committee on Toxicity of chemicals in food, consumer products and the environment (COT) considers that the derivation of this PTWI was sound and that it should be used in assessing potential risks from dietary exposure to aluminium.

The Federal Institute for Risk Assessment (BfR) of Germany has assessed the estimated aluminium absorption from antiperspirants. For this purpose, the data, derived from experimental studies, on dermal absorption of aluminium from antiperspirants for healthy and damaged skin was used as a basis. At about 10.5 µg, the calculated systemic intake values for healthy skin are above the 8.6 µg per day that are considered safe for an adult weighing 60 kg. If aluminium -containing antiperspirants are used on a daily basis, the tolerable weekly intake determined by the EFSA is therefore exceeded. The values for damaged skin, for example injuries from shaving, are many times higher. This means that in case of daily use of an aluminium-containing antiperspirant alone, the TWI may be completely exhausted. In addition, further aluminium absorption sources such as food, cooking utensils and other cosmetic products must be taken into account

Systemic toxicity after repeated exposure

No studies were located regarding dermal effects in animals following intermediate or chronic-duration dermal exposure to various forms of aluminium.

When orally administered to rats, aluminium compounds (including aluminium nitrate, aluminium sulfate and potassium aluminium sulfate) have produced various effects, including decreased gain in body weight and mild histopathological changes in the spleen, kidney and liver of rats (104 mg Al/kg bw/day) and dogs (88-93 mg Al/kg bw/day) during subchronic oral exposure. Effects on nerve cells, testes, bone and stomach have been reported at higher doses. Severity of effects increased with dose.

The main toxic effects of aluminium that have been observed in experimental animals are neurotoxicity and nephrotoxicity. Neurotoxicity has also been described in patients dialysed with water containing high concentrations of aluminium, but epidemiological data on possible adverse effects in humans at lower exposures are inconsistent

Reproductive and developmental toxicity:

Studies of reproductive toxicity in male mice (intraperitoneal or subcutaneous administration of aluminium nitrate or chloride) and rabbits (administration of aluminium chloride by gavage) have demonstrated the ability of aluminium to cause testicular toxicity, decreased sperm quality in mice and rabbits and reduced fertility in mice. No reproductive toxicity was seen in females given aluminium nitrate by gavage or dissolved in drinking water. Multi-generation reproductive studies in which aluminium sulfate and aluminium ammonium sulfate were administered to rats in drinking water, showed no evidence of reproductive toxicity

High doses of aluminium compounds given by gavage have induced signs of embryotoxicity in mice and rats in particular, reduced fetal body weight or pup weight at birth and delayed ossification. Developmental toxicity studies in which aluminium chloride was administered by gavage to pregnant rats showed evidence of foetotoxicity, but it was unclear whether the findings were secondary to maternal toxicity. A twelve-month neuro-development with aluminium citrate administered via the drinking water to Sprague-Dawley rats, was conducted according to Good Laboratory Practice (GLP). Aluminium citrate was selected for the study since it is the most soluble and bioavailable aluminium salt. Pregnant rats were exposed to aluminium citrate from gestational day 6 through lactation, and then the offspring were exposed post-weaning until postnatal day 364. An extensive functional observational battery of tests was performed at various times. Evidence of aluminium toxicity was demonstrated in the high (300 mg/kg bw/day of aluminium) and to a lesser extent, the mid-dose groups (100 mg/kg bw/day of aluminium). In the high-dose group, the main effect was renal damage, resulting in high mortality in the male offspring. No major neurological pathology or neurobehavioural effects were observed, other than in the neuromuscular subdomain (reduced grip strength and increased foot splay). Thus, the lowest observed adverse effect level (LOAEL) was 100 mg/kg bw/day and the no observed adverse effect level (NOAEL) was 30 mg/kg bw/day. Bioavailability of aluminium chloride, sulfate and nitrate and aluminium hydroxide was much lower than that of aluminium citrate. This study was used by JECFA as key study to derive the PTWI.

Genotoxicity

Aluminium compounds were non-mutagenic in bacterial and mammalian cell systems, but some produced DNA damage and effects on chromosome integrity and segregation in vitro. Clastogenic effects were also observed in vivo when aluminium sulfate was administered at high

Fibred Aluminum Roof Coating

doses by gavage or by the intraperitoneal route. Several indirect mechanisms have been proposed to explain the variety of genotoxic effects elicited by aluminium salts in experimental systems. Cross-linking of DNA with chromosomal proteins, interaction with microtubule assembly and mitotic spindle functioning, induction of oxidative damage, damage of lysosomal membranes with liberation of DNAase, have been suggested to explain the induction of structural chromosomal aberrations, sister chromatid exchanges, chromosome loss and formation of oxidized bases in experimental systems. The EFSA Panel noted that these indirect mechanisms of genotoxicity, occurring at relatively high levels of exposure, are unlikely to be of relevance for humans exposed to aluminium via the diet. Aluminium compounds do not cause gene mutations in either bacteria or mammalian cells. Exposure to aluminium compounds does result in both structural and numerical chromosome aberrations both in in-vitro and in-vivo mutagenicity tests. DNA damage is probably the result of indirect mechanisms. The DNA damage was observed only at high exposure levels.

Carcinogenicity.

The available epidemiological studies provide limited evidence that certain exposures in the aluminium production industry are carcinogenic to humans, giving rise to cancer of the lung and bladder. However, the aluminium exposure was confounded by exposure to other agents including polycyclic aromatic hydrocarbons, aromatic amines, nitro compounds and asbestos. There is no evidence of increased cancer risk in non-occupationally exposed persons.

Neurodegenerative diseases.

Following the observation that high levels of aluminium in dialysis fluid could cause a form of dementia in dialysis patients, a number of studies were carried out to determine if aluminium could cause dementia or cognitive impairment as a consequence of environmental exposure over long periods. Aluminium was identified, along with other elements, in the amyloid plaques that are one of the diagnostic lesions in the brain for Alzheimer disease, a common form of senile and pre-senile dementia. Some of the epidemiology studies suggest the possibility of an association of Alzheimer disease with aluminium in water, but other studies do not confirm this association. All studies lack information on ingestion of aluminium from food and how concentrations of aluminium in food affect the association between aluminium in water and Alzheimer disease."

There are suggestions that persons with some genetic variants may absorb more aluminium than others, but there is a need for more analytical research to determine whether aluminium from various sources has a significant causal association with Alzheimer disease and other neurodegenerative diseases. Aluminium is a neurotoxicant in experimental animals. However, most of the animal studies performed have several limitations and therefore cannot be used for quantitative risk assessment.

Contact sensitivity:

It has been suggested that the body burden of aluminium may be linked to different diseases. Macrophagic myofasciitis and chronic fatigue syndrome can be caused by aluminium-containing adjuvants in vaccines. Macrophagic myofasciitis (MMF) has been described as a disease in adults presenting with ascending myalgia and severe fatigue following exposure to aluminium hydroxide-containing vaccines. The corresponding histological findings include aluminium-containing macrophages infiltrating muscle tissue at the injection site. The hypothesis is that the long-lasting granuloma triggers the development of the systemic syndrome.

Aluminium acts not only as an adjuvant, stimulating the immune system either to fend off infections or to tolerate antigens, it also acts as a sensitiser causing contact allergy and allergic contact dermatitis. In general, metal allergies are very common and aluminium is considered to be a weak allergen. A metal must be ionised to be able to act as a contact allergen, then it has to undergo haptensation to be immunogenic and to initiate an immune response. Once inside the skin, the metal ions must bind to proteins to become immunologically reactive. The most important routes of exposure and sensitisation to aluminium are through aluminium-containing vaccines. One Swedish study showed a statistically significant association between contact allergy to aluminium and persistent itching nodules in children treated with allergen-specific immunotherapy (ASIT). Nodules were overrepresented in patients with contact allergy to aluminium.

Other routes of sensitisation reported in the literature are the prolonged use of aluminium-containing antiperspirants, topical medication, and tattooing of the skin with aluminium-containing pigments. Most of the patients experienced eczematous reactions whereas tattooing caused granulomas. Even though aluminium is used extensively in industry, only a low number of cases of occupational skin sensitisation to aluminium have been reported. Systemic allergic contact dermatitis in the form of flare-up reactions after re-exposure to aluminium has been documented: pruritic nodules at present and previous injection sites, eczema at the site of vaccination as well as at typically atopic localisations after vaccination with aluminium-containing vaccines and/or patch testing with aluminium, and also after use of aluminium-containing toothpaste.

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, xyleneol, 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 (REACH).

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).

Fibred Aluminum Roof Coating

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 discoloration 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 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 benzo[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 creosote 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,

Fibred Aluminum Roof Coating

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 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

N-NONANE

For alkanes:

Exposure to the commercial hexane (a representative of the ECHA group of hydrocarbons, C5-C7, n-alkanes, isoalkanes, n-hexane rich) had no effect on the behavior of rats. Rats were tested monthly throughout the exposure for hindlimb splay and grip strength. The NOAEC for sub-chronic neurological effects is 9000 ppm in rats.

In a 13 week subchronic inhalation study, the neurotoxicity of light alkylate naphtha distillate (LAND-2; carbon range C5-C8) was examined in male and female rats and aside from acute CNS effects, no treatment related neurotoxic effects found in any of the treatment groups. The NOAEC was determined to be > 24.3 g/m³ (6646 ppm). Additionally, no neurological effects were reported in the NTP 2 year carcinogenicity study on Stoddard solvent.

For hydrocarbons, C5-C7, n-alkanes, isoalkanes, n-hexane rich

n-Hexane was metabolized and excreted within 168 h of iv bolus administration, inhalation exposure or dermal application. Exhaled breath and urine were the two primary routes for the excretion and its metabolites. n-Hexane was widely distributed to the body tissues but were not concentrated significantly by any of those tissues. It was extensively metabolized and a number of radio labeled metabolites were excreted in the urine. n-Hexane and its radio labeled metabolites disappeared from the blood of rats with a half-life of approximately 9-10 h.

Repeated inhalation exposure had no apparent effect on the rates or routes of excretion of either of the test compounds or their metabolites. The absorption rates into the skin, normalised for exposure concentration, was determined to be 0.013 cm/h The maximum absorption rate into the blood was determined to be 0.005 nmol/h. A comparison of the estimated whole-body skin uptake with the inhalatory uptake from the same atmosphere, revealed that the dermal uptake contributed 0.1% to the total uptake

C9-C14 aliphatic, <2% aromatic hydrocarbon fluids are absorbed, they are typically metabolized by side chain oxidation to alcohol and carboxylic acid derivatives. These metabolites can be glucuronidated and excreted in the urine or further metabolized before being excreted. The majority of the metabolites are excreted in the urine and to a lower extent, in the faeces. Excretion is rapid with the majority of the elimination occurring within the first 24 hours of exposure. As a result of the lack of systemic toxicity and the ability of the parent material to undergo metabolism and rapid excretion, bioaccumulation of the test substance in the tissues is not likely to occur.

C9-C14 aliphatic, <2% aromatic hydrocarbon fluids are poorly absorbed dermally with an estimated overall percutaneous absorption rate of approximately 2ug/cm²/hr or 1% of the total applied fluid. Regardless of exposure route, C9-C14 aliphatic, <2% aromatic hydrocarbon fluids are rapidly metabolized and eliminated has been fully evaluated. All of the animal studies were performed in a manner similar or equivalent to currently established OECD guidelines. Based on these data, C9-C14 aliphatic, <2% aromatic hydrocarbons have a low order of acute toxicity by the oral, dermal, and inhalation routes of exposure.

In a study examining the oral toxicity of commercial hexane. 6 male rats were given doses of up to 25 ml/kg of test substance by oral gavage. The animals were then observed for 14 days for mortality. No mortality was observed at any of the doses. The oral LD50 is therefore > 25 ml/kg (16.75 g/kg; density of 0.67).

C9-C14 aliphatic, <2% aromatic hydrocarbons is minimally toxic via ingestion where the LD50 is >5000 mg/kg, via dermal exposure where the LD50 is >5000 mg/kg, and by inhalation where the LC50 > 5000 mg/m³. These findings do not warrant classification of C9-C14 aliphatic, <2% aromatic hydrocarbons under the Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP) do not warrant classification under the Directive 67/548/EEC for dangerous substances and Directive 1999/45/EC for preparations (DSD/DPD). C9-C14 aliphatic, <2% aromatic hydrocarbons are classified under EU CLP guidelines as a Category 1 aspiration hazard based on its physical and

Fibred Aluminum Roof Coating

chemical properties (hydrocarbon fluid, viscosity = 20.5 mm²/s) and as an R65 aspiration hazard under the EU DSD/DPD.

One study examined that acute inhalation toxicity of hexane to male rats. Groups of 10 male rats exposed to various large concentrations of hexane vapour for 4 hrs. Animals were then observed for clinical signs and mortality for at least the next 6 days. Several animals died during the exposure period. The LC50 was determined to be 73,680 ppm (259354 mg/m³). Due to the high concentration of the LC50, the test substance would not be classified as toxic by inhalation according to OECD GHS guidelines. Surviving animals experienced severe toxicological effects during the exposure.

Skin irritation:

For isoparaffinic, normal paraffinic, and mixed C9-C14 aliphatic, <2% aromatic hydrocarbon fluids, the weight of evidence indicates that the erythema and oedema scores (24, 48, and 72 average) are below the classification threshold requirements: 2.0, Directive 67/548/EEC for dangerous substances and Directive 1999/45/EC for preparation; 2.3, the new Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP).

For cycloparaffinic C9-C14 aliphatic, <2% aromatic hydrocarbon fluids, erythema and oedema scores (24, 48, and 72 average) are above the classification threshold requirements: 2.0, Directive 67/518/EEC for dangerous substances and Directive 1999/45/EC for preparation; 2.3, the new Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP). This finding warrants classification of the test material as a skin irritant (R38) under Directive 67/518/EEC for dangerous substances and Directive 1999/45/EC for preparations. This finding warrants classification of the test material as a Category 2 dermal irritant under the new Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP).

Eye irritation

Ocular lesion scores (24, 48, and 72 average) are below the classification threshold requirements.

Directive 67/548/EEC for dangerous substances and Directive 1999/45/EC for preparation: 0, cornea opacity; 0, iris lesion; >2.5, redness of the conjunctivae; >2.0, oedema of the conjunctivae (chemosis). Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP): 0, cornea opacity; 0, iris lesion; >2.0, redness of the conjunctivae; >2.0, oedema of the conjunctivae (chemosis).

Respiratory irritation

There are no studies that warrant classification as a respiratory irritant under either the Directive 67/518/EEC for dangerous substances and Directive 1999/45/EC or under the new Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP).

Sensitisation:

A study was performed to determine the concentration of hexane that would be expected to cause sensitization in humans.

Results of previous LLNA experiments were used to calculate the EC3 value, the concentration at which the test substance would produce a 3-fold increase in the proliferative activity of lymph nodes in the LLNA test. The 3-fold increase is considered a positive response for sensitization in the LLNA test. The EC3 value for hexane was determined to be > 100% concentration. The test substance is therefore not sensitizing.

There are no reports of respiratory sensitization from C9-C14 aliphatic, <2% aromatic hydrocarbons fluids in laboratory animals or humans.

However, skin sensitization studies utilizing C9-C14 aliphatic, <2% aromatic hydrocarbons fluids found no indication of skin sensitization in guinea pigs. Additional studies in humans also found no indication of skin sensitization. With these observations, it is presumed that C9-C14 aliphatic, <2% aromatic hydrocarbons fluids will not be a respiratory sensitizing agent.

Repeat dose toxicity,

In a study involving n-hexane, neurological effects were only seen at the highest dose level after an average of 101.3 days of exposure. The LOAEL for neurological effects is 46.2 mmol/kg bw (37973 mg/kg), and the NOAEL is 13.2 mmol/kg bw (1135 mg/kg). Reduced body weight gain was seen at all three dose levels, however was only considered treatment related in the 13.2 and 46.2 mmol/kg bw groups. The NOAEL is therefore 6.60 mmol/kg bw.

In a study involving n-hexane The NOAEC for male rats exposed via inhalation was 2984 ppm based on liver and kidney effects. The LOAEC for male rats was 8992 ppm. The NOAEC for female rats was 8992 ppm

C9-C14 aliphatic, <2% aromatic hydrocarbon fluids are expected to have a low order of repeated dose toxicity by the oral route of exposure. All tests were performed in a manner similar or equivalent to currently established OECD guidelines. In a repeated dose study where C9-C14 aliphatic, <2% aromatic hydrocarbon fluids were administered via oral gavage, no signs of toxicity were observed at the maximum experimental dose tested, 5000 mg/kg/day.

In a repeated dose study where C9-C14 aliphatic, <2% aromatic hydrocarbon fluids were administered via inhalation, no signs of toxicity were observed at 10400 mg/m³. Based on these observations, the repeat inhalation concentration NOAEL is =10400 mg/m³ (10.4 mg/L) for C9-C14 aliphatic, <2% aromatic hydrocarbon fluid

Genetic toxicity:

A study examined the in vitro mutagenicity of vapours of the test substance commercial hexane. Plates of *S. typhimurium* were exposed for 7-8 hrs to test atmospheres of 0, 600, 1000, 3000, 6000, or 9000 ppm of test substance. The test substance did not produce a positive response in any of the test strains. The test substance is not mutagenic.

In a study to determine the in vivo effect of inhalation exposure of commercial hexane on rat bone marrow. Groups of 5 male and 5 female rats were exposed to 0, 900, 3000, and 9000 ppm of test substance vapour for 6 hrs/day for 5 days. There was no statistically significant increase in cell aberrations in any treatment group. The test substance is not mutagenic.

C9-C14 aliphatic, <2% aromatic hydrocarbons fluids are not mutagenic using in vitro or in vivo genotoxicity assays. In bacterial tests, C9-C14 aliphatic, <2% aromatic hydrocarbons fluids were not mutagenic in *Salmonella* strains tested in the presence or absence of metabolic activation.

C9-C14 aliphatic, <2% aromatic hydrocarbon fluids were negative in a in vitro mammalian cell gene mutation assay. In sister chromatid exchange and in chromosomal aberration studies, C9-C14 aliphatic, <2% aromatic hydrocarbons fluids did not produce an effect. C9-C14 aliphatic, <2% aromatic hydrocarbons fluids were also non-mutagenic when tested in an in vivo mouse bone marrow micronucleus assay and when tested in dominant lethal studies utilizing an inhalation route of exposure. All studies were conducted in a manner similar or equivalent to currently established OECD guidelines.

C9-C14 aliphatic, <2% aromatic hydrocarbons fluids are a non-genotoxic agent and classification is not warranted under the Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP) or under the Directive 67/518/EEC for dangerous substances and Directive 1999/45/EC for preparations.

Toxicity to reproduction.

In a study examining the effects of commercial hexane the NOAEC for both male and female rats (adults and offspring) was 3000 ppm (10560 mg/m³). The LOAEC for these groups was 9000 ppm based on reduced body weight. There were no adverse effects to reproduction, therefore the NOAEC for reproduction is 9000 ppm (31680 mg/m³).

A study to examine the developmental toxicity of commercial hexane in mice, found the maternal NOAEC was 900 ppm, and the maternal LOAEC was 3000 ppm (10560 mg/m³) based on colour changes in the lungs. The developmental NOAEC was 3000 ppm and the LOAEC was 9000 ppm (31680 mg/m³) in mice.

C9-C14 aliphatic, <2% aromatic hydrocarbon fluids are not developmental toxicants. In two developmental studies (OECD TG 414), pregnant dams were dosed by inhalation with 0, 300, or 900 ppm C9-C14 aliphatic, <2% aromatic hydrocarbon fluids during gestational days 6 through 15. No adverse maternal or fetal effects were noted at any dose level. Thus, C9-C14 aliphatic, <2% aromatic hydrocarbon fluids did not produce any maternal or fetal toxicity or any developmental effects in rats. Based on the study results, the maternal and developmental toxicity NOAEC is >= 900 ppm (5220 mg/m³). Based on this study and the lack of systemic toxicity, C9-C14 aliphatic, <2% aromatic hydrocarbon fluids, are not expected to be developmental toxicants.

TRIMETHYLBENZENE (MIXED ISOMERS)	NOTE: This data is for mixed isomers of unstated proportions.
WHITE SPIRIT	white spirit, as CAS RN 8052-41-3
BITUMEN (PETROLEUM)	WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
1,2,4-TRIMETHYL BENZENE	CHEMWATCH 2325 1,3,5-trimethylbenzene
1,3,5-TRIMETHYL BENZENE	CHEMWATCH 12171 1,2,4-trimethylbenzene

Fibred Aluminum Roof Coating

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/m³ 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 NOEL 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 NOEL 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 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 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].

**NAPHTHA PETROLEUM,
LIGHT AROMATIC SOLVENT**

Fibred Aluminum Roof Coating

<p>Fibred Aluminum Roof Coating & N-NONANE & TRIMETHYLBENZENE (MIXED ISOMERS) & GILSONITE & BITUMEN (PETROLEUM) & 1,2,4-TRIMETHYL BENZENE & 1,3,5-TRIMETHYL BENZENE & PERLITE & CELLULOSE & NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT</p>	<p>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.</p>
<p>Fibred Aluminum Roof Coating & GILSONITE</p>	<p>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.</p>
<p>Fibred Aluminum Roof Coating & N-NONANE</p>	<p>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 than iso- or cyclo-paraffins.</p> <p>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.</p>
<p>Fibred Aluminum Roof Coating & TRIMETHYLBENZENE (MIXED ISOMERS) & 1,2,4-TRIMETHYL BENZENE & 1,3,5-TRIMETHYL BENZENE & NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT</p>	<p>For trimethylbenzenes:</p> <p>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-trimethylbenzene 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.</p> <p>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 trimethylbenzenes at 1700 ppm for 10 to 21 days.</p> <p>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.</p> <p>Results of the developmental toxicity study indicate that the C9 fraction caused adverse neurological effects at the highest dose (1500 ppm) tested.</p> <p>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.</p> <p>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.</p> <p>Genotoxicity: Results of mutagenicity testing, indicate that the C9 fraction does not induce gene mutations in prokaryotes (Salmonella typhimurium/mammalian microsome assay); or in mammalian cells in culture (in Chinese hamster ovary cells with and without activation). The C9 fraction 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.</p> <p>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 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 developmental neurotoxicity, was evident in rats in a 3-generation reproductive study.</p> <p>No effects on fecundity or fertility occurred in rats treated dermally with up to 0.3 mL/rat/day of a mixture of trimethylbenzenes, 4-6 hours/day, 5 days/week over one generation.</p>
<p>Fibred Aluminum Roof Coating & WHITE SPIRIT</p>	<p>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.</p> <p>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.</p> <p>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).</p> <p>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.</p> <p>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.</p>

Fibred Aluminum Roof Coating

	Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable.
TRIMETHYLBENZENE (MIXED ISOMERS) & 1,3,5-TRIMETHYLBENZENE	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.
ALUMINIUM POWDER COATED & GILSONITE & BITUMEN (PETROLEUM)	No significant acute toxicological data identified in literature search.
1,2,4-TRIMETHYL BENZENE & 1,3,5-TRIMETHYL BENZENE	Other Toxicity data is available for CHEMWATCH 12172 1,2,3-trimethylbenzene
Acute Toxicity	✗
Skin Irritation/Corrosion	✓
Serious Eye Damage/Irritation	✓
Respiratory or Skin sensitisation	✓
Mutagenicity	✓
Carcinogenicity	✓
Reproductivity	✗
STOT - Single Exposure	✓
STOT - Repeated Exposure	✓
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

	Endpoint	Test Duration (hr)	Species	Value	Source
Fibred Aluminum Roof Coating	Not Available	Not Available	Not Available	Not Available	Not Available
n-nonane	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	0.2mg/l	2
	NOEC(ECx)	504h	Crustacea	0.17mg/l	2
trimethylbenzene (mixed isomers)	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
aluminium powder coated	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	48h	Crustacea	>100mg/l	1
	EC50	72h	Algae or other aquatic plants	0.2mg/l	2
	EC50	48h	Crustacea	1.5mg/l	2
	LC50	96h	Fish	0.078-0.108mg/l	2
	EC50	96h	Algae or other aquatic plants	0.024mg/l	2
gilsonite	Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	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	Not Available
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

Continued...

Fibred Aluminum Roof Coating

	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
perlite	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
cellulose	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
Legend:	<i>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</i>				

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 Metal:

Atmospheric Fate - Metal-containing inorganic substances generally have negligible vapour pressure and are not expected to partition to air.

Environmental Fate: Environmental processes, such as oxidation, the presence of acids or bases and microbiological processes, may transform insoluble metals to more soluble ionic forms. Environmental processes may enhance bioavailability and may also be important in changing solubilities.

Aquatic/Terrestrial Fate: When released to dry soil, most metals will exhibit limited mobility and remain in the upper layer; some will leach locally into ground water and/ or surface water ecosystems when soaked by rain or melt ice. A metal ion is considered infinitely persistent because it cannot degrade further. Once released to surface waters and moist soils their fate depends on solubility and dissociation in water. A significant proportion of dissolved/ sorbed metals will end up in sediments through the settling of suspended particles. The remaining metal ions can then be taken up by aquatic organisms. Ionic species may bind to dissolved ligands or sorb to solid particles in water.

Ecotoxicity: Even though many metals show few toxic effects at physiological pH levels, transformation may introduce new or magnified effects.

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. Anthracene 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

Continued...

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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 $\log K_{ow} > -4.5$

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 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/L. 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, 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

Cellulosic products, including cellulose ethers, generally have a low biodegradation rate and are generally of low toxicity to fish.

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, $\log K_{ow}$ 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.

For aluminium and its compounds and salts:

Despite its prevalence in the environment, no known form of life uses aluminium salts metabolically. In keeping with its pervasiveness, aluminium is well tolerated by plants and animals. Owing to their prevalence, potential beneficial (or otherwise) biological roles of aluminium compounds are of continuing interest.

Environmental fate:

Aluminium occurs in the environment in the form of silicates, oxides and hydroxides, combined with other elements such as sodium, fluorine and arsenic complexes with organic matter.

Acidification of soils releases aluminium as a transportable solution. Mobilisation of aluminium by acid rain results in aluminium becoming available for plant uptake.

As an element, aluminium cannot be degraded in the environment, but may undergo various precipitation or ligand exchange reactions. Aluminium in compounds has only one oxidation state (+3), and would not undergo oxidation-reduction reactions under environmental conditions. Aluminium can be complexed by various ligands present in the environment (e.g., fulvic and humic acids). The solubility of aluminium in the environment will depend on the ligands present and the pH.

The trivalent aluminium ion is surrounded by six water molecules in solution. The hydrated aluminium ion, $[Al(H_2O)_6]^{3+}$, undergoes hydrolysis, in which a stepwise deprotonation of the coordinated water ligands forms bound hydroxide ligands (e.g., $[Al(H_2O)_5(OH)]^{2+}$, $[Al(H_2O)_4(OH)_2]^{+}$). The speciation of aluminium in water is pH dependent. The hydrated trivalent aluminium ion is the predominant form at pH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are $Al(OH)_2^{+}$ and $Al(OH)_2^{+}$, while the solid $Al(OH)_3$ is most prevalent between pH 5.2 and 8.8. The soluble species $Al(OH)_4^{-}$ is the predominant species above pH 9, and is the only species present above pH 10. Polymeric aluminium hydroxides appear between pH 4.7 and 10.5, and increase in size until they are transformed into colloidal particles of amorphous $Al(OH)_3$, which crystallise to gibbsite in acid waters. Polymerisation is affected by the presence of dissolved silica; when enough silica is present, aluminium is precipitated as poorly crystallised clay mineral species.

Hydroxyaluminum compounds are considered amphoteric (e.g., they can act as both acids and bases in solution). Because of this property, aluminium hydroxides can act as buffers and resist pH changes within the narrow pH range of 4-5.

Monomeric aluminium compounds, typified by aluminium fluoride, chloride, and sulfate, are considered reactive or labile compounds, whereas polymeric aluminium species react much more slowly in the environment. Aluminium has a stronger attraction for fluoride in an acidic environment compared to other inorganic ligand.

The adsorption of aluminium onto clay surfaces can be a significant factor in controlling aluminium mobility in the environment, and these adsorption reactions, measured in one study at pH 3.0-4.1, have been observed to be very rapid. However, clays may act either as a sink or a source for soluble aluminium depending on the degree of aluminium saturation on the

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clay surface.

Within the pH range of 5-6, aluminum complexes with phosphate and is removed from solution. Because phosphate is a necessary nutrient in ecological systems, this immobilization of both aluminum and phosphate may result in depleted nutrient states in surface water.

Plant species and cultivars of the same species differ considerably in their ability to take up and translocate aluminum to above-ground parts. Tea leaves may contain very high concentrations of aluminum, >5,000 mg/kg in old leaves. Other plants that may contain high levels of aluminum include Lycopodium (Lycopodiaceae), a few ferns, Symlocos (Symlocaceae), and Orites (Proteaceae). Aluminum is often taken up and concentrated in root tissue. In sub-alpine ecosystems, the large root biomass of the Douglas fir, *Abies amabilis*, takes up aluminum and immobilizes it, preventing large accumulation in above-ground tissue. It is unclear to what extent aluminum is taken up into root food crops and leafy vegetables. An uptake factor (concentration of aluminum in the plant/concentration of aluminum in soil) of 0.004 for leafy vegetables and 0.00065 for fruits and tubers has been reported, but the pH and plant species from which these uptake factors were derived are unclear. Based upon these values, however, it is clear that aluminum is not taken up in plants from soil, but is instead bioluted.

Aluminum concentrations in rainbow trout from an alum-treated lake, an untreated lake, and a hatchery were highest in gill tissue and lowest in muscle. Aluminum residue analyses in brook trout have shown that whole-body aluminum content decreases as the fish advance from larvae to juveniles. These results imply that the aging larvae begin to decrease their rate of aluminum uptake, to eliminate aluminum at a rate that exceeds uptake, or to maintain approximately the same amount of aluminum while the body mass increases. The decline in whole-body aluminum residues in juvenile brook trout may be related to growth and dilution by edible muscle tissue that accumulated less aluminum than did the other tissues.

The greatest fraction of the gill-associated aluminum was not sorbed to the gill tissue, but to the gill mucus. It is thought that mucus appears to retard aluminum transport from solution to the membrane surface, thus delaying the acute biological response of the fish. It has been reported that concentrations of aluminum in whole-body tissue of the Atlantic salmon exposed to high concentrations of aluminum ranging from 3 ug/g (for fish exposed to 33 ug/L) to 96 ug/g (for fish exposed to 264 ug/L) at pH 5.5. After 60 days of exposure, BCFs ranged from 76 to 190 and were directly related to the aluminum exposure concentration. In acidic waters (pH 4.6-5.3) with low concentrations of calcium (0.5-1.5 mg Ca/L), labile aluminum between 25 and 75 ug/L is toxic. Because aluminum is toxic to many aquatic species, it is not bioaccumulated to a significant degree (BCF <300) in most fish and shellfish; therefore, consumption of contaminated fish does not appear to be a significant source of aluminum exposure in humans.

Bioconcentration of aluminum has also been reported for several aquatic invertebrate species. BCF values ranging from 0.13 to 0.5 in the whole-body were reported for the snail.

Bioconcentration of aluminum has also been reported for aquatic insects.

Ecotoxicity:

Freshwater species pH >6.5

Fish: Acute LC50 (48-96 h) 5 spp: 0.6 (*Salmo salar*) - 106 mg/L; Chronic NOEC (8-28 d): 7 spp, NOEC, 0.034-7.1 mg/L. The lowest measured chronic figure was an 8-d LC50 of 0.17 mg/L for *Micropterus* sp.

Amphibian: Acute LC50 (4 d): *Bufo americanus*, 0.86-1.66 mg/L; Chronic LC50 (8-d) 2.28 mg/L

Crustaceans LC50 (48 h): 1 sp 2.3-36 9 mg/L; Chronic NOEC (7-28 d) 3 spp, 0.136-1.72 mg/L

Algae EC50 (96 h): population growth, 0.46-0.57 mg/L; 2 spp, chronic NOEC, 0.8-2.0 mg/L

Freshwater species pH <6.5 (all between pH 4.5 and 6.0)

Fish LC50 (24-96 h): 4 spp, 0.015 (*S. trutta*) - 4.2 mg/L; chronic data on *Salmo trutta*, LC50 (21-42 d) 0.015- 0.105 mg/L

Amphibians LC50 (4-5 d): 2 spp, 0.540-2.670 mg/L (absolute range 0.40-5.2 mg/L)

Alga: 1 sp NOEC growth 2.0 mg/L

Among freshwater aquatic plants, single-celled plants are generally the most sensitive to aluminum. Fish are generally more sensitive to aluminum than aquatic invertebrates.

Aluminum is a gill toxicant to fish, causing both ionoregulatory and respiratory effects.

The bioavailability and toxicity of aluminum is generally greatest in acid solutions. Aluminum in acid habitats has been observed to be toxic to fish and phytoplankton. Aluminum is generally more toxic over the pH range 4.4-5.4, with a maximum toxicity occurring around pH 5.0-5.2. The inorganic single unit aluminum species (Al(OH)₂⁺) is thought to be the most toxic. Under very acid conditions, the toxic effects of the high H⁺ concentration appear to be more important than the effects of low concentrations of aluminum; at approximately neutral pH values, the toxicity of aluminum is greatly reduced. The solubility of aluminum is also enhanced under alkaline conditions, due to its amphoteric character, and some researchers found that the acute toxicity of aluminum increased from pH 7 to pH 9. However, the opposite relationship was found in other studies. The uptake and toxicity of aluminum in freshwater organisms generally decreases with increasing water hardness under acidic, neutral and alkaline conditions. Complexing agents such as fluoride, citrate and humic substances reduce the availability of aluminum to organisms, resulting in lower toxicity. Silicon can also reduce aluminum toxicity to fish.

Drinking Water Standards:

aluminum: 200 ug/l (UK max.)

200 ug/l (WHO guideline)

chloride: 400 mg/l (UK max.)

250 mg/l (WHO guideline)

fluoride: 1.5 mg/l (UK max.)

1.5 mg/l (WHO guideline)

nitrate: 50 mg/l (UK max.)

50 mg/l (WHO guideline)

sulfate: 250 mg/l (UK max.)

Soil Guideline: none available.

Air Quality Standards: none available.

DO NOT discharge into sewer or waterways.

Persistence and degradability

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

Bioaccumulative potential

Ingredient	Bioaccumulation
n-nonane	HIGH (LogKOW = 4.7613)
1,2,4-trimethyl benzene	LOW (BCF = 275)
1,3,5-trimethyl benzene	LOW (BCF = 342)
cellulose	LOW (LogKOW = -5.1249)

Mobility in soil

Ingredient	Mobility
n-nonane	LOW (KOC = 934.6)
1,2,4-trimethyl benzene	LOW (KOC = 717.6)
1,3,5-trimethyl benzene	LOW (KOC = 703)
cellulose	LOW (KOC = 10)

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SECTION 13 Disposal considerations

Waste treatment methods

<p>Product / Packaging disposal</p>	<ul style="list-style-type: none"> ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▶ 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. <p>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.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) <p>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.</p> <ul style="list-style-type: none"> ▶ 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.
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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	

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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	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
n-nonane	Not Available
trimethylbenzene (mixed isomers)	Not Available
white spirit	Not Available
aluminium powder coated	Not Available
gilsonite	Not Available
bitumen (petroleum)	Not Available
1,2,4-trimethyl benzene	Not Available
1,3,5-trimethyl benzene	Not Available
perlite	Not Available
cellulose	Not Available
naphtha petroleum, light aromatic solvent	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
n-nonane	Not Available
trimethylbenzene (mixed isomers)	Not Available
white spirit	Not Available
aluminium powder coated	Not Available
gilsonite	Not Available
bitumen (petroleum)	Not Available
1,2,4-trimethyl benzene	Not Available
1,3,5-trimethyl benzene	Not Available
perlite	Not Available
cellulose	Not Available
naphtha petroleum, light aromatic solvent	Not Available

SECTION 15 Regulatory information

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

n-nonane is found on the following regulatory lists

US - Massachusetts - Right To Know Listed Chemicals
 US DOE Temporary Emergency Exposure Limits (TEELs)
 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 12(b) - List of Chemical Substances Subject to Export Notification Requirements
 US TSCA Section 4/12 (b) - Sunset Dates/Status

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

white spirit is found on the following regulatory lists

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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)

aluminium powder coated 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 - Massachusetts - Right To Know Listed Chemicals
 US ATSDR Minimal Risk Levels for Hazardous Substances (MRLs)
 US Department of Homeland Security (DHS) - Chemical Facility Anti-Terrorism Standards (CFATS) - Chemicals of Interest
 US EPCRA Section 313 Chemical List

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)

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)

1,2,4-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 EPCRA Section 313 Chemical List

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)

perlite 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 - Massachusetts - Right To Know Listed Chemicals
 US DOE Temporary Emergency Exposure Limits (TEELs)

cellulose 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 - Massachusetts - Right To Know Listed Chemicals
 US List of Active Substances Exempt from the TSCA Inventory Notifications (Active-Inactive) Rule
 US NIOSH Recommended Exposure Limits (RELs)

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 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

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

US OSHA Permissible Exposure Limits (PELs) Table Z-1
 US OSHA Permissible Exposure Limits (PELs) Table Z-3

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

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 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

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 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
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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

Continued...

Fibered Aluminum Roof Coating

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
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**

None Reported

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (n-nonane; trimethylbenzene (mixed isomers); white spirit; aluminium powder coated; gilsonite; bitumen (petroleum); 1,2,4-trimethyl benzene; 1,3,5-trimethyl benzene; perlite; naphtha petroleum, light aromatic solvent)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (gilsonite)
Japan - ENCS	No (aluminium powder coated; gilsonite; bitumen (petroleum); perlite)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	No (gilsonite; perlite)
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	No (perlite)
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	12/02/2022
Initial Date	12/03/2022

CONTACT POINT

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

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

Continued...

Fibered Aluminum Roof Coating

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
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index
AIIIC: 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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