

Carboguard 635HAR Part A

RESENE PAINTS AUSTRALIA

Chemwatch Hazard Alert Code: 4

Version No: 1.1
Safety Data Sheet according to WHS and ADG requirements

Issue Date: 14/01/2015
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S.GHS.AUS.EN

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

Product Identifier

Product name	Carboguard 635HAR Part A
Synonyms	Not Available
Proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
Other means of identification	Not Available

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

Relevant identified uses	Part% A of a two pack epoxy coating
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Details of the supplier of the safety data sheet

Registered company name	RESENE PAINTS AUSTRALIA
Address	7 Production Ave, Molendinar QLD 4214 Australia
Telephone	+61 7 55126600
Fax	+61 7 55126697
Website	Not Available
Email	Not Available

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	131126
Other emergency telephone numbers	Not Available

CHEMWATCH EMERGENCY RESPONSE

Primary Number	Alternative Number 1	Alternative Number 2
1800 039 008	1800 039 008	+612 9186 1132

Once connected and if the message is not in your preferred language then please dial 01


SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

Poisons Schedule	Not Applicable
Classification [1]	Chronic Aquatic Hazard Category 3, Flammable Liquid Category 3, Skin Corrosion/Irritation Category 2, Specific target organ toxicity - repeated exposure Category 2, Eye Irritation Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HSIS; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

Label elements

GHS label elements	
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SIGNAL WORD	WARNING
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Hazard statement(s)

H412	Harmful to aquatic life with long lasting effects.
H226	Flammable liquid and vapour.
H315	Causes skin irritation.

Continued...

H373	May cause damage to organs through prolonged or repeated exposure.
H319	Causes serious eye irritation.

Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P210	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
P233	Keep container tightly closed.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
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.
P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Precautionary statement(s) Response

P362	Take off contaminated clothing and wash before reuse.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam for extinction.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P314	Get medical advice/attention if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P303+P361+P353	IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower.
P332+P313	If skin irritation occurs: Get medical advice/attention.

Precautionary statement(s) Storage

P403+P235	Store in a well-ventilated place. Keep cool.
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Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.
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SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
21645-51-2	2.05	<u>aluminium hydroxide</u>
13463-67-7	18.24	<u>titanium dioxide</u>
7631-86-9	2.51	<u>silica amorphous</u>
14808-60-7	15.02	<u>silica crystalline - quartz</u>
1330-20-7	3.82	<u>xylene</u>
25036-25-3	11.1	<u>bisphenol A/ bisphenol A diglycidyl ether polymer</u>
7727-43-7	13.7	<u>barium sulfate</u>
25068-38-6	7.7	<u>bisphenol A/ diglycidyl ether resin, liquid</u>
14807-96-6	6.21	<u>talc</u>
108-10-1	3.5	<u>methyl isobutyl ketone</u>
64742-16-1	1.9	<u>hydrocarbon resin, postpolymerised with maleic anhydride</u>
39317-41-4	1.7	<u>vinyl chloride/ vinyl acetate/ hydroxypropyl acrylate</u>
71011-25-1	0.58	<u>tallow alkyldimethylammonium chloride/ bentonite complex</u>
108-67-8	0.06	<u>1,3,5-trimethyl benzene</u>
95-63-6	0.18	<u>1,2,4-trimethyl benzene</u>
526-73-8	0.06	<u>1,2,3-trimethyl benzene</u>
98-82-8	0.06	<u>isopropyl benzene - cumene</u>
103-65-1	0.06	<u>propylbenzene</u>
108-65-6	0.13	<u>propylene glycol monomethyl ether acetate, alpha-isomer</u>
64742-95-6	0.13	<u>naphtha petroleum, light aromatic solvent</u>
7664-38-2	0.02	<u>phosphoric acid</u>

112945-52-5	0.4	silica amorphous, fumed, crystalline free
51274-00-1	0.4	C.I. Pigment Yellow 42
71-36-3	0.2	n-butanol
1317-61-9	0.01	C.I. Pigment Black 11
1333-86-4	0.01	carbon black

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	<p>If this product comes in contact with eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with water. ▶ If irritation continues, 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. <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. ▶ 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.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

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 acute or short term repeated exposures to xylene:

- ▶ Gastro-intestinal absorption is significant with ingestions. For ingestions exceeding 1-2 ml (xylene)/kg, intubation and lavage with cuffed endotracheal tube is recommended. The use of charcoal and cathartics is equivocal.
- ▶ Pulmonary absorption is rapid with about 60-65% retained at rest.
- ▶ Primary threat to life from ingestion and/or inhalation, is respiratory failure.
- ▶ Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO₂ < 50 mm Hg or pCO₂ > 50 mm Hg) should be intubated.
- ▶ Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- ▶ A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- ▶ Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g.

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- Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.

BIOLOGICAL EXPOSURE INDEX - BEI

These represent the determinants observed in specimens collected from a healthy worker exposed at the Exposure Standard (ES or TLV):

Determinant	Index	Sampling Time	Comments
Methylhippu-ric acids in urine	1.5 gm/gm creatinine 2 mg/min	End of shift Last 4 hrs of shift	

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

Special hazards arising from the substrate or mixture

Fire Incompatibility	<ul style="list-style-type: none"> Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> When silica dust is dispersed in air, firefighters should wear inhalation protection as hazardous substances from the fire may be adsorbed on the silica particles. When heated to extreme temperatures, (>1700 deg.C) amorphous silica can fuse.
Fire/Explosion Hazard	<ul style="list-style-type: none"> Liquid and vapour are flammable. Moderate fire hazard when exposed to heat or flame. Vapour forms an explosive mixture with air. Moderate explosion hazard when exposed to heat or flame. Vapour may travel a considerable distance to source of ignition. Heating may cause expansion or decomposition leading to violent rupture of containers. On combustion, may emit toxic fumes of carbon monoxide (CO). <p>Combustion products include:</p> <ul style="list-style-type: none"> carbon dioxide (CO₂) carbon monoxide (CO) aldehydes sulfur oxides (SO_x) silicon dioxide (SiO₂) other pyrolysis products typical of burning organic material.
HAZCHEM	<ul style="list-style-type: none"> 3Y

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	#

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<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. DO NOT allow clothing wet with material to stay in contact with skin Electrostatic discharge may be generated during pumping - this may result in fire. Ensure electrical continuity by bonding and grounding (earthing) all equipment. Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (<=1 m/sec until fill pipe submerged to twice its diameter, then <= 7 m/sec). Avoid splash filling. Do NOT use compressed air for filling discharging or handling operations. 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.
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	<ul style="list-style-type: none"> ▶ 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.
Other information	<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

Suitable container	<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.
Storage incompatibility	<p>Barium sulfate (barytes)</p> <ul style="list-style-type: none"> ▶ reacts violently with dimethyl sulfoxide, sodium acetylide, finely divided carbon, aluminium, magnesium, zirconium, and possibly other active metals, especially at elevated temperatures ▶ is incompatible with potassium, phosphorus (ignites when primed with nitrate-calcium silicide) <p>Xylenes:</p> <ul style="list-style-type: none"> ▶ may ignite or explode in contact with strong oxidisers, 1,3-dichloro-5,5-dimethylhydantoin, uranium fluoride ▶ attack some plastics, rubber and coatings ▶ may generate electrostatic charges on flow or agitation due to low conductivity. ▶ Vigorous reactions, sometimes amounting to explosions, can result from the contact between aromatic rings and strong oxidising agents. ▶ Aromatics can react exothermally with bases and with diazo compounds. <p>Silicas:</p> <ul style="list-style-type: none"> ▶ react with hydrofluoric acid to produce silicon tetrafluoride gas ▶ react with xenon hexafluoride to produce explosive xenon trioxide ▶ reacts exothermally with oxygen difluoride, and explosively with chlorine trifluoride (these halogenated materials are not commonplace industrial materials) and other fluorine-containing compounds ▶ may react with fluorine, chlorates ▶ are incompatible with strong oxidisers, manganese trioxide, chlorine trioxide, strong alkalis, metal oxides, concentrated orthophosphoric acid, vinyl acetate ▶ may react vigorously when heated with alkali carbonates. <p>Titanium dioxide</p> <ul style="list-style-type: none"> ▶ reacts with strong acids, strong oxidisers ▶ reacts violently with aluminium, calcium, hydrazine, lithium (at around 200 deg C.), magnesium, potassium, sodium, zinc, especially at elevated temperatures - these reactions involves reduction of the oxide and are accompanied by incandescence ▶ dust or powders can ignite and then explode in a carbon dioxide atmosphere <p>For alkyl aromatics:</p> <p>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>

Glycidyl ethers:

- ▶ may form unstable peroxides on storage in air, light, sunlight, UV light or other ionising radiation, trace metals - inhibitor should be maintained at adequate levels
- ▶ may polymerise in contact with heat, organic and inorganic free radical producing initiators
- ▶ may polymerise with evolution of heat in contact with oxidisers, strong acids, bases and amines
- ▶ react violently with strong oxidisers, permanganates, peroxides, acyl halides, alkalis, ammonium persulfate, bromine dioxide
- ▶ attack some forms of plastics, coatings, and rubber



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- X** — Must not be stored together
O — May be stored together with specific precautions
+ — May be stored together

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	aluminium hydroxide	Fume (thermally generated) (respirable dust)	2 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	titanium dioxide	Titanium dioxide	10 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Precipitated silica / Silica - Amorphous: Silica gel / Precipitated silica / Silica gel	10 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Diatomaceous earth (uncalcined) / Diatomaceous earth (uncalcined)	10 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Fume (thermally generated) (respirable dust)	2 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Fumed silica (respirable dust) / Fumed silica (respirable dust)	2 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	silica crystalline - quartz	Silica - Crystalline: Quartz (respirable dust) / Quartz (respirable dust)	0.1 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	xylene	Xylene (o-, m-, p- isomers)	350 mg/m ³ / 80 ppm	655 mg/m ³ / 150 ppm	Not Available	Not Available
Australia Exposure Standards	barium sulfate	Barium sulphate	10 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	talc	Soapstone (respirable dust) / Talc, (containing no asbestos fibres)	3 mg/m ³ / 2.5 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	methyl isobutyl ketone	Methyl isobutyl ketone	205 mg/m ³ / 50 ppm	307 mg/m ³ / 75 ppm	Not Available	Not Available
Australia Exposure Standards	isopropyl benzene - cumene	Cumene	125 mg/m ³ / 25 ppm	375 mg/m ³ / 75 ppm	Not Available	Sk
Australia Exposure Standards	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxy-2-propanol acetate	274 mg/m ³ / 50 ppm	548 mg/m ³ / 100 ppm	Not Available	Sk
Australia Exposure Standards	phosphoric acid	Phosphoric acid	1 mg/m ³	3 mg/m ³	Not Available	Not Available
Australia Exposure Standards	n-butanol	n-Butyl alcohol	Not Available	Not Available	152 mg/m ³ / 50 ppm	Sk
Australia Exposure Standards	carbon black	Carbon black	3 mg/m ³	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
aluminium hydroxide	Aluminum hydroxide	8.7 mg/m ³	73 mg/m ³	440 mg/m ³
titanium dioxide	Titanium oxide; (Titanium dioxide)	30 mg/m ³	330 mg/m ³	2,000 mg/m ³
silica amorphous	Silica gel, amorphous synthetic	18 mg/m ³	200 mg/m ³	1,200 mg/m ³
silica amorphous	Silica, amorphous fumed	18 mg/m ³	100 mg/m ³	630 mg/m ³
silica amorphous	Siloxanes and silicones, dimethyl, reaction products with silica; (Hydrophobic silicon dioxide, amorphous)	120 mg/m ³	1,300 mg/m ³	7,900 mg/m ³
silica amorphous	Silica, amorphous fume	45 mg/m ³	500 mg/m ³	3,000 mg/m ³
silica amorphous	Silica amorphous hydrated	18 mg/m ³	220 mg/m ³	1,300 mg/m ³
silica crystalline - quartz	Silica, crystalline-quartz; (Silicon dioxide)	0.075 mg/m ³	33 mg/m ³	200 mg/m ³
xylene	Xylenes	Not Available	Not Available	Not Available
bisphenol A/ bisphenol A diglycidyl ether polymer	Epoxy resin; (Bisphenol A-Bisphenol A diglycidyl ether polymer)	6 mg/m ³	66 mg/m ³	400 mg/m ³

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barium sulfate	Barium sulfate	15 mg/m3	170 mg/m3	990 mg/m3
bisphenol A/ diglycidyl ether resin, liquid	Epoxy resin includes EPON 1001, 1007, 820, ERL-2795	90 mg/m3	990 mg/m3	5,900 mg/m3
talc	Talc	6 mg/m3	66 mg/m3	400 mg/m3
methyl isobutyl ketone	Methyl isobutyl ketone; (Hexone)	75 ppm	500 ppm	3000 ppm
1,3,5-trimethyl benzene	Mesitylene; (1,3,5-Trimethylbenzene)	Not Available	Not Available	480 ppm
1,2,4-trimethyl benzene	Permafluor E+	140 mg/m3	360 mg/m3	2,200 mg/m3
1,2,4-trimethyl benzene	Trimethylbenzene, 1,2,4-; (Pseudocumene)	Not Available	Not Available	480 ppm
1,2,3-trimethyl benzene	Trimethylbenzene, 1,2,3-	Not Available	Not Available	480 ppm
isopropyl benzene - cumene	Cumene; (Isopropyl benzene)	Not Available	Not Available	Not Available
propylbenzene	Propylbenzene, n-; (Isocumene)	3 ppm	33 ppm	2300 ppm
propylene glycol monomethyl ether acetate, alpha-isomer	Propylene glycol monomethyl ether acetate, alpha-isomer; (1-Methoxypropyl-2-acetate)	Not Available	Not Available	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Propylene glycol monomethyl ether acetate, beta-isomer; (2-Methoxypropyl-1-acetate)	Not Available	Not Available	Not Available
phosphoric acid	Phosphoric acid	Not Available	Not Available	Not Available
silica amorphous, fumed, crystalline free	Silica, amorphous fumed	18 mg/m3	100 mg/m3	630 mg/m3
n-butanol	Butyl alcohol, n-; (n-Butanol)	60 ppm	800 ppm	8000 ppm
C.I. Pigment Black 11	Iron(II,III) oxide; (Ferrosferric oxide)	21 mg/m3	230 mg/m3	1,400 mg/m3
carbon black	Carbon black	9 mg/m3	99 mg/m3	590 mg/m3

Ingredient	Original IDLH	Revised IDLH
aluminium hydroxide	Not Available	Not Available
titanium dioxide	N.E. mg/m3 / N.E. ppm	5,000 mg/m3
silica amorphous	N.E. mg/m3 / N.E. ppm	3,000 mg/m3
silica crystalline - quartz	N.E. mg/m3 / N.E. ppm	50 mg/m3
xylene	1,000 ppm	900 ppm
bisphenol A/ bisphenol A diglycidyl ether polymer	Not Available	Not Available
barium sulfate	Not Available	Not Available
bisphenol A/ diglycidyl ether resin, liquid	Not Available	Not Available
talc	N.E. mg/m3 / N.E. ppm	1,000 mg/m3
methyl isobutyl ketone	3,000 ppm	500 ppm
hydrocarbon resin, postpolymerised with maleic anhydride	Not Available	Not Available
vinyl chloride/ vinyl acetate/ hydroxypropyl acrylate	Not Available	Not Available
tallow alkyldimethylammonium chloride/ bentonite complex	Not Available	Not Available
1,3,5-trimethyl benzene	Not Available	Not Available
1,2,4-trimethyl benzene	Not Available	Not Available
1,2,3-trimethyl benzene	Not Available	Not Available
isopropyl benzene - cumene	8,000 ppm	900 [LEL] ppm
propylbenzene	Not Available	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available
naphtha petroleum, light aromatic solvent	Not Available	Not Available
phosphoric acid	10,000 mg/m3	1,000 mg/m3
silica amorphous, fumed, crystalline free	N.E. mg/m3 / N.E. ppm	3,000 mg/m3
C.I. Pigment Yellow 42	Not Available	Not Available
n-butanol	8,000 ppm	1,400 [LEL] ppm
C.I. Pigment Black 11	Not Available	Not Available
carbon black	N.E. mg/m3 / N.E. ppm	1,750 mg/m3

Exposure controls

Appropriate engineering controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are:

- Process controls which involve changing the way a job activity or process is done to reduce the risk.
- Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and

Continued...

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"removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.

For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant.

Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Personal protection



Eye and face protection

- ▶ Safety glasses with side shields.
- ▶ Chemical goggles.
- ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection

See Hand protection below

Hands/feet protection

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.

The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.

Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturizer is recommended.

Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

- frequency and duration of contact,
- chemical resistance of glove material,
- glove thickness and
- dexterity

Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).

- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
- Contaminated gloves should be replaced.

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

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.

- ▶ When handling liquid-grade epoxy resins wear chemically protective gloves (e.g nitrile or nitrile-butadiene rubber), boots and aprons.
- ▶ **DO NOT use cotton or leather (which absorb and concentrate the resin), polyvinyl chloride, rubber or polyethylene gloves (which absorb the resin).**
- ▶ **DO NOT use barrier creams containing emulsified fats and oils as these may absorb the resin; silicone-based barrier creams should be reviewed prior to**

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	▶ use.
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ PVC Apron. ▶ PVC protective suit may be required if exposure severe. ▶ Eyewash unit. ▶ Ensure there is ready access to a safety shower. <ul style="list-style-type: none"> • Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity. • For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets). • Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.
Thermal hazards	Not Available

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:

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Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE	C
PE/EVAL/PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
SARANEX-23	C
TEFLON	C
VITON	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

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	BAX-AUS P2	-	BAX-PAPR-AUS / Class 1 P2
up to 50 x ES	-	BAX-AUS / Class 1 P2	-
up to 100 x ES	-	BAX-2 P2	BAX-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)

If inhalation risk above the TLV exists, wear approved dust respirator.

Use respirators with protection factors appropriate for the exposure level.

▶ Up to 5 X TLV, use valveless mask type; up to 10 X TLV, use 1/2 mask dust respirator

▶ Up to 50 X TLV, use full face dust respirator or demand type C air supplied respirator

▶ Up to 500 X TLV, use powered air-purifying dust respirator or a Type C pressure demand supplied-air respirator

▶ Over 500 X TLV wear full-face self-contained breathing apparatus with positive pressure mode or a combination respirator with a Type C positive pressure supplied-air full-face respirator and an auxiliary self-contained breathing apparatus operated in pressure demand or other positive pressure mode

Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content. The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	coloured viscous liquid		
Physical state	Liquid	Relative density (Water = 1)	1.78
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	482
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	1544.94
Initial boiling point and boiling range (°C)	133	Molecular weight (g/mol)	Not Available

Continued...

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Flash point (°C)	25	Taste	Not Available
Evaporation rate	0.9 BuAC = 1	Explosive properties	Not Available
Flammability	Flammable.	Oxidising properties	Not Available
Upper Explosive Limit (%)	7.8	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	1.1	Volatile Component (%vol)	16.5
Vapour pressure (kPa)	0.6	Gas group	Not Available
Solubility in water (g/L)	Immiscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	3.65	VOC g/L	Not Available

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.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	<p>The material can cause respiratory irritation in some persons. The body's response to such irritation can cause further lung damage. The acute toxicity of inhaled alkylbenzenes is best described by central nervous system depression. As a rule, these compounds may also act as general anaesthetics.</p> <p>Systemic poisoning produced by general anaesthesia is characterised by lightheadedness, nervousness, apprehension, euphoria, confusion, dizziness, drowsiness, tinnitus, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness and respiratory depression and arrest. Cardiac arrest may result from cardiovascular collapse. Bradycardia, and hypotension may also be produced.</p> <p>Inhaled alkylbenzene vapours cause death in animals at air levels that are relatively similar (typically LC50s are in the range 5000 -8000 ppm for 4 to 8 hour exposures). It is likely that acute inhalation exposure to alkylbenzenes resembles that to general anaesthetics.</p> <p>Alkylbenzenes are not generally toxic other than at high levels of exposure. This may be because their metabolites have a low order of toxicity and are easily excreted. There is little or no evidence to suggest that metabolic pathways can become saturated leading to spillover to alternate pathways. Nor is there evidence that toxic reactive intermediates, which may produce subsequent toxic or mutagenic effects, are formed</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.</p> <p>On exposure to mixed trimethylbenzenes, some people may become nervous, tensed, anxious and have difficult breathing. There may be a reduction red blood cells and bleeding abnormalities. There may also be drowsiness.</p> <p>Headache, fatigue, tiredness, irritability and digestive disturbances (nausea, loss of appetite and bloating) are the most common symptoms of xylene overexposure. Injury to the heart, liver, kidneys and nervous system has also been noted amongst workers.</p> <p>Xylene is a central nervous system depressant</p>		
Ingestion	<p>Ingestion of soluble barium compounds may result in ulceration of the mucous membranes of the gastrointestinal tract, tightness in the muscles of the face and neck, gastroenteritis, vomiting, diarrhoea, muscular tremors and paralysis, anxiety, weakness, laboured breathing, cardiac irregularity due to contractions of smooth striated and cardiac muscles (often violent and painful), slow irregular pulse, hypertension, convulsions and respiratory failure.</p> <p>Swallowing of the liquid may cause aspiration into the lungs with the risk of chemical pneumonitis; serious consequences may result. (ICSC13733)</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.</p>		
Skin Contact	<p>This material can cause inflammation of the skin on contact in some persons.</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>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 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>		
Eye	<p>Although the liquid is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).</p>		
Chronic	<p>Studies show that inhaling this substance for over a long period (e.g. in an occupational setting) may increase the risk of cancer.</p> <p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>Harmful: danger of serious damage to health by prolonged exposure through inhalation.</p> <p>This material can cause serious damage if one is exposed to it for long periods. It can be assumed that it contains a substance which can produce severe defects.</p> <p>Substance accumulation, in the human body, may occur and may cause some concern following repeated or long-term occupational exposure.</p> <p>There is some evidence that inhaling this product is more likely to cause a sensitisation reaction in some persons compared to the general population.</p> <p>In animal testing, a liquid bisphenol A diglycidyl ether resin caused severe skin irritation when applied daily for 4 hours over 20 days. Animal testing suggests this group of substances may also cause tumours of the skin, kidney, blood and smooth muscle.</p> <p>Crystalline silicas activate the inflammatory response of white blood cells after they injure the lung epithelium. Chronic exposure to crystalline silicas reduces lung capacity and predisposes to chest infections.</p> <p>Glycidyl ethers can cause genetic damage and cancer.</p> <p>There has been concern that this material can cause cancer or mutations, but there is not enough data to make an assessment.</p> <p>Women exposed to xylene in the first 3 months of pregnancy showed a slightly increased risk of miscarriage and birth defects. Evaluation of workers chronically exposed to xylene has demonstrated lack of genetic toxicity.</p>		
Carboguard 635HAR Part A	<table border="0"> <tr> <td style="text-align: center;">TOXICITY</td> <td style="text-align: center;">IRRITATION</td> </tr> </table>	TOXICITY	IRRITATION
TOXICITY	IRRITATION		

	Not Available	Not Available
aluminium hydroxide	TOXICITY	IRRITATION
	Oral (rat) LD50: >2000 mg/kg ^[1]	Not Available
titanium dioxide	TOXICITY	IRRITATION
	Inhalation (rat) LC50: >2.28 mg/l/4hr ^[1]	Skin (human): 0.3 mg /3D (int)-mild *
	Inhalation (rat) LC50: >3.56 mg/l/4hr ^[1]	
	Inhalation (rat) LC50: >6.82 mg/l/4hr ^[1]	
	Inhalation (rat) LC50: 3.43 mg/l/4hr ^[1]	
	Inhalation (rat) LC50: 5.09 mg/l/4hr ^[1]	
silica amorphous	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[1]	Eye (rabbit): non-irritating *
	Inhalation (rat) LC50: >0.139 mg/l/14hr ^{+[2]}	Skin (rabbit): non-irritating *
silica crystalline - quartz	TOXICITY	IRRITATION
	Not Available	Not Available
xylene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1700 mg/kg ^[2]	Eye (human): 200 ppm irritant
	Inhalation (rat) LC50: 5000 ppm/4hr ^[2]	Eye (rabbit): 5 mg/24h SEVERE
	Oral (rat) LD50: 4300 mg/kg ^[2]	Eye (rabbit): 87 mg mild Skin (rabbit):500 mg/24h moderate
bisphenol A/ bisphenol A diglycidyl ether polymer	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[2]	Not Available
barium sulfate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Not Available
bisphenol A/ diglycidyl ether resin, liquid	TOXICITY	IRRITATION
	Oral (rat) LD50: 13447 mg/kg ^[1]	Eye (rabbit): 100mg - Mild
talc	TOXICITY	IRRITATION
	Not Available	Skin (human): 0.3 mg/3d-I mild
methyl isobutyl ketone	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >16000 mg/kg ^[1]	Eye (human): 200 ppm/15m
	Oral (rat) LD50: 2984 mg/kg ^[1]	Eye (rabbit): 40 mg - SEVERE Eye (rabbit): 500 mg/24h - mild Skin (rabbit): 500 mg/24h - mild
hydrocarbon resin, postpolymerised with maleic anhydride	TOXICITY	IRRITATION
	Not Available	Not Available
vinyl chloride/ vinyl acetate/ hydroxypropyl acrylate	TOXICITY	IRRITATION
	Not Available	Not Available
tallow alkyldimethylammonium chloride/ bentonite complex	TOXICITY	IRRITATION
	Not Available	Not Available
1,3,5-trimethyl benzene	TOXICITY	IRRITATION
	dermal (rat) LD50: >3460 mg/kg ^[1]	Eye (rabbit): 500 mg/24h mild

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	Inhalation (rat) LC50: 24 mg/L/4hr ^[2]	Skin (rabbit): 20 mg/24h moderate
	Oral (rat) LD50: ca.3460 mg/kg ^[1]	
1,2,4-trimethyl benzene	TOXICITY	IRRITATION
	dermal (rat) LD50: 3504 mg/kg ^[1]	Not Available
	Inhalation (rat) LC50: 18 mg/L/4hr ^[2]	
	Oral (rat) LD50: ca.3504 mg/kg ^[1]	
1,2,3-trimethyl benzene	TOXICITY	IRRITATION
	Not Available	Not Available
isopropyl benzene - cumene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 10602.6 mg/kg ^[1]	Eye (rabbit): 500 mg/24h mild
	Inhalation (rat) LC50: 39 mg/L/4hr ^[2]	Eye (rabbit): 86 mg mild
	Oral (rat) LD50: 1400 mg/kg ^[2]	Skin (rabbit): 10 mg/24h mild
		Skin (rabbit):100 mg/24h moderate
propylbenzene	TOXICITY	IRRITATION
	Inhalation (rat) LC50: 65000 ppm/2hr ^[2]	Not Available
	Oral (rat) LD50: 6040 mg/kg ^[2]	
propylene glycol monomethyl ether acetate, alpha-isomer	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Not Available
	Inhalation (rat) LC50: 4345 ppm/6hr ^[2]	
	Oral (rat) LD50: >14.1 ml ^[1]	
naphtha petroleum, light aromatic solvent	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1900 mg/kg ^[1]	Not Available
	Inhalation (rat) LC50: >3670 ppm/8 h ^[2]	
	Oral (rat) LD50: >4500 mg/kg ^[1]	
phosphoric acid	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1260 mg/kg ^[2]	Eye (rabbit): 119 mg - SEVERE
	Inhalation (rat) LC50: 0.0255 mg/L/4hr ^[2]	Skin (rabbit):595 mg/24h - SEVERE
	Oral (rat) LD50: 1.7 ml ^[1]	
silica amorphous, fumed, crystalline free	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	Not Available
	Oral (rat) LD50: 3160 mg/kg ^[2]	
C.I. Pigment Yellow 42	TOXICITY	IRRITATION
	Oral (rat) LD50: >5000 mg/kg ^[2]	Not Available
n-butanol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 3434.4 mg/kg ^[1]	Eye (human): 50 ppm - irritant
	Inhalation (rat) LC50: 24 mg/L/4hr ^[2]	Eye (rabbit): 1.6 mg-SEVERE
	Inhalation (rat) LC50: 8000 ppm/4hr ^[2]	Eye (rabbit): 24 mg/24h-SEVERE
	Oral (rat) LD50: 2292.3 mg/kg ^[1]	Skin (rabbit): 405 mg/24h-moderate
C.I. Pigment Black 11	TOXICITY	IRRITATION
	Oral (rat) LD50: >2000 mg/kg ^[1]	Not Available
carbon black	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >3000 mg/kg ^[2]	Not Available
	Oral (rat) LD50: >8000 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

Carboguard 635HAR Part A	<p>Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit many common characteristics with respect to animal toxicology. One such oxirane is ethyloxirane; data presented here may be taken as representative.</p> <p>for 1,2-butylene oxide (ethyloxirane):</p> <p>Ethyloxirane increased the incidence of tumours of the respiratory system in male and female rats exposed via inhalation. Significant increases in nasal papillary adenomas and combined alveolar/bronchiolar adenomas and carcinomas were observed in male rats exposed to 1200 mg/m³ ethyloxirane via inhalation for 103 weeks. There was also a significant positive trend in the incidence of combined alveolar/bronchiolar adenomas and carcinomas. Nasal papillary adenomas were also observed in 2/50 high-dose female rats with none occurring in control or low-dose animals. In mice exposed chronically via inhalation, one male mouse developed a squamous cell papilloma in the nasal cavity (300 mg/m³) but other tumours were not observed. Tumours were not observed in mice exposed chronically via dermal exposure. When trichloroethylene containing 0.8% ethyloxirane was administered orally to mice for up to 35 weeks, followed by 0.4% from weeks 40 to 69, squamous-cell carcinomas of the forestomach occurred in 3/49 males (p=0.029, age-adjusted) and 1/48 females at week 106. Trichloroethylene administered alone did not induce these tumours and they were not observed in control animals. Two structurally related substances, oxirane (ethylene oxide) and methyloxirane (propylene oxide), which are also direct-acting alkylating agents, have been classified as carcinogenic</p>
TITANIUM DIOXIDE	<p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. Exposure to titanium dioxide is via inhalation, swallowing or skin contact. When inhaled, it may deposit in lung tissue and lymph nodes causing dysfunction of the lungs and immune system. Absorption by the stomach and intestines depends on the size of the particle. It penetrated only the outermost layer of the skin, suggesting that healthy skin may be an effective barrier. There is no substantive data on genetic damage, though cases have been reported in experimental animals. Studies have differing conclusions on its cancer-causing potential.</p> <p>* IUCLID</p>
SILICA AMORPHOUS	<p>Reports indicate high/prolonged exposures to amorphous silicas induced lung fibrosis in experimental animals; in some experiments these effects were reversible. [PATTYS]</p>
SILICA CRYSTALLINE - QUARTZ	<p>WARNING: For inhalation exposure <u>ONLY</u>: This substance has been classified by the IARC as Group 1: CARCINOGENIC TO HUMANS</p> <p>The International Agency for Research on Cancer (IARC) has classified occupational exposures to respirable (<5 µm) crystalline silica as being carcinogenic to humans. This classification is based on what IARC considered sufficient evidence from epidemiological studies of humans for the carcinogenicity of inhaled silica in the forms of quartz and cristobalite. Crystalline silica is also known to cause silicosis, a non-cancerous lung disease. Intermittent exposure produces; focal fibrosis, (pneumoconiosis), cough, dyspnoea, liver tumours.</p> <p>* Millions of particles per cubic foot (based on impinger samples counted by light field techniques).</p> <p>NOTE : the physical nature of quartz in the product determines whether it is likely to present a chronic health problem. To be a hazard the material must enter the breathing zone as respirable particles.</p>
XYLENE	<p>Reproductive effector in rats</p>
BISPHENOL A/ BISPHENOL A DIGLYCIDYL ETHER POLYMER	<p>*Hexion MSDS Epikote 1001</p>
BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID	<p>Foetotoxicity has been observed in animal studies Oral (rabbit, female) NOEL 180 mg/kg (teratogenicity); NOEL (maternal 60 mg/kg)</p>
TALC	<p>The overuse of talc in nursing infants has resulted in respiratory damage causing fluid in the lungs and lung inflammation which may lead to death within hours of inhalation.</p> <p>Long-term exposure can also cause a variety of respiratory symptoms.</p>
METHYL ISOBUTYL KETONE	<p>MIBK is primarily absorbed by the lungs in animals and humans but can be absorbed by the skin, stomach and gut. If inhaled, it may be found in the brain, liver, lung, vitreous fluid, kidney and blood. Oral and respiratory routes of exposure are of minimal effect with changes seen only in the liver and kidney. MIBK does not cause genetic damage or harm the foetus or offspring, and has low toxicity to aquatic organisms.</p>
HYDROCARBON RESIN, POSTPOLYMERISED WITH MALEIC ANHYDRIDE	<p>Oral (-) LD50: 7000-10000 mg/kg Nil reported. [Manufacturer]</p>
TALLOW ALKYLDIMETHYLAMMONIUM CHLORIDE/ BENTONITE COMPLEX	<p>for organoclays:</p> <p>Acute toxicity: Based on the toxicokinetic data with B(Alk)2M bentonite, organoclay compounds are not expected to be absorbed following oral (gavage) exposure and will be excreted directly and rapidly in faeces with negligible elimination via urine and bile. There is no evidence of any tissue retention or systemic uptake of these substances. Based on reported particle size distribution data for consumer and industrial products, these materials are not expected to be respirable. These materials are also not expected to be absorbed through the skin based on the physical chemical properties as well as these reported particle sizes.</p> <p>Numerous acute toxicity studies have been conducted with all organoclay category members. The studies show a low order of toxicity with inhalation 4-hr LC50s and oral (gavage) LD50s greater than 5.0 mg/L and 5,000 mg/kg bw, respectively. The inhalation 1 hr LC50 > 200 mg/L for B(Alk)2M hectorite. Common clinical signs associated with acute inhalation in several studies included transient weight loss and respiratory irregularity. Clinical signs observed in the oral studies included diarrhea, rapid breathing, piloerection, swollen abdomen, ataxia and lethargy. Acute dermal toxicity studies were not located for the organoclays. The organoclays are not irritating to the skin. Eye irritation is generally minimal in humans (irritation due to the physical nature of the compounds were noted), although observations of moderate irritation have been reported in animals. Skin sensitisation tests conducted with 2M(2Alk) bentonite, and B(Alk)2M bentonite and B(2Alk)M hectorite indicate the organoclay materials are not sensitisers.</p> <p>Repeated-dose toxicity studies by the oral route of exposure (gavage or dietary) and using methods similar to OECD TG 407 have been conducted with 2M(2Alk) bentonite, and B(Alk)2M bentonite and B(2Alk)M hectorite. The NOAEL for 2M(2Alk) bentonite in a 12-week rat dietary study was 25% (approx. 12,500-25,000 mg/kg-bw/day), the highest dose tested. The NOAEL for B(Alk)2M bentonite in a 28-day rat oral (gavage) study was 1,000 mg/kg bw/d, the only dose tested. The LOAEL for B(2Alk)M hectorite in a 28-day rat oral (gavage) study was 1,000 mg/kg bw/d, the only dose tested based on decreased thrombotest time, decreased chloride and calcium levels, and increased adrenal weights. The repeated-dose toxicity of the remaining members of the Organoclays Category is expected to be similar.</p> <p>Genotoxicity: The category members B(Alk)2M hectorite, 2M(2Alk) bentonite, and 2M(2Alk) hectorite were tested in bacterial reverse mutation assays (with and without metabolic activation) and B[Alk]2M hectorite was tested in vitro using mouse lymphoma cells; these substances were negative for gene mutations in these assays. B(Alk)2M bentonite, was also negative in a reverse mutation assay. In in vivo chromosomal aberrations and in vivo micronucleus assays, B[Alk]2M bentonite and B[2Alk]M hectorite were both negative for chromosomal aberrations.</p> <p>Carcinogenicity: There are no data available regarding the carcinogenicity of these materials. The impurity, respirable crystalline silica, (which may be present at levels of 0.2% in B(Alk)2M hectorite; 0.1-5% in 2M(2Alk) bentonite; and 0.1-10% in B(2Alk)M bentonite) is considered a known human carcinogen (Group 1 according to IARC).</p> <p>Developmental and reproductive toxicity: A one-generation reproduction study using B(2Alk)M hectorite at dose levels of 0, 50, 225, and 1,000 mg/kg bw/d has been conducted for the potential to cause developmental toxicity in rats. The compound was not found to be teratogenic, nor was there any reproductive toxicity at any dose level. Based on the lack of reproductive toxicity or developmental effects with B(2Alk)M hectorite, organoclays are not expected to cause developmental toxicity or to demonstrate reproductive toxicity at doses up to 1,000 mg/kg bw/day.</p>
1,3,5-TRIMETHYL BENZENE	<p>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>CHEMWATCH 12171 1,2,4-trimethylbenzene</p>
1,2,4-TRIMETHYL BENZENE	<p>CHEMWATCH 2325 1,3,5-trimethylbenzene</p>

ISOPROPYL BENZENE - CUMENE	<p>For aromatic terpenes: p-cymene and cumene have low toxic potential and are excreted in the urine. At very high doses in animal testing, inco-ordination, damage to the kidneys and lung inflammation, with decrease in thymus weight, occurred. This group of substances does not seem to cause cancer, genetic damage or developmental toxicity and has low potential for reproductive toxicity.</p> <p>Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [National Toxicology Program: U.S. Dep. of Health & Human Services 2002]</p> <p>Cumene is reasonably anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity from studies in experimental animals. Cumene caused tumours at several tissue sites, including lung and liver in mice and kidney in male rats. Several proposed mechanisms of carcinogenesis support the relevance to humans of lung and liver tumours in experimental animals. Specifically, there is evidence that humans and experimental animals metabolise cumene through similar metabolic pathways. There is also evidence that cumene is genotoxic in some tissues, based on findings of DNA damage in rodent lung and liver. Furthermore, mutations of the K-ras oncogene and p53 tumor-suppressor gene observed in cumene-induced lung tumours in mice, along with altered expression of many other genes, resemble molecular alterations found in human lung and other cancers. The relevance of the kidney tumours to cancer in humans is uncertain; there is evidence that a species-specific mechanism not relevant to humans contributes to their induction, but it is possible that other mechanisms relevant to humans, such as genotoxicity, may also contribute to kidney-tumour formation in male rats.</p>
PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER	<p>for propylene glycol ethers (PGEs): Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM).</p> <p>Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids. Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).</p> <p>This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.</p> <p>Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body.</p> <p>As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces.</p> <p>As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating.</p> <p>None are skin sensitizers.</p> <p>In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested).</p> <p>Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members.</p> <p>One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. In a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health.</p> <p>In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity.</p> <p>The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. <i>In vitro</i>, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic <i>in vivo</i>. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.</p> <p>A BASF report (in ECETOC) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects.</p> <p>The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.]</p> <p>A BASF report (in ECETOC) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects. The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.] *Shin-Etsu SDS</p>
NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT	<p>Inhalation (rat) TClO: 1320 ppm/6h/90D-I * [Devoe]</p>
PHOSPHORIC ACID	<p>for acid mists, aerosols, vapours</p> <p>Data from assays for genotoxic activity <i>in vitro</i> suggest that eukaryotic cells are susceptible to genetic damage when the pH falls to about 6.5. Cells from the respiratory tract have not been examined in this respect. Mucous secretion may protect the cells of the airways from direct exposure to inhaled acidic mists, just as mucous plays an important role in protecting the gastric epithelium from its auto-secreted hydrochloric acid. In considering whether pH itself induces genotoxic events <i>in vivo</i> in the respiratory system, comparison should be made with the human stomach, in which gastric juice may be at pH 1-2 under fasting or nocturnal conditions, and with the human urinary bladder, in which the pH of urine can range from <5 to > 7 and normally averages 6.2. Furthermore, exposures to low pH <i>in vivo</i> differ from exposures <i>in vitro</i> in that, <i>in vivo</i>, only a portion of the cell surface is subjected to the adverse conditions, so that perturbation of intracellular homeostasis may be maintained more readily than <i>in vitro</i>.</p>

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	<p>The material may cause severe skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin. Repeated exposures may produce severe ulceration.</p> <p>phosphoric acid (85%)</p>
N-BUTANOL	<p>for n-butanol</p> <p>Acute toxicity: n-Butanol (BA) was only slightly toxic to experimental animals following acute oral, dermal, or inhalation exposure. The acute oral LD50 values for female rats ranged from 790 to 4360 mg/kg. Different strains of rat were used in each of four studies, which may account for the variability. Oral LD50 values for mice, rabbits, hamsters, dogs, and male rats all fell within the same range. The rat inhalation LC0 of 8000 ppm (24000 mg/m3) indicates very low inhalation toxicity (no lethality at 8000 ppm). The rabbit dermal LD50 was 3402 mg/kg, indicating that BA can penetrate the skin, but not very readily. Animal experiments and human experience indicate that BA is, at most, moderately irritating to the skin, but it is a severe eye irritant. These effects are most likely due to BA's localised defatting and drying characteristics. Although no animal data are available, human studies and experience show that BA is not likely to be a skin sensitiser.</p> <p>The median odor threshold for BA (0.17 ppm) is well below the lowest nasal irritation threshold in humans (289 ppm), allowing warning of possible chemical exposure prior to nasal irritation occurring. Human studies are complicated by the odor characteristics of the material, as the odor threshold is well below the levels at which irritation is observed.</p> <p>Repeat dose toxicity: An in vivo toxicokinetics study confirmed the rapid metabolism of n-butyl acetate (BAC) to BA. Hydrolysis of BAC in blood and brain was estimated to be 99 percent complete within 2.7 minutes (elimination t1/2 = 0.41 minute). Thus, organisms exposed to BAC can experience appreciable tissue concentrations of BA. In this way, the results of toxicity studies with BAC can be used as supplemental, surrogate data to provide information on the toxicity of BA.</p> <p>A thirteen-week, subchronic exposure to BAC, the metabolic precursor of BA, produced transient hypoactivity (during exposure only) at 1500 and 3000 ppm (7185 and 14370 mg/m3) along with decreased body weight and food consumption, but no post exposure neurotoxicity even at 3000 ppm. A concurrent subchronic neurotoxicity study under the same exposure conditions showed no evidence of cumulative neurotoxicity based upon functional observational battery endpoints, quantitative motor activity, neuropathology and scheduled-controlled operant behavior endpoints. A no observable effect level (NOAEL) of 500 ppm (2395 mg/m3) was reported for systemic effects in rats, and a NOAEL of 3000 ppm (14370 mg/m3) was reported for post exposure neurotoxicity in rats.</p> <p>Reproductive toxicity: Several studies indicate that BA is not a reproductive toxicant.</p> <p>Female rats exposed to 6000 ppm (18000 mg/m3) BA throughout gestation and male rats exposed to 6000 ppm (18000 mg/m3) BA for six weeks prior to mating showed no effects on fertility or pregnancy rate. Male rats given BA at 533 mg/kg/day for 5 days had no testicular toxicity.</p> <p>Developmental toxicity: BA produced only mild foetotoxicity and developmental alterations at or near the maternally toxic (even lethal) dose of 8000 ppm (24000 mg/m3) throughout gestation.</p> <p>Genotoxicity: An entire battery of negative in vitro tests and a negative in vivo micronucleus test indicate that BA is not genotoxic.</p> <p>Carcinogenicity: Based upon the battery of negative mutagenicity and clastogenicity findings, BA presents a very small potential for carcinogenicity.</p>
C.I. PIGMENT BLACK 11	No data of toxicological significance identified in literature search.
CARBON BLACK	Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported
Carboguard 635HAR Part A & BISPHENOL A/ BISPHENOL A DIGLYCIDYL ETHER POLYMER & TALC & METHYL ISOBUTYL KETONE & 1,3,5-TRIMETHYL BENZENE & 1,2,4-TRIMETHYL BENZENE & 1,2,3-TRIMETHYL BENZENE & ISOPROPYL BENZENE - CUMENE & PROPYLBENZENE & PHOSPHORIC ACID & C.I. PIGMENT YELLOW 42 & N-BUTANOL & C.I. PIGMENT BLACK 11	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.</p>
Carboguard 635HAR Part A & ALUMINIUM HYDROXIDE & BISPHENOL A/ BISPHENOL A DIGLYCIDYL ETHER POLYMER & BARIUM SULFATE & TALC & TALLOW ALKYLDIMETHYLAMMONIUM CHLORIDE/ BENTONITE COMPLEX & PHOSPHORIC ACID & C.I. PIGMENT YELLOW 42 & C.I. PIGMENT BLACK 11 & CARBON BLACK	No significant acute toxicological data identified in literature search.
Carboguard 635HAR Part A & BISPHENOL A/ BISPHENOL A DIGLYCIDYL ETHER POLYMER & BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID	<p>The chemical structure of hydroxylated diphenylalkanes or bisphenols consists of two phenolic rings joined together through a bridging carbon. This class of endocrine disruptors that mimic oestrogens is widely used in industry, particularly in plastics</p> <p>Bisphenol A (BPA) and some related compounds exhibit oestrogenic activity in human breast cancer cell line MCF-7, but there were remarkable differences in activity. Several derivatives of BPA exhibited significant thyroid hormonal activity towards rat pituitary cell line GH3, which releases growth hormone in a thyroid hormone-dependent manner. However, BPA and several other derivatives did not show such activity. Results suggest that the 4-hydroxyl group of the A-phenyl ring and the B-phenyl ring of BPA derivatives are required for these hormonal activities, and substituents at the 3,5-positions of the phenyl rings and the bridging alkyl moiety markedly influence the activities.</p> <p>Bisphenols promoted cell proliferation and increased the synthesis and secretion of cell type-specific proteins. When ranked by proliferative potency, the longer the alkyl substituent at the bridging carbon, the lower the concentration needed for maximal cell yield; the most active compound contained two propyl chains at the bridging carbon. Bisphenols with two hydroxyl groups in the para position and an angular configuration are suitable for appropriate hydrogen bonding to the acceptor site of the oestrogen receptor.</p>
Carboguard 635HAR Part A & 1,3,5-TRIMETHYL BENZENE & 1,2,4-TRIMETHYL BENZENE & 1,2,3-TRIMETHYL BENZENE & NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT	<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</p>

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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.

Neurotoxicity 1,2,4-Trimethylbenzene depresses the central nervous system. Exposure to solvent mixtures containing the chemical causes headache, fatigue, nervousness, and drowsiness. Occupationally, workers exposed to a solvent containing 50% 1,2,4-trimethylbenzene had nervousness, headaches, drowsiness, and vertigo (U.S. EPA). Headache, fatigue, and drowsiness were reported for workers exposed (no dose given) to paint thinner containing 80% 1,2,4- and 1,3,5-trimethylbenzenes.

Results of the developmental toxicity study indicate that the C9 fraction caused adverse neurological effects at the highest dose (1500 ppm) tested.

Subchronic/Chronic Toxicity Long-term exposure to solvents containing 1,2,4-trimethylbenzene may cause nervousness, tension, and bronchitis. Painters that worked for several years with a solvent containing 50% 1,2,4- and 30% 1,3,5-trimethylbenzene showed nervousness, tension and anxiety, asthmatic bronchitis, anemia, and alterations in blood clotting; haematological effects may have been due to trace amounts of benzene.

Rats given 1,2,4-trimethylbenzene orally at doses of 0.5 or 2.0 g/kg/day, 5 days/week for 4 weeks. All rats exposed to the high dose died and 1 rat in the low dose died (no times given); no other effects were reported. Rats exposed by inhalation to 1700 ppm of a trimethylbenzene isomeric mixture for 4 months had decreased weight gain, lymphopenia and neutrophilia.

Genotoxicity: Results of mutagenicity testing, indicate that the C9 fraction does not induce gene mutations in prokaryotes (Salmonella 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.

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.

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.

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 NOAEL for systemic and/or neurotoxicity was 6,500 mg/m³, the highest concentration tested. In an inhalation study of a commercial blend, rats were exposed to C9 aromatic naphtha concentrations of 0, 96, 198, or 373 ppm (0, 470, 970, 1830 mg/m³) for 6 hr/day, 5 days/week, for 12 months. Liver and kidney weights were increased in the high exposure group but no accompanying histopathology was observed in these organs.

The NOAEL was considered to be the high exposure level of 373 ppm, or 1830 mg/m³. In two subchronic rat inhalation studies, both of three months duration, rats were exposed to the individual TMB isomers (1,2,4- and 1,3,5-) to nominal concentrations of 0, 25, 100, or 250 ppm (0, 123, 492, or 1230 mg/m³). Respiratory irritation was observed at 492 (100 ppm) and 1230 mg/m³ (250 ppm) and no systemic toxicity was observed in either study. For both pure isomers, the NOELs are 25 ppm or 123 mg/m³ for respiratory irritation and 250 ppm or 1230 mg/m³ for systemic effects.

Oral: The C9 aromatic naphtha has not been tested via the oral route of exposure. Individual TMB isomers have been evaluated in a series of repeated-dose oral studies ranging from 14 days to 3 months over a wide range of doses. The effects observed in these studies included increased liver and kidney weights, changes in blood chemistry, increased salivation, and decreased weight gain at higher doses. Organ weight changes appeared to be adaptive as they were not accompanied by histopathological effects. Blood changes appeared sporadic and without pattern. One study reported hyaline droplet nephropathy in male rats at the highest dose (1000 mg/kg bw-day), an effect that is often associated with alpha-2mu-globulin-induced nephropathy and not considered relevant to humans. The doses at which effects were detected were 100 mg/kg-bw day or above (an exception was the pilot 14 day oral study - LOAEL 150 mg/kg bw-day - but the follow up three month study had a LOAEL of 600 mg/kg/bw-day with a NOAEL of 200 mg/kg bw-day). Since effects generally were not severe and could be considered adaptive or spurious, oral exposure does not appear to pose a high toxicity hazard for pure trimethylbenzene isomers.

Mutagenicity

In vitro genotoxicity testing of a variety of C9 aromatics has been conducted in both bacterial and mammalian cells. In vitro point mutation tests were conducted with Salmonella typhimurium and Escherichia coli bacterial strains, as well as with cultured mammalian cells such as the Chinese hamster cell ovary cells (HGPRT assay) with and without metabolic activation. In addition, several types of in vitro chromosomal aberration tests have been performed (chromosome aberration frequency in Chinese hamster ovary and lung cells, sister chromatid exchange in CHO cells). Results were negative both with and without metabolic activation for all category members. For the supporting chemical 1,2,3-TMB, a single in vitro chromosome aberration test was weakly positive. In in vivo bone marrow cytogenetics test, rats were exposed to C9 aromatic naphtha at concentrations of 0, 153, 471, or 1540 ppm (0, 750, 2,310, or 7,560 mg/m³) 6 hr/day, for 5 days. No evidence of in vivo somatic cell 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

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	<p>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).</p> <p>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.</p> <p>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.</p> <p>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.</p>
<p>Carboguard 635HAR Part A & BISPHENOL A/ BISPHENOL A DIGLYCIDYL ETHER POLYMER & BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID</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>TITANIUM DIOXIDE & XYLENE & METHYL ISOBUTYL KETONE & 1,3,5-TRIMETHYL BENZENE & ISOPROPYL BENZENE - CUMENE & N-BUTANOL</p>	<p>The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.</p>
<p>TITANIUM DIOXIDE & METHYL ISOBUTYL KETONE & ISOPROPYL BENZENE - CUMENE & CARBON BLACK</p>	<p>WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.</p>
<p>SILICA AMORPHOUS & SILICA AMORPHOUS, FUMED, CRYSTALLINE FREE</p>	<p>For silica amorphous:</p> <p>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.</p> <p>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.</p> <p>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 sensitizer.</p> <p>Repeated-dose and chronic toxicity studies confirm the absence of toxicity when SAS is swallowed or upon skin contact.</p> <p>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.</p> <p>Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted with SAS in a number of species, at airborne concentrations ranging from 0.5 mg/m3 to 150 mg/m3. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m3. When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m3. The difference in values may be explained by different particle size, and therefore the number of particles administered per unit dose. In general, as particle size decreases so does the NOAEL/LOAEL.</p> <p>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. In humans, 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.</p> <p>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.</p>
<p>SILICA AMORPHOUS & XYLENE & BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID & TALC & C.I. PIGMENT YELLOW 42</p>	<p>The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.</p>
<p>XYLENE & PHOSPHORIC ACID & N-BUTANOL</p>	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
<p>BISPHENOL A/ BISPHENOL A DIGLYCIDYL ETHER POLYMER & BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID</p>	<p>In mice, dermal application of bisphenol A diglycidyl ether (BADGE) (1, 10, or 100 mg/kg) for 13 weeks produced mild to moderate chronic active dermatitis. At the high dose, spongiosis and epidermal micro abscess formation were observed. In rats, dermal application of BADGE (10, 100, or 1000 mg/kg) for 13 weeks resulted in a decrease in body weight at the high dose. The no-observable effect level (NOEL) for dermal exposure was 100 mg/kg for both sexes. In a separate study, application of BADGE (same doses) five times per week for ~13 weeks not only caused a decrease in body weight but also produced chronic dermatitis at all dose levels in males and at >100 mg/kg in females (as well as in a satellite group of females given 1000 mg/kg).</p> <p>Reproductive and Developmental Toxicity: BADGE (50, 540, or 750 mg/kg) administered to rats via gavage for 14 weeks (P1) or 12 weeks (P2) produced decreased body weight in all males at the mid dose and in both males and females at the high dose, but had no reproductive effects. The NOEL for reproductive effects was 750 mg/kg.</p>

Carcinogenicity: IARC concluded that "there is limited evidence for the carcinogenicity of bisphenol A diglycidyl ether in experimental animals." Its overall evaluation was "Bisphenol A diglycidyl ether is not classifiable as to its carcinogenicity to humans (Group 3).

In a lifetime tumourigenicity study in which 90-day-old C3H mice received three dermal applications per week of BADGE (undiluted dose) for 23 months, only one out of 32 animals developed a papilloma after 16 months. A retest, in which skin paintings were done for 27 months, however, produced no tumours (Weil et al., 1963). In another lifetime skin-painting study, BADGE (dose n.p.) was also reported to be noncarcinogenic to the skin of C3H mice; it was, however, weakly carcinogenic to the skin of C57BL/6 mice (Holland et al., 1979; cited by Canter et al., 1986). In a two-year bioassay, female Fisher 344 rats dermally exposed to BADGE (1, 100, or 1000 mg/kg) showed no evidence of dermal carcinogenicity but did have low incidences of tumours in the oral cavity (U.S. EPA, 1997).

Genotoxicity: In *S. typhimurium* strains TA100 and TA1535, BADGE (10-10,000 ug/plate) was mutagenic with and without S9; negative results were obtained in TA98 and TA1537 (Canter et al., 1986; Pullin, 1977). In a spot test, BADGE (0.05 or 10.00 mg) failed to show mutagenicity in strains TA98 and TA100 (Wade et al., 1979). Negative results were also obtained in the body fluid test using urine of female BDF and ICR mice (1000 mg/kg BADGE), the mouse host-mediated assay (1000 mg/kg), micronucleus test (1000 mg/kg), and dominant lethal assay (~3000 mg/kg).

Immunotoxicity: Intracutaneous injection of diluted BADGE (0.1 mL) three times per week on alternate days (total of 8 injections) followed by a three-week incubation period and a challenge dose produced sensitisation in 19 of 20 guinea pigs

Consumer exposure to BADGE is almost exclusively from migration of BADGE from can coatings into food. Using a worst-case scenario that assumes BADGE migrates at the same level into all types of food, the estimated per capita daily intake for a 60-kg individual is approximately 0.16 ug/kg body weight/day. A review of one- and two-generation reproduction studies and developmental investigations found no evidence of reproductive or endocrine toxicity, the upper ranges of dosing being determined by maternal toxicity. The lack of endocrine toxicity in the reproductive and developmental toxicological tests is supported by negative results from both in vivo and in vitro assays designed specifically to detect oestrogenic and androgenic properties of BADGE. An examination of data from sub-chronic and chronic toxicological studies support a NOAEL of 50 mg/kg body weight/day from the 90-day study, and a NOAEL of 15 mg/kg body weight/day (male rats) from the 2-year carcinogenicity study. Both NOAELs are considered appropriate for risk assessment. Comparing the estimated daily human intake of 0.16 ug/kg body weight/day with the NOAELs of 50 and 15 mg/kg body weight/day shows human exposure to BADGE from can coatings is between 250,000 and 100,000-fold lower than the NOAELs from the most sensitive toxicology tests. These large margins of safety together with lack of reproductive, developmental, endocrine and carcinogenic effects supports the continued use of BADGE for use in articles intended to come into contact with foodstuffs.

1,3,5-TRIMETHYL BENZENE & 1,2,4-TRIMETHYL BENZENE	Other Toxicity data is available for
1,3,5-TRIMETHYL BENZENE & 1,2,4-TRIMETHYL BENZENE	CHEMWATCH 12172 1,2,3-trimethylbenzene

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

Legend: ☒ – Data available but does not fill the criteria for classification
 ☑ – Data available to make classification
 ☒ – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
aluminium hydroxide	LC50	96	Fish	0.2262mg/L	2
aluminium hydroxide	EC50	48	Crustacea	0.7364mg/L	2
aluminium hydroxide	EC50	96	Algae or other aquatic plants	0.0054mg/L	2
aluminium hydroxide	EC50	168	Crustacea	0.0076mg/L	2
aluminium hydroxide	NOEC	72	Algae or other aquatic plants	>=0.004mg/L	2
titanium dioxide	LC50	96	Fish	9.214mg/L	3
titanium dioxide	EC50	48	Crustacea	>10mg/L	2
titanium dioxide	EC50	72	Algae or other aquatic plants	5.83mg/L	4
titanium dioxide	EC20	72	Algae or other aquatic plants	1.81mg/L	4
titanium dioxide	NOEC	336	Fish	0.089mg/L	4
silica amorphous	LC50	96	Fish	120.743mg/L	3
silica amorphous	EC50	48	Crustacea	ca.7600mg/L	1
silica amorphous	EC50	72	Algae or other aquatic plants	440mg/L	1
silica amorphous	EC50	384	Crustacea	28.000mg/L	3
silica amorphous	NOEC	72	Algae or other aquatic plants	60mg/L	1
xylene	LC50	96	Fish	2.6mg/L	2
xylene	EC50	48	Crustacea	>3.4mg/L	2
xylene	EC50	72	Algae or other aquatic plants	4.6mg/L	2
xylene	EC50	24	Crustacea	0.711mg/L	4
xylene	NOEC	73	Algae or other aquatic plants	0.44mg/L	2
barium sulfate	LC50	96	Fish	>3.5mg/L	2
barium sulfate	EC50	48	Crustacea	32mg/L	4
barium sulfate	EC50	72	Algae or other aquatic plants	>1.15mg/L	2
barium sulfate	EC50	72	Algae or other aquatic plants	>30.07mg/L	2

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barium sulfate	NOEC	72	Algae or other aquatic plants	>=1.15mg/L	2
bisphenol A/ diglycidyl ether resin, liquid	LC50	96	Fish	1.2mg/L	2
bisphenol A/ diglycidyl ether resin, liquid	EC50	72	Algae or other aquatic plants	9.4mg/L	2
bisphenol A/ diglycidyl ether resin, liquid	EC50	24	Crustacea	3.6mg/L	2
bisphenol A/ diglycidyl ether resin, liquid	NOEC	72	Algae or other aquatic plants	2.4mg/L	2
methyl isobutyl ketone	LC50	96	Fish	69.808mg/L	3
methyl isobutyl ketone	EC50	48	Crustacea	=170mg/L	1
methyl isobutyl ketone	EC50	96	Algae or other aquatic plants	275.488mg/L	3
methyl isobutyl ketone	EC50	384	Crustacea	16.425mg/L	3
methyl isobutyl ketone	NOEC	504	Crustacea	30mg/L	2
1,3,5-trimethyl benzene	LC50	96	Fish	1.318mg/L	3
1,3,5-trimethyl benzene	EC50	48	Crustacea	13mg/L	5
1,3,5-trimethyl benzene	EC50	96	Algae or other aquatic plants	2.154mg/L	3
1,3,5-trimethyl benzene	EC50	384	Crustacea	0.328mg/L	3
1,3,5-trimethyl benzene	NOEC	504	Crustacea	0.4mg/L	4
1,2,4-trimethyl benzene	LC50	96	Fish	1.318mg/L	3
1,2,4-trimethyl benzene	EC50	48	Crustacea	ca.6.14mg/L	1
1,2,4-trimethyl benzene	EC50	96	Algae or other aquatic plants	2.154mg/L	3
1,2,4-trimethyl benzene	EC50	384	Crustacea	0.328mg/L	3
1,2,3-trimethyl benzene	LC50	96	Fish	1.318mg/L	3
1,2,3-trimethyl benzene	EC50	96	Algae or other aquatic plants	2.154mg/L	3
1,2,3-trimethyl benzene	EC50	384	Crustacea	0.328mg/L	3
isopropyl benzene - cumene	LC50	96	Fish	1.784mg/L	3
isopropyl benzene - cumene	EC50	48	Crustacea	=0.6mg/L	1
isopropyl benzene - cumene	EC50	72	Algae or other aquatic plants	1.29mg/L	2
isopropyl benzene - cumene	EC50	384	Crustacea	0.442mg/L	3
isopropyl benzene - cumene	NOEC	72	Algae or other aquatic plants	0.22mg/L	2
propylbenzene	LC50	96	Fish	1.55mg/L	4
propylbenzene	EC50	48	Crustacea	109mg/L	4
propylbenzene	EC50	72	Algae or other aquatic plants	1.8mg/L	4
propylbenzene	EC50	384	Crustacea	0.394mg/L	3
propylene glycol monomethyl ether acetate, alpha-isomer	LC50	96	Fish	100mg/L	1
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	48	Crustacea	=408mg/L	1
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	96	Algae or other aquatic plants	9.337mg/L	3
propylene glycol monomethyl ether acetate, alpha-isomer	EC0	24	Crustacea	=500mg/L	1
propylene glycol monomethyl ether acetate, alpha-isomer	NOEC	336	Fish	47.5mg/L	2
naphtha petroleum, light aromatic solvent	EC50	48	Crustacea	=6.14mg/L	1
naphtha petroleum, light aromatic solvent	EC50	72	Algae or other aquatic plants	3.29mg/L	1
naphtha petroleum, light aromatic solvent	EC10	72	Algae or other aquatic plants	1.13mg/L	1
naphtha petroleum, light aromatic solvent	NOEC	72	Algae or other aquatic plants	=1mg/L	1
phosphoric acid	LC50	96	Fish	75.1mg/L	2
phosphoric acid	EC50	48	Crustacea	>376mg/L	2
phosphoric acid	EC50	72	Algae or other aquatic plants	77.9mg/L	2
phosphoric acid	EC50	24	Crustacea	>376mg/L	2
phosphoric acid	NOEC	72	Algae or other aquatic plants	<7.5mg/L	2
C.I. Pigment Yellow 42	LC50	96	Fish	0.05mg/L	2
C.I. Pigment Yellow 42	EC50	72	Algae or other aquatic plants	18mg/L	2

C.I. Pigment Yellow 42	EC50	504	Crustacea	4.49mg/L	2
C.I. Pigment Yellow 42	NOEC	504	Fish	0.52mg/L	2
n-butanol	LC50	96	Fish	88.462mg/L	3
n-butanol	EC50	48	Crustacea	>500mg/L	1
n-butanol	EC50	96	Algae or other aquatic plants	225mg/L	2
n-butanol	BCF	24	Fish	921mg/L	4
n-butanol	EC50	384	Crustacea	20.661mg/L	3
n-butanol	NOEC	48	Crustacea	415mg/L	2
C.I. Pigment Black 11	LC50	96	Fish	0.05mg/L	2
C.I. Pigment Black 11	EC50	72	Algae or other aquatic plants	18mg/L	2
C.I. Pigment Black 11	EC50	504	Crustacea	4.49mg/L	2
C.I. Pigment Black 11	NOEC	504	Fish	0.52mg/L	2
carbon black	LC50	96	Fish	=1000mg/L	1
carbon black	EC50	24	Crustacea	>5600mg/L	1
carbon black	NOEC	96	Fish	=1000mg/L	1

Legend:

Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

For 1,2,4 - Trimethylbenzene:

Half-life (hr) air: 0.48-16;

Half-life (hr) H₂O surface water: 0.24 -672;

Half-life (hr) H₂O ground: 336-1344;

Half-life (hr) soil: 168-672;

Henry's Pa m³/mol: 385 -627;

Bioaccumulation: not significant. 1,2,4-Trimethylbenzene is a volatile organic compound (VOC) substance.

Atmospheric Fate: 1,2,4-trimethylbenzene can contribute to the formation of photochemical smog in the presence of other VOCs. Degradation of 1,2,4-trimethylbenzene in the atmosphere occurs by reaction with hydroxyl radicals. Reaction also occurs with ozone but very slowly (half life 8820 days).

Aquatic Fate: 1,2,4-Trimethylbenzene volatilizes rapidly from surface waters with volatilization half-life from a model river calculated to be 3.4 hours. Biodegradation of 1,2,4-trimethylbenzene has been noted in both seawater and ground water. Various strains of Pseudomonas can biodegrade 1,2,4-trimethylbenzene.

Terrestrial Fate: 1,2,4-Trimethylbenzene also volatilizes from soils however; moderate adsorption to soils and sediments may occur. Volatilization is the major route of removal of 1,2,4-trimethylbenzene from soils; although, biodegradation may also occur. Due to the high volatility of the chemical it is unlikely to accumulate in soil or surface water to toxic concentrations.

Ecotoxicity: No significant bioaccumulation has been noted. 1,2,4-Trimethylbenzene is moderately toxic to fathead minnow and slightly toxic to dungeness crab. 1,2,4-Trimethylbenzene has moderate acute toxicity to aquatic organisms. No stress was observed in rainbow trout, sea lamprey and Daphnia magna water fleas. The high concentrations required to induce toxicity in laboratory animals are not likely to be reached in the environment.

For Aromatic Substances Series:

Environmental Fate: Large, molecularly complex polycyclic aromatic hydrocarbons, or PAHs, are persistent in the environment longer than smaller PAHs.

Atmospheric Fate: PAHs are 'semi-volatile substances' which can move between the atmosphere and the Earth's surface in repeated, temperature-driven cycles of deposition and volatilization.

Terrestrial Fate: BTEX compounds have the potential to move through soil and contaminate ground water, and their vapors are highly flammable and explosive.

Ecotoxicity - Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. The order of most toxic to least in a study using grass shrimp and brown shrimp was dimethylnaphthalenes > methylnaphthalenes > naphthalenes. 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 Silica:

Environmental Fate: Most documentation on the fate of silica in the environment concerns dissolved silica, in the aquatic environment, regardless of origin, (man-made or natural), or structure, (crystalline or amorphous).

Terrestrial Fate: Silicon makes up 25.7% of the Earth's crust, by weight, and is the second most abundant element, being exceeded only by oxygen. Silicon is not found free in nature, but occurs chiefly as the oxide and as silicates. Once released into the environment, no distinction can be made between the initial forms of silica.

Aquatic Fate: At normal environmental pH, dissolved silica exists exclusively as monosilicic acid. At pH 9.4, amorphous silica is highly soluble in water. Crystalline silica, in the form of quartz, has low solubility in water. Silicic acid plays an important role in the biological/geological/chemical cycle of silicon, especially in the ocean. Marine organisms such as diatoms, silicoflagellates and radiolarians use silicic acid in their skeletal structures and their skeletal remains leave silica in sea sediment

Ecotoxicity: Silicon is important to plant and animal life and is practically non-toxic to fish including zebrafish, and Daphnia magna water fleas.

For bisphenol A and related bisphenols:

Environmental fate:

Biodegradability (28 d) 89% - Easily biodegradable

Bioconcentration factor (BCF) 7.8 mg/l

Bisphenol A, its derivatives and analogues, can be released from polymers, resins and certain substances by metabolic products

Substance does not meet the criteria for PBT or vPvB according to Regulation (EC) No 1907/2006, Annex XIII

As an environmental contaminant, bisphenol A interferes with nitrogen fixation at the roots of leguminous plants associated with the bacterial symbiont Sinorhizobium meliloti. Despite a half-life in the soil of only 1-10 days, its ubiquity makes it an important pollutant. According to Environment Canada, "initial assessment shows that at low levels, bisphenol A can harm fish and organisms over time. Studies also indicate that it can currently be found in municipal wastewater." However, a study conducted in the United States found that 91-98% of bisphenol A may be removed from water during treatment at municipal water treatment plants.

Ecotoxicity:

Fish LC50 (96 h): 4.6 mg/l (freshwater fish); 11 mg/l (saltwater fish); NOEC 0.016 mg/l (freshwater fish- 144 d); 0.064 mg/l (saltwater fish 164 d)

Fresh water invertebrates EC50 (48 h): 10.2 mg/l; NOEC 0.025 mg/l - 328 d)

Marine water invertebrate EC50 (96 h): 1.1 mg/l; NOEC 0.17 mg/l (28 d)

Freshwater algae (96 h): 2.73 mg/l

Marine water algae (96 h): 1.1 mg/l

Fresh water plant EC50 (7 d): 20 mg/l; NOEC 7.8 mg/l

In general, studies have shown that bisphenol A can affect growth, reproduction and development in aquatic organisms.

Among freshwater organisms, fish appear to be the most sensitive species. Evidence of endocrine-related effects in fish, aquatic invertebrates, amphibians and reptiles has been reported at environmentally relevant exposure levels lower than those required for acute toxicity. There is a widespread variation in reported values for endocrine-related effects, but many fall in the range of 1 ug/L to 1 mg/L

A 2009 review of the biological impacts of plasticisers on wildlife published by the Royal Society with a focus on annelids (both aquatic and terrestrial), molluscs, crustaceans, insects, fish and amphibians concluded that bisphenol A has been shown to affect reproduction in all studied animal groups, to impair development in crustaceans and amphibians and to induce genetic aberrations. A large 2010 study of two rivers in Canada found that areas contaminated with hormone-like chemicals including bisphenol A showed females made up 85 per cent of the population of a certain fish,

while females made up only 55 per cent in uncontaminated areas.

Although abundant data are available on the toxicity of bisphenol-A (2,2-bis (4-hydroxydiphenyl)propane;(BPA) A variety of BPs were examined for their acute toxicity against *Daphnia magna*, mutagenicity, and oestrogenic activity using the Daphtoxkit (Creasel Ltd.), the umu test system, and the yeast two-hybrid system, respectively, in comparison with BPA. BPA was moderately toxic to *D. magna* (48-h EC50 was 10 mg/l) according to the current U.S. EPA acute toxicity evaluation standard, and it was weakly oestrogenic with 5 orders of magnitude lower activity than that of the natural estrogen 17 beta-oestradiol in the yeast screen, while no mutagenicity was observed. All seven BPs tested here showed moderate to slight acute toxicity, no mutagenicity, and weak oestrogenic activity as well as BPA. Some of the BPs showed considerably higher oestrogenic activity than BPA, and others exhibited much lower activity. Bisphenol S (bis(4-hydroxydiphenyl)sulfone) and bis(4-hydroxyphenyl)sulfide showed oestrogenic activity.

Biodegradation is a major mechanism for eliminating various environmental pollutants. Studies on the biodegradation of bisphenols have mainly focused on bisphenol A. A number of BPA-degrading bacteria have been isolated from enrichments of sludge from wastewater treatment plants. The first step in the biodegradation of BPA is the hydroxylation of the carbon atom of a methyl group or the quaternary carbon in the BPA molecule. Judging from these features of the biodegradation mechanisms, it is possible that the same mechanism used for BPA is used to biodegrade all bisphenols that have at least one methyl or methylene group bonded at the carbon atom between the two phenol groups. However, bisphenol F (1-bis(4-hydroxyphenyl)methane; BPF), which has no substituent at the bridging carbon, is unlikely to be metabolised by such a mechanism. Nevertheless BPF is readily degraded by river water microorganisms under aerobic conditions. From this evidence, it was clear that a specific mechanism for biodegradation of BPF does exist in the natural ecosystem. Algae can enhance the photodegradation of bisphenols. The photodegradation rate of BPF increased with increasing algae concentration. Humic acid and Fe³⁺ ions also enhanced the photodegradation of BPF. The effect of pH value on the BPF photodegradation was also important.

Significant environmental findings are limited. Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit common characteristics with respect to environmental fate and ecotoxicology. One such oxirane is ethyloxirane and data presented here may be taken as representative.

#90oxirane

For 1,2-Butylene oxide (Ethyloxirane):

log Kow values of 0.68 and 0.86. BAF and BCF : 1 to 17 L/kg.

Aquatic Fate - Ethyloxirane is highly soluble in water and has a very low soil-adsorption coefficient, which suggests that, if released to water, adsorption of ethyloxirane to sediment and suspended solids is not expected. Volatilization of ethyloxirane from water surfaces would be expected. Ethyloxirane is hydrolysable, with a half-life of 6.5 days, and biodegradable up to 100% degradation and is not expected to persist in water. Models have predicted a biodegradation half-life in water of 15 days.

Terrestrial Fate: When released to soil, ethyloxirane is expected to have low adsorption and thus very high mobility. Volatilization from moist soil and dry soil surfaces is expected. Ethyloxirane is not expected to be persistent in soil.

Atmospheric Fate: It is expected that ethyloxirane exists solely as a vapor in ambient atmosphere. Ethyloxirane may also be removed from the atmosphere by wet deposition processes. The half-life in air is about 5.6 days from the reaction of ethyloxirane with photochemically produced hydroxyl radicals which indicates that this chemical meets the persistence criterion in air (half-life of = 2 days).

Ecotoxicity - The potential for bioaccumulation of ethyloxirane in organisms is likely to be low and has low to moderate toxicity to aquatic organisms. Ethyloxirane is acutely toxic to water fleas and toxicity values for bacteria are close to 5000 mg/L. For algae, toxicity values exceed 500 mg/L.

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log Kow values of 0.68 and 0.86. BAF and BCF : 1 to 17 L/kg.

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For Inorganic Sulfate:

Environmental Fate - Sulfates can produce a laxative effect at concentrations of 1000 - 1200 mg/liter, but no increase in diarrhea, dehydration or weight loss. The presence of sulfate in drinking-water can also result in a noticeable taste. Sulfate may also contribute to the corrosion of distribution systems. No health-based guideline value for sulfate in drinking water is proposed. **Atmospheric Fate**: Sulfates are removed from the air by both dry and wet deposition processes. Wet deposition processes including rain-out (a process that occurs within the clouds) and washout (removal by precipitation below the clouds) which contribute to the removal of sulfate from the atmosphere.

Terrestrial Fate: Soil - In soil, the inorganic sulfates can adsorb to soil particles or leach into surface water and groundwater. Plants - Sodium sulfate is not very toxic to terrestrial plants however; sulfates can be taken up by plants and be incorporated into the parenchyma of the plant. Some plants (e.g. corn and *Kochia Scoparia*) are capable of accumulating sulfate to concentrations that are potentially toxic to ruminants. Jack pine are the most sensitive plant species.

Aquatic Fate: Sulfate in water can also be reduced by sulfate bacteria (*Thiobacilli*) which use them as a source of energy. In anaerobic environments sulfate is biologically reduced to (hydrogen) sulfide by sulfate reducing bacteria, or incorporated into living organisms as source of sulfur. Sodium sulfate is not reactive in aqueous solution at room temperature. Sodium sulfate will completely dissolve, ionize and distribute across the entire planetary "aquasphere". Some sulfates may eventually be deposited with the majority of sulfates participating in the sulfur cycle in which natural and industrial sodium sulfates are not distinguishable.

Ecotoxicity: Significant bioconcentration or bioaccumulation is not expected. Algae are the most sensitive to sodium sulfate and toxicity occurs in bacteria from 2500mg/L. Sulfates are not acutely toxic to fish or invertebrates. *Daphnia magna* water fleas and fathead minnow appear to be the least sensitive species. Activated sludge showed a very low sensitivity to sodium sulfate. Overall it can be concluded that sodium sulfate has no acute adverse effect on aquatic and sediment dwelling organisms. No data were found for long term toxicity.

For Barium and its Compounds:

Environmental Fate: Barium is a highly reactive metal occurring naturally only in a combined state, primarily as inorganic complexes. Conditions such as pH, oxidation-reduction potential, cation exchange capacity, and the presence of sulfate, carbonate, and the presence of metal oxides will affect the partitioning of barium and its compounds in the environment. The element is released to environmental by both natural processes and man-made sources. Most barium released to the environment from industrial sources is in forms that do not become widely dispersed.

Atmospheric Fate: In the atmosphere, barium is likely to be present in particulate form. Barium compounds will be removed from the atmosphere via wet/dry deposition. The substance may change to different forms of barium in the air.

Terrestrial Fate: Soil - Barium will leach from geological formations to groundwater and will adsorb to soil. Barium is not very mobile in most soil systems and will form soluble complexes with fulvic/humic acids. Transportation rates of barium in soil are dependent on the characteristics of soil material. In soils with high positive ion exchange capacity, (e.g., fine textured mineral soils or soils with high organic matter content), barium mobility will be limited by adsorption. Soils high in calcium carbonate leave barium carbonate residues, which limit mobility. Barium produces barium sulfate residues in the presence of sulfates. Barium is more mobile, and is more likely to be leached, from soils in the presence of chloride and under acidic conditions. Barium binds with fatty acids, (e.g., in acidic landfill leachate), and will be much more mobile in soils containing fatty acids. Plants - Barium is not expected to concentrate in plants, relative to amounts found in soils; however, there are some plants, (beans, forage plants, Brazil nuts, and mushrooms), which accumulate barium.

Aquatic Fate: Barium will adsorb to sediment/suspended particulate matter. Precipitation of barium sulfate salts is accelerated where rivers enter the ocean. Sedimentation of suspended solids removes a large portion of the barium content from surface waters. Barium in sediments is found largely in the form of barium sulfate, (barite).

Ecotoxicity: Barium concentration will increase as it moves up the food chain in both land and aquatic species. In aquatic media, barium is likely to precipitate out of solution as an insoluble salt, (i.e. barium sulfate/barium sulfite). The uptake of barium by fish and marine organisms is also an important removal mechanism. Barium may concentrate in marine plants by a factor of 400-4,000 times the level present in the water. The substance may concentrate in marine animals, plankton, and brown algae.

For Xylenes:

log Koc : 2.05-3.08; Koc : 25.4-204; Half-life (hr) air : 0.24-42; Half-life (hr) H₂O surface water : 24-672; Half-life (hr) H₂O ground : 336-8640; Half-life (hr) soil : 52-672; Henry's Pa m³/mol : 637-879; Henry's atm m³/mol - 7.68E-03; BOD 5 if unstated - 1.4,1%; COD - 2.56,13% ThOD - 3.125 : BCF : 23; log BCF : 1.17-2.41.

Environmental Fate: Most xylenes released to the environment will occur in the atmosphere and volatilisation is the dominant environmental fate process. Soil - Xylenes are expected to have moderate mobility in soil evaporating rapidly from soil surfaces. The extent of the degradation is expected to depend on its concentration, residence time in the soil, the nature of the soil, and whether resident microbial populations have been acclimated. Xylene can remain below the soil surface for several days and may travel through the soil profile and enter groundwater. Soil and water microbes may transform it into other, less harmful compounds, although this happens slowly. It is not clear how long xylene remains trapped deep underground in soil or groundwater, but it may be months or years.

Atmospheric Fate: Xylene evaporates quickly into the air from surface soil and water and can remain in the air for several days until it is broken down by sunlight into other less harmful chemicals. In the ambient atmosphere, xylenes are expected to exist solely in the vapour phase. Xylenes are degraded in the atmosphere with an estimated atmospheric lifetime of about 0.5 to 2 days. Xylene

may contribute to photochemical smog formation. p-Xylene has a moderately high photochemical reactivity under smog conditions, higher than the other xylene isomers. The photooxidation of p-xylene results in the production of carbon monoxide, formaldehyde, glyoxal, methylglyoxal, 3-methylbenzyl nitrate, m-tolualdehyde, 4-nitro-3-xylene, 5-nitro-3-xylene, 2,6-dimethyl-p-benzoquinone, 2,4-dimethylphenol, 6-nitro-2,4-dimethylphenol, 2,6-dimethylphenol, and 4-nitro-2,6-dimethylphenol.

Aquatic Fate: p-xylene may adsorb to suspended solids and sediment in water and is expected to volatilise from water surfaces. Estimated volatilisation half-lives for a model river and model lake are 3 hours and 4 days, respectively. Measurements taken from goldfish, eels and clams indicate that bioconcentration in aquatic organisms is low. Photo-oxidation in the presence of humic acids may play an important role in the abiotic degradation of p-xylene. p-Xylene is biodegradable and has been observed to degrade in pond water however; it is unclear if it degrades in surface waters. p-Xylene has been observed to degrade in anaerobic and aerobic groundwater; however, it is known to persist for many years in groundwater, at least at sites where the concentration might have been quite high. Ecotoxicity: Xylenes are slightly toxic to fathead minnow, rainbow trout and bluegill and not acutely toxic to water fleas. For Photobacterium phosphoreum EC50 (24 h): 0.0084 mg/L. and Gammarus lacustris LC50 (48 h): 0.6 mg/L.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
titanium dioxide	HIGH	HIGH
silica amorphous	LOW	LOW
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
bisphenol A/ diglycidyl ether resin, liquid	HIGH	HIGH
methyl isobutyl ketone	HIGH (Half-life = 7001 days)	LOW (Half-life = 1.9 days)
1,3,5-trimethyl benzene	HIGH	HIGH
1,2,4-trimethyl benzene	LOW (Half-life = 56 days)	LOW (Half-life = 0.67 days)
1,2,3-trimethyl benzene	HIGH	HIGH
isopropyl benzene - cumene	HIGH	HIGH
propylbenzene	HIGH	HIGH
propylene glycol monomethyl ether acetate, alpha-isomer	LOW	LOW
phosphoric acid	HIGH	HIGH
n-butanol	LOW (Half-life = 54 days)	LOW (Half-life = 3.65 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
titanium dioxide	LOW (BCF = 10)
silica amorphous	LOW (LogKOW = 0.5294)
xylene	MEDIUM (BCF = 740)
bisphenol A/ diglycidyl ether resin, liquid	LOW (LogKOW = 2.6835)
methyl isobutyl ketone	LOW (LogKOW = 1.31)
1,3,5-trimethyl benzene	LOW (BCF = 342)
1,2,4-trimethyl benzene	LOW (BCF = 275)
1,2,3-trimethyl benzene	LOW (BCF = 259)
isopropyl benzene - cumene	LOW (BCF = 35.5)
propylbenzene	LOW (LogKOW = 3.69)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW (LogKOW = 0.56)
phosphoric acid	LOW (LogKOW = -0.7699)
n-butanol	LOW (BCF = 0.64)

Mobility in soil

Ingredient	Mobility
titanium dioxide	LOW (KOC = 23.74)
silica amorphous	LOW (KOC = 23.74)
bisphenol A/ diglycidyl ether resin, liquid	LOW (KOC = 51.43)
methyl isobutyl ketone	LOW (KOC = 10.91)
1,3,5-trimethyl benzene	LOW (KOC = 703)
1,2,4-trimethyl benzene	LOW (KOC = 717.6)
1,2,3-trimethyl benzene	LOW (KOC = 732.5)
isopropyl benzene - cumene	LOW (KOC = 817.2)
propylbenzene	LOW (KOC = 955)
propylene glycol monomethyl ether acetate, alpha-isomer	HIGH (KOC = 1.838)
phosphoric acid	HIGH (KOC = 1)
n-butanol	MEDIUM (KOC = 2.443)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	<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 licenced to accept chemical and / or pharmaceutical wastes or Incineration in a licenced 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
HAZCHEM	•3Y

Land transport (ADG)

UN number	1263				
UN proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)				
Transport hazard class(es)	<table border="0"> <tr> <td>Class</td> <td>3</td> </tr> <tr> <td>Subrisk</td> <td>Not Applicable</td> </tr> </table>	Class	3	Subrisk	Not Applicable
Class	3				
Subrisk	Not Applicable				
Packing group	III				
Environmental hazard	Not Applicable				
Special precautions for user	<table border="0"> <tr> <td>Special provisions</td> <td>163 223 367</td> </tr> <tr> <td>Limited quantity</td> <td>5 L</td> </tr> </table>	Special provisions	163 223 367	Limited quantity	5 L
Special provisions	163 223 367				
Limited quantity	5 L				

Air transport (ICAO-IATA / DGR)

UN number	1263														
UN proper shipping name	Paint (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base); Paint related material (including paint thinning or reducing compounds)														
Transport hazard class(es)	<table border="0"> <tr> <td>ICAO/IATA Class</td> <td>3</td> </tr> <tr> <td>ICAO / IATA Subrisk</td> <td>Not Applicable</td> </tr> <tr> <td>ERG Code</td> <td>3L</td> </tr> </table>	ICAO/IATA Class	3	ICAO / IATA Subrisk	Not Applicable	ERG Code	3L								
ICAO/IATA Class	3														
ICAO / IATA Subrisk	Not Applicable														
ERG Code	3L														
Packing group	III														
Environmental hazard	Not Applicable														
Special precautions for user	<table border="0"> <tr> <td>Special provisions</td> <td>A3 A72 A192</td> </tr> <tr> <td>Cargo Only Packing Instructions</td> <td>366</td> </tr> <tr> <td>Cargo Only Maximum Qty / Pack</td> <td>220 L</td> </tr> <tr> <td>Passenger and Cargo Packing Instructions</td> <td>355</td> </tr> <tr> <td>Passenger and Cargo Maximum Qty / Pack</td> <td>60 L</td> </tr> <tr> <td>Passenger and Cargo Limited Quantity Packing Instructions</td> <td>Y344</td> </tr> <tr> <td>Passenger and Cargo Limited Maximum Qty / Pack</td> <td>10 L</td> </tr> </table>	Special provisions	A3 A72 A192	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
Special provisions	A3 A72 A192														
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Passenger and Cargo Packing Instructions	355														
Passenger and Cargo Maximum Qty / Pack	60 L														
Passenger and Cargo Limited Quantity Packing Instructions	Y344														
Passenger and Cargo Limited Maximum Qty / Pack	10 L														

Sea transport (IMDG-Code / GGVSee)

UN number	1263
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Carboguard 635HAR Part A

UN proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac solutions, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)	
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	163 223 367 955
	Limited Quantities	5 L

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

ALUMINIUM HYDROXIDE(21645-51-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Inventory of Chemical Substances (AICS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

TITANIUM DIOXIDE(13463-67-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Inventory of Chemical Substances (AICS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

SILICA AMORPHOUS(7631-86-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

SILICA CRYSTALLINE - QUARTZ(14808-60-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

XYLENE(1330-20-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

BISPHENOL A/ BISPHENOL A DIGLYCIDYL ETHER POLYMER(25036-25-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

BARIUM SULFATE(7727-43-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Inventory of Chemical Substances (AICS)

BISPHENOL A/ DIGLYCIDYL ETHER RESIN, LIQUID(25068-38-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

TALC(14807-96-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

METHYL ISOBUTYL KETONE(108-10-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

HYDROCARBON RESIN, POSTPOLYMERISED WITH MALEIC ANHYDRIDE(64742-16-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

VINYL CHLORIDE/ VINYL ACETATE/ HYDROXYPROPYL ACRYLATE(39317-41-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

TALLOW ALKYL DIMETHYLAMMONIUM CHLORIDE/ BENTONITE COMPLEX(71011-25-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

1,3,5-TRIMETHYL BENZENE(108-67-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

1,2,4-TRIMETHYL BENZENE(95-63-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

1,2,3-TRIMETHYL BENZENE(526-73-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

ISOPROPYL BENZENE - CUMENE(98-82-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

PROPYLBENZENE(103-65-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER(108-65-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT(64742-95-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

PHOSPHORIC ACID(7664-38-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

SILICA AMORPHOUS, FUMED, CRYSTALLINE FREE(112945-52-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

C.I. PIGMENT YELLOW 42(51274-00-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

N-BUTANOL(71-36-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

C.I. PIGMENT BLACK 11(1317-61-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

CARBON BLACK(1333-86-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (propylene glycol monomethyl ether acetate, alpha-isomer; n-butanol; hydrocarbon resin, postpolymerised with maleic anhydride; vinyl chloride/ vinyl acetate/ hydroxypropyl acrylate; talc; silica amorphous, fumed, crystalline free; tallow alkylidimethylammonium chloride/ bentonite complex; xylene; phosphoric acid; naphtha petroleum, light aromatic solvent; bisphenol A/ diglycidyl ether resin, liquid; silica crystalline - quartz; bisphenol A/ bisphenol A diglycidyl ether polymer; methyl isobutyl ketone; C.I. Pigment Black 11; 1,3,5-trimethyl benzene; 1,2,3-trimethyl benzene; propylbenzene; isopropyl benzene - cumene; 1,2,4-trimethyl benzene; aluminium hydroxide; carbon black; barium sulfate; C.I. Pigment Yellow 42)
China - IECSC	N (vinyl chloride/ vinyl acetate/ hydroxypropyl acrylate)
Europe - EINEC / ELINCS / NLP	N (vinyl chloride/ vinyl acetate/ hydroxypropyl acrylate; silica amorphous, fumed, crystalline free; bisphenol A/ bisphenol A diglycidyl ether polymer)
Japan - ENCS	N (vinyl chloride/ vinyl acetate/ hydroxypropyl acrylate; silica amorphous, fumed, crystalline free; tallow alkylidimethylammonium chloride/ bentonite complex; bisphenol A/ diglycidyl ether resin, liquid; bisphenol A/ bisphenol A diglycidyl ether polymer)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	N (silica amorphous, fumed, crystalline free)
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION**Other information****Ingredients with multiple cas numbers**

Name	CAS No
aluminium hydroxide	21645-51-2, 1330-44-5, 1302-29-0, 12252-70-9, 51330-22-4
titanium dioxide	13463-67-7, 1317-70-0, 1317-80-2, 12188-41-9, 1309-63-3, 100292-32-8, 101239-53-6, 116788-85-3, 12000-59-8, 12701-76-7, 12767-65-6, 12789-63-8, 1344-29-2, 185323-71-1, 185828-91-5, 188357-76-8, 188357-79-1, 195740-11-5, 221548-98-7, 224963-00-2, 246178-32-5, 252962-41-7, 37230-92-5, 37230-94-7, 37230-95-8, 37230-96-9, 39320-58-6, 39360-64-0, 39379-02-7, 416845-43-7, 494848-07-6, 494848-23-6, 494851-77-3, 494851-98-8, 55068-84-3, 55068-85-4, 552316-51-5, 62338-64-1, 767341-00-4, 97929-50-5, 98084-96-9
silica amorphous	7631-86-9, 112945-52-5, 67762-90-7, 68611-44-9, 68909-20-6, 112926-00-8, 61790-53-2, 60676-86-0, 91053-39-3, 69012-64-2, 844491-94-7
silica crystalline - quartz	14808-60-7, 122304-48-7, 122304-49-8, 12425-26-2, 1317-79-9, 70594-95-5, 87347-84-0
barium sulfate	7727-43-7, 13462-86-7

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bisphenol A/ diglycidyl ether resin, liquid	25068-38-6, 25085-99-8
propylene glycol monomethyl ether acetate, alpha-isomer	108-65-6, 84540-57-8, 142300-82-1
naphtha petroleum, light aromatic solvent	64742-95-6., 25550-14-5.
phosphoric acid	7664-38-2, 16271-20-8
silica amorphous, fumed, crystalline free	112945-52-5, 67256-35-3
C.I. Pigment Yellow 42	51274-00-1, 12259-21-1, 105478-30-6, 53028-10-7, 1342-51-4, 12000-32-7, 50641-37-7, 51109-85-4, 99241-66-4, 131462-81-2, 147625-38-5, 12001-03-5, 185464-57-7, 182761-12-2, 94809-98-0, 934248-40-5
C.I. Pigment Black 11	1317-61-9, 12227-89-3

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

PC – TWA: Permissible Concentration-Time Weighted Average
 PC – STEL: Permissible Concentration-Short Term Exposure Limit
 IARC: International Agency for Research on Cancer
 ACGIH: American Conference of Governmental Industrial Hygienists
 STEL: Short Term Exposure Limit
 TEEL: Temporary Emergency Exposure Limit,
 IDLH: Immediately Dangerous to Life or Health Concentrations
 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

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 TEL (+61 3) 9572 4700.

Carboguard 635 Part B

RESENE PAINTS AUSTRALIA

Chemwatch Hazard Alert Code: 4

Version No: 3.9
Safety Data Sheet according to WHS and ADG requirements

Issue Date: 15/01/2015
Print Date: 08/02/2017
S.GHS.AUS.EN

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

Product Identifier

Product name	Carboguard 635 Part B
Synonyms	Not Available
Proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
Other means of identification	Not Available

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

Relevant identified uses	Part B of a two pack epoxy coating
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Details of the supplier of the safety data sheet

Registered company name	RESENE PAINTS AUSTRALIA
Address	7 Production Ave, Molendinar QLD 4214 Australia
Telephone	+61 7 55126600
Fax	+61 7 55126697
Website	Not Available
Email	Not Available

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	131126
Other emergency telephone numbers	Not Available

CHEMWATCH EMERGENCY RESPONSE

Primary Number	Alternative Number 1	Alternative Number 2
1800 039 008	1800 039 008	+612 9186 1132

Once connected and if the message is not in your preferred language then please dial 01


SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

Poisons Schedule	Not Applicable
Classification [1]	Flammable Liquid Category 3, Respiratory Sensitizer Category 1A, Serious Eye Damage Category 1, Skin Corrosion/Irritation Category 2, Specific target organ toxicity - repeated exposure Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HSIS; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

Label elements

GHS label elements	
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SIGNAL WORD	DANGER
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Hazard statement(s)

H226	Flammable liquid and vapour.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H318	Causes serious eye damage.

Continued...

Carboguard 635 Part B

H315	Causes skin irritation.
H373	May cause damage to organs through prolonged or repeated exposure.

Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P210	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
P233	Keep container tightly closed.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P285	In case of inadequate ventilation wear respiratory 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.

Precautionary statement(s) Response

P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P310	Immediately call a POISON CENTER or doctor/physician.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER or doctor/physician.
P362	Take off contaminated clothing and wash before reuse.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam for extinction.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P303+P361+P353	IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower.
P332+P313	If skin irritation occurs: Get medical advice/attention.

Precautionary statement(s) Storage

P403+P235	Store in a well-ventilated place. Keep cool.
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Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.
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SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
55353-47-4	0	<u>castor oil, hydrogenated, modified</u>
1330-20-7	10-20	<u>xylene</u>
71-36-3	1-10	<u>n-butanol</u>
107-15-3	<=1	<u>ethylenediamine</u>
14808-60-7	20-30	<u>silica crystalline - quartz</u>
95-63-6	<=1	<u>1,2,4-trimethyl benzene</u>
90-72-2	1-10	<u>2,4,6-tris(dimethylamino)methylphenol</u>

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"> ▶ Immediately hold eyelids apart and flush the eye continuously with 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. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ 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.

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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.
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. ▶ 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.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

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 acute or short term repeated exposures to xylene:

- ▶ Gastro-intestinal absorption is significant with ingestions. For ingestions exceeding 1-2 ml (xylene)/kg, intubation and lavage with cuffed endotracheal tube is recommended. The use of charcoal and cathartics is equivocal.
- ▶ Pulmonary absorption is rapid with about 60-65% retained at rest.
- ▶ Primary threat to life from ingestion and/or inhalation, is respiratory failure.
- ▶ Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO₂ < 50 mm Hg or pCO₂ > 50 mm Hg) should be intubated.
- ▶ Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- ▶ A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- ▶ Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.

BIOLOGICAL EXPOSURE INDEX - BEI

These represent the determinants observed in specimens collected from a healthy worker exposed at the Exposure Standard (ES or TLV):

Determinant	Index	Sampling Time	Comments
Methylhippu-ric acids in urine	1.5 gm/gm creatinine 2 mg/min	End of shift Last 4 hrs of shift	

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

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

Special hazards arising from the substrate or mixture

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

Fire Fighting	<ul style="list-style-type: none"> ▶ 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. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ If safe, switch off electrical equipment until vapour fire hazard removed. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ Avoid spraying water onto liquid pools. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Liquid and vapour are flammable. ▶ Moderate fire hazard when exposed to heat or flame. ▶ Vapour forms an explosive mixture with air. ▶ Moderate explosion hazard when exposed to heat or flame. ▶ Vapour may travel a considerable distance to source of ignition. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). <p>Combustion products include:</p> <ul style="list-style-type: none"> , carbon dioxide (CO₂) , carbon monoxide (CO) , sulfur oxides (SO_x) , silicon dioxide (SiO₂)

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	other pyrolysis products typical of burning organic material.
HAZCHEM	*3Y

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	<ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Consider evacuation (or protect in place). ▶ No smoking, naked lights or ignition sources. ▶ Increase ventilation. ▶ Stop leak if safe to do so. ▶ Water spray or fog may be used to disperse /absorb vapour. ▶ Contain spill with sand, earth or vermiculite. ▶ Use only spark-free shovels and explosion proof equipment. ▶ Collect recoverable product into labelled containers for recycling. ▶ Absorb remaining product with sand, earth or vermiculite. ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<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. ▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ Electrostatic discharge may be generated during pumping - this may result in fire. ▶ Ensure electrical continuity by bonding and grounding (earthing) all equipment. ▶ Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (<=1 m/sec until fill pipe submerged to twice its diameter, then <= 7 m/sec). ▶ Avoid splash filling. ▶ Do NOT use compressed air for filling discharging or handling operations. ▶ 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.
Other information	<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

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- ▶ detectors.
 - ▶ Keep adsorbents for leaks and spills readily available.
 - ▶ Protect containers against physical damage and check regularly for leaks.
 - ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
- In addition, for tank storages (where appropriate):
- ▶ Store in grounded, properly designed and approved vessels and away from incompatible materials.
 - ▶ For bulk storages, consider use of floating roof or nitrogen blanketed vessels; where venting to atmosphere is possible, equip storage tank vents with flame arrestors; inspect tank vents during winter conditions for vapour/ ice build-up.
 - ▶ Storage tanks should be above ground and diked to hold entire contents.

Conditions for safe storage, including any incompatibilities

Suitable container	<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.
Storage incompatibility	<p>Barium sulfate (barytes)</p> <ul style="list-style-type: none"> ▶ reacts violently with dimethyl sulfoxide, sodium acetylide, finely divided carbon, aluminium, magnesium, zirconium, and possibly other active metals, especially at elevated temperatures ▶ is incompatible with potassium, phosphorus (ignites when primed with nitrate-calcium silicide) <p>Xylenes:</p> <ul style="list-style-type: none"> ▶ may ignite or explode in contact with strong oxidisers, 1,3-dichloro-5,5-dimethylhydantoin, uranium fluoride ▶ attack some plastics, rubber and coatings ▶ may generate electrostatic charges on flow or agitation due to low conductivity. ▶ 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. <p>Silicas:</p> <ul style="list-style-type: none"> ▶ react with hydrofluoric acid to produce silicon tetrafluoride gas ▶ react with xenon hexafluoride to produce explosive xenon trioxide ▶ reacts exothermically with oxygen difluoride, and explosively with chlorine trifluoride (these halogenated materials are not commonplace industrial materials) and other fluorine-containing compounds ▶ may react with fluorine, chlorates ▶ are incompatible with strong oxidisers, manganese trioxide, chlorine trioxide, strong alkalis, metal oxides, concentrated orthophosphoric acid, vinyl acetate ▶ may react vigorously when heated with alkali carbonates. <p>For alkyl aromatics:</p> <p>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>



+ X X X + + +

- X** — Must not be stored together
O — May be stored together with specific preventions
+ — May be stored together

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	xylene	Xylene (o-, m-, p- isomers)	350 mg/m ³ / 80 ppm	655 mg/m ³ / 150 ppm	Not Available	Not Available
Australia Exposure Standards	n-butanol	n-Butyl alcohol	Not Available	Not Available	152 mg/m ³ / 50 ppm	Sk
Australia Exposure Standards	ethylenediamine	Ethylenediamine	25 mg/m ³ / 10 ppm	Not Available	Not Available	Sen

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Australia Exposure Standards	silica crystalline - quartz	Silica - Crystalline: Quartz (respirable dust) / Quartz (respirable dust)	0.1 mg/m3	Not Available	Not Available	Not Available
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EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
xylene	Xylenes	Not Available	Not Available	Not Available
n-butanol	Butyl alcohol, n-; (n-Butanol)	60 ppm	800 ppm	8000 ppm
ethylenediamine	Ethylenediamine, 1,2-	0.88 ppm	Not Available	Not Available
silica crystalline - quartz	Silica, crystalline-quartz; (Silicon dioxide)	0.075 mg/m3	33 mg/m3	200 mg/m3
1,2,4-trimethyl benzene	Permafluor E+	140 mg/m3	360 mg/m3	2,200 mg/m3
1,2,4-trimethyl benzene	Trimethylbenzene, 1,2,4-; (Pseudocumene)	Not Available	Not Available	480 ppm
2,4,6-tris[(dimethylamino)methyl]phenol	Tris(dimethylaminomethyl)phenol, 2,4,6-	3.6 mg/m3	40 mg/m3	240 mg/m3

Ingredient	Original IDLH	Revised IDLH
castor oil, hydrogenated, modified	Not Available	Not Available
xylene	1,000 ppm	900 ppm
n-butanol	8,000 ppm	1,400 [LEL] ppm
ethylenediamine	2,000 ppm	1,000 ppm
silica crystalline - quartz	N.E. mg/m3 / N.E. ppm	50 mg/m3
1,2,4-trimethyl benzene	Not Available	Not Available
2,4,6-tris[(dimethylamino)methyl]phenol	Not Available	Not Available

Exposure controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are:
 Process controls which involve changing the way a job activity or process is done to reduce the risk.
 Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
 Employers may need to use multiple types of controls to prevent employee overexposure.

For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant.
 Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Personal protection

Eye and face protection

- ▶ Safety glasses with side shields.
- ▶ Chemical goggles.
- ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be

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	<ul style="list-style-type: none"> ▶ 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 <p>NOTE:</p> <ul style="list-style-type: none"> ▶ 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. <p>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.</p> <p>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.</p> <p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturizer is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> • frequency and duration of contact, • chemical resistance of glove material, • glove thickness and • dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> • 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. <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>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.</p> <p>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.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> • 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 <p>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.</p>
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ 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.
Thermal hazards	Not Available

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:

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Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE	C

Respiratory protection

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	BKAX-AUS P2	-	BKAX-PAPR-AUS / Class 1 P2
up to 50 x ES	-	BKAX-AUS / Class 1 P2	-
up to 100 x ES	-	BKAX-2 P2	BKAX-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)

If inhalation risk above the TLV exists, wear approved dust respirator.

Use respirators with protection factors appropriate for the exposure level.

- ▶ Up to 5 X TLV, use valveless mask type; up to 10 X TLV, use 1/2 mask dust respirator
- ▶ Up to 50 X TLV, use full face dust respirator or demand type C air supplied respirator
- ▶ Up to 500 X TLV, use powered air-purifying dust respirator or a Type C pressure demand

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PE/EVAL/PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
SARANEX-23	C
TEFLON	C
VITON	C

- ▶ supplied-air respirator
- ▶ Over 500 X TLV wear full-face self-contained breathing apparatus with positive pressure mode or a combination respirator with a Type C positive pressure supplied-air full-face respirator and an auxiliary self-contained breathing apparatus operated in pressure demand or other positive pressure mode

Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content. The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.

* 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.

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Coloured with Characteristic Odour		
Physical state	Liquid	Relative density (Water = 1)	1.45
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	460
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	134	Molecular weight (g/mol)	Not Available
Flash point (°C)	29	Taste	Not Available
Evaporation rate	0.7 BuAC = 1	Explosive properties	Not Available
Flammability	Flammable.	Oxidising properties	Not Available
Upper Explosive Limit (%)	8.4	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	1.1	Volatile Component (%vol)	20
Vapour pressure (kPa)	0.65	Gas group	Not Available
Solubility in water (g/L)	Immiscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	3.5	VOC g/L	Not Available

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.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	<p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. The material is not thought to produce respiratory irritation (as classified by EC Directives using animal models). Nevertheless inhalation of vapours, fumes or aerosols, especially for prolonged periods, may produce respiratory discomfort and occasionally, distress. The acute toxicity of inhaled alkylbenzenes is best described by central nervous system depression. As a rule, these compounds may also act as general anaesthetics.</p> <p>Systemic poisoning produced by general anaesthesia is characterised by lightheadedness, nervousness, apprehension, euphoria, confusion, dizziness, drowsiness, tinnitus, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness and respiratory depression and arrest. Cardiac arrest may result from cardiovascular collapse. Bradycardia, and hypotension may also be produced. Inhaled alkylbenzene vapours cause death in animals at air levels that are relatively similar (typically LC50s are in the range 5000 -8000 ppm for 4 to 8 hour exposures). It is likely that acute inhalation exposure to alkylbenzenes resembles that to general anaesthetics.</p> <p>Alkylbenzenes are not generally toxic other than at high levels of exposure. This may be because their metabolites have a low order of toxicity and are easily</p>
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	<p>excreted. There is little or no evidence to suggest that metabolic pathways can become saturated leading to spillover to alternate pathways. Nor is there evidence that toxic reactive intermediates, which may produce subsequent toxic or mutagenic effects, are formed</p> <p>On exposure to mixed trimethylbenzenes, some people may become nervous, tensed, anxious and have difficult breathing. There may be a reduction red blood cells and bleeding abnormalities. There may also be drowsiness.</p> <p>Headache, fatigue, tiredness, irritability and digestive disturbances (nausea, loss of appetite and bloating) are the most common symptoms of xylene overexposure. Injury to the heart, liver, kidneys and nervous system has also been noted amongst workers.</p> <p>Xylene is a central nervous system depressant</p>
Ingestion	<p>The material is not thought to produce adverse health effects following ingestion (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum.</p> <p>Ingestion of soluble barium compounds may result in ulceration of the mucous membranes of the gastrointestinal tract, tightness in the muscles of the face and neck, gastroenteritis, vomiting, diarrhoea, muscular tremors and paralysis, anxiety, weakness, laboured breathing, cardiac irregularity due to contractions of smooth striated and cardiac muscles (often violent and painful), slow irregular pulse, hypertension, convulsions and respiratory failure.</p> <p>Swallowing of the liquid may cause aspiration into the lungs with the risk of chemical pneumonitis; serious consequences may result. (ICSC13733)</p>
Skin Contact	<p>Skin contact with the material may be harmful; systemic effects may result following absorption.</p> <p>This material can cause inflammation of the skin on contact in some persons.</p> <p>The material may accentuate any pre-existing dermatitis condition</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 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>
Eye	<p>If applied to the eyes, this material causes severe eye damage.</p>
Chronic	<p>Skin contact with the material is more likely to cause a sensitisation reaction in some persons compared to the general population.</p> <p>Harmful: danger of serious damage to health by prolonged exposure through inhalation.</p> <p>This material can cause serious damage if one is exposed to it for long periods. It can be assumed that it contains a substance which can produce severe defects.</p> <p>Based on experience with animal studies, exposure to the material may result in toxic effects to the development of the foetus, at levels which do not cause significant toxic effects to the mother.</p> <p>Substance accumulation, in the human body, may occur and may cause some concern following repeated or long-term occupational exposure.</p> <p>Crystalline silicas activate the inflammatory response of white blood cells after they injure the lung epithelium. Chronic exposure to crystalline silicas reduces lung capacity and predisposes to chest infections.</p> <p>There has been some concern that this material can cause cancer or mutations but there is not enough data to make an assessment.</p> <p>Women exposed to xylene in the first 3 months of pregnancy showed a slightly increased risk of miscarriage and birth defects. Evaluation of workers chronically exposed to xylene has demonstrated lack of genetic toxicity.</p>

Carboguard 635 Part B	TOXICITY	IRRITATION
	Not Available	Not Available
castor oil, hydrogenated, modified	TOXICITY	IRRITATION
	Not Available	Not Available
xylene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1700 mg/kg ^[2]	Eye (human): 200 ppm irritant
	Inhalation (rat) LC50: 5000 ppm/4hr ^[2]	Eye (rabbit): 5 mg/24h SEVERE
	Oral (rat) LD50: 4300 mg/kg ^[2]	Eye (rabbit): 87 mg mild
		Skin (rabbit):500 mg/24h moderate
n-butanol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 3434.4 mg/kg ^[1]	Eye (human): 50 ppm - irritant
	Inhalation (rat) LC50: 24 mg/L/4hr ^[2]	Eye (rabbit): 1.6 mg-SEVERE
	Inhalation (rat) LC50: 8000 ppm/4hr ^[2]	Eye (rabbit): 24 mg/24h-SEVERE
		Skin (rabbit): 405 mg/24h-moderate
ethylenediamine	TOXICITY	IRRITATION
	dermal (rat) LD50: ca.1000 mg/kg ^[1]	Eye (rabbit):0.67 mg SEVERE
	Inhalation (mouse) LC50: 0.3 mg/L/4hr ^[2]	Eye (rabbit):0.75mg/24h SEVERE
	Oral (rat) LD50: 500 mg/kg ^[2]	Skin(rabbit):10 mg/24h open SEVERE
		Skin(rabbit):450 mg open moderate
silica crystalline - quartz	TOXICITY	IRRITATION
	Not Available	Not Available
1,2,4-trimethyl benzene	TOXICITY	IRRITATION
	dermal (rat) LD50: 3504 mg/kg ^[1]	Not Available
	Inhalation (rat) LC50: 18 mg/L/4hr ^[2]	
	Oral (rat) LD50: ca.3504 mg/kg ^[1]	
2,4,6-tris(dimethylamino)methylphenol	TOXICITY	IRRITATION
	dermal (rat) LD50: >973 mg/kg ^[1]	Eye (rabbit): 0.05 mg/24h - SEVERE

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Inhalation (rat) LC50: >0.5 mg/l/1 hr^[2]

Skin (rabbit): 2 mg/24h - SEVERE

Oral (rat) LD50: 1200 mg/kg^[2]

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

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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 NOAEL for systemic and/or neurotoxicity was 6,500 mg/m³, the highest concentration tested. In an inhalation study of a commercial blend, rats were exposed to C9 aromatic naphtha concentrations of 0, 96, 198, or 373 ppm (0, 470, 970, 1830 mg/m³) for 6 hr/day, 5 days/week, for 12 months. Liver and kidney weights were increased in the high exposure group but no accompanying histopathology was observed in these organs. The NOAEL was considered to be the high exposure level of 373 ppm, or 1830 mg/m³. In two subchronic rat inhalation studies, both of three months duration, rats were exposed to the individual TMB isomers (1,2,4- and 1,3,5-) to nominal concentrations of 0, 25, 100, or 250 ppm (0, 123, 492, or 1230 mg/m³). Respiratory irritation was observed at 492 (100 ppm) and 1230 mg/m³ (250 ppm) and no systemic toxicity was observed in either study. For both pure isomers, the NOELs are 25 ppm or 123 mg/m³ for respiratory irritation and 250 ppm or 1230 mg/m³ for systemic effects.

Oral: The C9 aromatic naphtha has not been tested via the oral route of exposure. Individual TMB isomers have been evaluated in a series of repeated-dose oral studies ranging from 14 days to 3 months over a wide range of doses. The effects observed in these studies included increased liver and kidney weights, changes in blood chemistry, increased salivation, and decreased weight gain at higher doses. Organ weight changes appeared to be adaptive as they were not accompanied by histopathological effects. Blood changes appeared sporadic and without pattern. One study reported hyaline droplet nephropathy in male rats at the highest dose (1000 mg/kg bw-day), an effect that is often associated with alpha-2mu-globulin-induced nephropathy and not considered relevant to humans. The doses at which effects were detected were 100 mg/kg-bw day or above (an exception was the pilot 14 day oral study - LOAEL 150 mg/kg bw-day - but the follow up three month study had a LOAEL of 600 mg/kg-bw-day with a NOAEL of 200 mg/kg bw-day). Since effects generally were not severe and could be considered adaptive or spurious, oral exposure does not appear to pose a high toxicity hazard for pure trimethylbenzene isomers.

Mutagenicity

In vitro genotoxicity testing of a variety of C9 aromatics has been conducted in both bacterial and mammalian cells. In vitro point mutation tests were conducted with Salmonella typhimurium and Escherichia coli bacterial strains, as well as with cultured mammalian cells such as the Chinese hamster cell ovary cells (HGPRT assay) with and without metabolic activation. In addition, several types of in vitro chromosomal aberration tests have been performed (chromosome aberration frequency in Chinese hamster ovary and lung cells, sister chromatid exchange in CHO cells). Results were negative both with and without metabolic activation for all category members. For the supporting chemical 1,2,3-TMB, a single in vitro chromosome aberration test was weakly positive. In in vivo bone marrow cytogenetics test, rats were exposed to C9 aromatic naphtha at concentrations of 0, 153, 471, or 1540 ppm (0, 750, 2,310, or 7,560 mg/m³) 6 hr/day, for 5 days. No evidence of in vivo somatic cell 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

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	<p>excessive mortality.</p> <p>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.</p> <p>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.</p>
<p>CASTOR OIL, HYDROGENATED, MODIFIED</p>	<p>This product contains partially hydrogenated fatty acids and/ or trans fatty acids.</p> <p>The consumption of trans fats increases the risk of coronary heart disease by raising levels of LDL cholesterol and lowering levels of "good" HDL cholesterol. There is an ongoing debate about a possible differentiation between trans fats of natural origin and trans fats of man-made origin but so far no scientific consensus has been found. Two Canadian studies have shown that the natural trans fat vaccenic acid, found in beef and dairy products, may have an opposite health effect and could actually be beneficial compared to hydrogenated vegetable shortening, or a mixture of pork lard and soy fat, by lowering total and LDL cholesterol and triglyceride levels. In lack of recognized evidence and scientific agreement, nutritional authorities consider all trans fats as equally harmful for health and recommend that consumption of trans fats be reduced to trace amounts.</p> <p>The use of hydrogenated oils in foods has never been completely satisfactory. Because the center arm of the triglyceride is shielded somewhat by the end fatty acids, most of the hydrogenation occurs on the end fatty acids.</p> <p>While full hydrogenation produces largely saturated fatty acids, partial hydrogenation results in the transformation of unsaturated cis fatty acids to trans fatty acids in the oil mixture due to the heat used in hydrogenation. Partially hydrogenated oils and their trans fats have increasingly been viewed as "unhealthy".</p> <p>Trans fat is the common name for unsaturated fat with trans-isomer (E-isomer) fatty acid(s). Because the term refers to the configuration of a double carbon-carbon bond, trans fats are sometimes monounsaturated or polyunsaturated, but never saturated. Trans fats do exist in nature but also occur during the processing of polyunsaturated fatty acids in food production. Trans fats occur naturally in a limited number of cases: vaccenyl and conjugated linoleyl (CLA) containing trans fats occur naturally in trace amounts in meat and dairy products from ruminants.</p> <p>The exact biochemical methods by which trans fats produce specific health problems are a topic of continuing research. One theory is that the human lipase enzyme works only on the cis configuration and cannot metabolise a trans fat. A lipase is a water-soluble enzyme that helps digest, transport, and process dietary lipids such as triglycerides, fats, and oils in most - if not all - living organisms. While the mechanisms through which trans fats contribute to coronary heart disease are fairly well understood, the mechanism for trans fat's effect on diabetes is still under investigation. Trans fatty acids may impair the metabolism of long-chain polyunsaturated fatty acids (LCPUFAs), but maternal pregnancy trans fatty acid intake has been inversely associated with LCPUFAs levels in infants at birth thought to underlie the positive association between breastfeeding and intelligence.</p> <p>There are suggestions that the negative consequences of trans fat consumption go beyond the cardiovascular risk. In general, there is much less scientific consensus asserting that eating trans fat specifically increases the risk of other chronic health problems:</p> <p>It has been suggested that the intake of both trans fats and saturated fats promote the development of Alzheimer disease, although not confirmed in an animal model. It has been found that trans fats impaired memory and learning in middle-age rats. The rats' brains of trans-fat eaters had fewer proteins critical to healthy neurological function. Inflammation in and around the hippocampus, the part of the brain responsible for learning and memory. These are the exact types of changes normally seen at the onset of Alzheimer's, but seen after six weeks, even though the rats were still young.</p> <p>There is a growing concern that the risk of type 2 diabetes increases with trans fat consumption.[52] However, consensus has not been reached. For example, one study found that risk is higher for those in the highest quartile of trans fat consumption. Another study has found no diabetes risk once other factors such as total fat intake and BMI were accounted for.</p> <p>Research indicates that trans fat may increase weight gain and abdominal fat, despite a similar caloric intake. A 6-year experiment revealed that monkeys fed a trans fat diet gained 7.2% of their body weight, as compared to 1.8% for monkeys on a mono-unsaturated fat diet. Although obesity is frequently linked to trans fat in the popular media, this is generally in the context of eating too many calories; there is not a strong scientific consensus connecting trans fat and obesity, although the 6-year experiment did find such a link, concluding that "under controlled feeding conditions, long-term TFA consumption was an independent factor in weight gain. TFAs enhanced intra-abdominal deposition of fat, even in the absence of caloric excess, and were associated with insulin resistance, with evidence that there is impaired post-insulin receptor binding signal transduction.</p> <p>Liver Dysfunction: Trans fats are metabolised differently by the liver than other fats and interfere with delta 6 desaturase. Delta 6 desaturase is an enzyme involved in converting essential fatty acids to arachidonic acid and prostaglandins, both of which are important to the functioning of cells.</p> <p>Infertility in women: One 2007 study found, "Each 2% increase in the intake of energy from trans unsaturated fats, as opposed to that from carbohydrates, was associated with a 73% greater risk of ovulatory infertility...".</p> <p>Major depressive disorder: Spanish researchers analysed the diets of 12,059 people over six years and found those who ate the most trans fats had a 48 per cent higher risk of depression than those who did not eat trans fats. One mechanism may be trans-fats' substitution for docosahexaenoic acid (DHA) levels in the orbitofrontal cortex (OFC). Very high intake of trans-fatty acids (43% of total fat) in mice from 2 to 16 months of age was associated with lowered DHA levels in the brain (p=0.001) When the brains of 15 major depressive subjects who had committed suicide were examined post-mortem and compared against 27 age-matched controls, the suicidal brains were found to have 16% less (male average) to 32% (female average) less DHA in the OFC. The OFC is known to control reward, reward expectation and empathy, which are all negatively impacted in depressive mood disorders, as well as regulating the limbic system-></p>
<p>XYLENE</p>	<p>The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing. Reproductive effector in rats</p>
<p>N-BUTANOL</p>	<p>for n-butanol</p> <p>Acute toxicity: n-Butanol (BA) was only slightly toxic to experimental animals following acute oral, dermal, or inhalation exposure. The acute oral LD50 values for female rats ranged from 790 to 4360 mg/kg. Different strains of rat were used in each of four studies, which may account for the variability. Oral LD50 values for mice, rabbits, hamsters, dogs, and male rats all fell within the same range. The rat inhalation LCO of 8000 ppm (24000 mg/m3) indicates very low inhalation toxicity (no lethality at 8000 ppm). The rabbit dermal LD50 was 3402 mg/kg, indicating that BA can penetrate the skin, but not very readily. Animal experiments and human experience indicate that BA is, at most, moderately irritating to the skin, but it is a severe eye irritant. These effects are most likely due to BA's localised defatting and drying characteristics. Although no animal data are available, human studies and experience show that BA is not likely to be a skin sensitiser.</p> <p>The median odor threshold for BA (0.17 ppm) is well below the lowest nasal irritation threshold in humans (289 ppm), allowing warning of possible chemical exposure prior to nasal irritation occurring. Human studies are complicated by the odor characteristics of the material, as the odor threshold is well below the levels at which irritation is observed.</p> <p>Repeat dose toxicity: An in vivo toxicokinetics study confirmed the rapid metabolism of n-butyl acetate (BAc) to BA. Hydrolysis of BAc in blood and brain was estimated to be 99 percent complete within 2.7 minutes (elimination t1/2 = 0.41 minute). Thus, organisms exposed to BAc can experience appreciable tissue concentrations of BA. In this way, the results of toxicity studies with BAc can be used as supplemental, surrogate data to provide information on the toxicity of BA.</p> <p>A thirteen-week, subchronic exposure to BAc, the metabolic precursor of BA, produced transient hypoactivity (during exposure only) at 1500 and 3000 ppm (7185 and 14370 mg/m3) along with decreased body weight and food consumption, but no post exposure neurotoxicity even at</p>

	<p>3000 ppm. A concurrent subchronic neurotoxicity study under the same exposure conditions showed no evidence of cumulative neurotoxicity based upon functional observational battery endpoints, quantitative motor activity, neuropathology and scheduled-controlled operant behavior endpoints. A no observable effect level (NOAEL) of 500 ppm (2395 mg/m³) was reported for systemic effects in rats, and a NOAEL of 3000 ppm (14370 mg/m³) was reported for post exposure neurotoxicity in rats.</p> <p>Reproductive toxicity: Several studies indicate that BA is not a reproductive toxicant.</p> <p>Female rats exposed to 6000 ppm (18000 mg/m³) BA throughout gestation and male rats exposed to 6000 ppm (18000 mg/m³) BA for six weeks prior to mating showed no effects on fertility or pregnancy rate. Male rats given BA at 533 mg/kg/day for 5 days had no testicular toxicity.</p> <p>Developmental toxicity: BA produced only mild foetotoxicity and developmental alterations at or near the maternally toxic (even lethal) dose of 8000 ppm (24000 mg/m³) throughout gestation.</p> <p>Genotoxicity: An entire battery of negative in vitro tests and a negative in vivo micronucleus test indicate that BA is not genotoxic.</p> <p>Carcinogenicity: Based upon the battery of negative mutagenicity and clastogenicity findings, BA presents a very small potential for carcinogenicity.</p>
ETHYLENEDIAMINE	<p>Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.</p> <p>Acute toxicity of ethylenediamine (EDA) is considered to be low to moderate. In animal testing, it affected the eyes (causing clouding of the lens and atrophy of the retina) and kidneys. EDA is also capable of causing hypersensitivity to the airway and asthma in the work environment, although the levels required for this to occur are not known. EDA is corrosive to the skin and eyes due to its high alkalinity. It sensitises the skin and airways in humans and cross-sensitisation can occur with chemicals that are structurally similar. Weight loss has been reported in animal testing. Evidence generally shows that ethylenediamine is unlikely to cause genetic, developmental or reproductive damage.</p> <p>Acute toxicity of ethylenediamine (LD50, rat, oral range from 637 mg/kg to 1850 mg/kg; LC50, rat, inhalation >29 mg/l and LD50, rabbit, dermal 560 mg/kg) is considered to be low to moderate. Due to the high alkalinity, ethylenediamine is corrosive to the skin and eyes. It is a dermal and respiratory sensitiser in humans and has been reported to cross-sensitize for chemicals of similar structure. In repeat dose studies, decreased body weight along with decreased water and feed consumption were observed. Every attempt was made to minimise the irritating nature of EDA and reduce the pH by using EDA-2HCL. Hepatocellular pleomorphism was noted in every study following dietary administration of longer than 13 weeks duration. Gavage administration resulted in effects in the eyes and kidneys. Kidney effects consisted of degenerative and regenerative changes in the tubular epithelium. The Lowest-Observable-Adverse-Effect -Level (LOAEL) is 100 mg/kg/day with a No-Observable-Effect-Level (NOEL) of 20 mg/kg/day observed in the chronic dietary feeding study. Ethylenediamine was rapidly excreted with most of the material eliminated in the urine within 24 hours. Ethylenediamine has produced weakly positive results, 2-3 times greater than control values, in several Ames tests, which may or may not be related to an impurity. Subsequent studies conducted with purer material were negative. All other tests including several in vitro assays (CHO gene mutation, sister chromatid exchange with CHO cells and UDS with primary rat hepatocytes) and a rat dominant lethal assay were negative. The weight of evidence from both in vitro and in vivo tests indicates that ethylenediamine is unlikely to be genotoxic. In chronic bioassays via two routes of exposure there was no carcinogenic effect. In developmental toxicity studies, growth retardation was noted at maternally toxic levels. However, there was no evidence of developmental toxicity at maternally toxic doses when compared with a pair-fed control. There was no effect on reproductive parameters at levels, which produced parental toxicity.</p>
SILICA CRYSTALLINE - QUARTZ	<p>WARNING: For inhalation exposure <u>ONLY</u>: This substance has been classified by the IARC as Group 1: CARCINOGENIC TO HUMANS</p> <p>The International Agency for Research on Cancer (IARC) has classified occupational exposures to respirable (<5 µm) crystalline silica as being carcinogenic to humans. This classification is based on what IARC considered sufficient evidence from epidemiological studies of humans for the carcinogenicity of inhaled silica in the forms of quartz and cristobalite. Crystalline silica is also known to cause silicosis, a non-cancerous lung disease.</p> <p>Intermittent exposure produces; focal fibrosis, (pneumoconiosis), cough, dyspnoea, liver tumours.</p> <p>* Millions of particles per cubic foot (based on impinger samples counted by light field techniques).</p> <p>NOTE : the physical nature of quartz in the product determines whether it is likely to present a chronic health problem. To be a hazard the material must enter the breathing zone as respirable particles.</p>
1,2,4-TRIMETHYL BENZENE	<p>Other Toxicity data is available for CHEMWATCH 12172 1,2,3-trimethylbenzene CHEMWATCH 2325 1,3,5-trimethylbenzene</p>
2,4,6-TRIS(DIMETHYLAMINO)METHYLPHENOL	<p>While it is difficult to generalise about the full range of potential health effects posed by exposure to the many different amine compounds, characterised by those used in the manufacture of polyurethane and polyisocyanurate foams, it is agreed that overexposure to the majority of these materials may cause adverse health effects.</p> <ul style="list-style-type: none"> ▶ Many amine-based compounds can induce histamine liberation, which, in turn, can trigger allergic and other physiological effects, including bronchoconstriction or bronchial asthma and rhinitis. ▶ Systemic symptoms include headache, nausea, faintness, anxiety, a decrease in blood pressure, tachycardia (rapid heartbeat), itching, erythema (reddening of the skin), urticaria (hives), and facial edema (swelling). Systemic effects (those affecting the body) that are related to the pharmacological action of amines are usually transient. <p>Typically, there are four routes of possible or potential exposure: inhalation, skin contact, eye contact, and ingestion.</p> <p>Inhalation:</p> <p>Inhalation of vapors may, depending upon the physical and chemical properties of the specific product and the degree and length of exposure, result in moderate to severe irritation of the tissues of the nose and throat and can irritate the lungs.</p> <p>Products with higher vapour pressures have a greater potential for higher airborne concentrations. This increases the probability of worker exposure.</p> <p>Higher concentrations of certain amines can produce severe respiratory irritation, characterised by nasal discharge, coughing, difficulty in breathing, and chest pains.</p> <p>Chronic exposure via inhalation may cause headache, nausea, vomiting, drowsiness, sore throat, bronchopneumonia, and possible lung damage. Also, repeated and/or prolonged exposure to some amines may result in liver disorders, jaundice, and liver enlargement. Some amines have been shown to cause kidney, blood, and central nervous system disorders in laboratory animal studies.</p> <p>While most polyurethane amine catalysts are not sensitizers, some certain individuals may also become sensitized to amines and may experience respiratory distress, including asthma-like attacks, whenever they are subsequently exposed to even very small amounts of vapor. Once sensitized, these individuals must avoid any further exposure to amines. Although chronic or repeated inhalation of vapor concentrations below hazardous or recommended exposure limits should not ordinarily affect healthy individuals, chronic overexposure may lead to permanent pulmonary injury, including a reduction in lung function, breathlessness, chronic bronchitis, and immunologic lung disease.</p> <p>Inhalation hazards are increased when exposure to amine catalysts occurs in situations that produce aerosols, mists, or heated vapors. Such situations include leaks in fitting or transfer lines. Medical conditions generally aggravated by inhalation exposure include asthma, bronchitis, and emphysema.</p> <p>Skin Contact:</p> <p>Skin contact with amine catalysts poses a number of concerns. Direct skin contact can cause moderate to severe irritation and injury-i.e., from simple redness and swelling to painful blistering, ulceration, and chemical burns. Repeated or prolonged exposure may also result in severe cumulative dermatitis.</p> <p>Skin contact with some amines may result in allergic sensitization. Sensitized persons should avoid all contact with amine catalysts. Systemic effects resulting from the absorption of the amines through skin exposure may include headaches, nausea, faintness, anxiety, decrease in blood pressure, reddening of the skin, hives, and facial swelling. These symptoms may be related to the pharmacological action of the amines, and they are usually transient.</p>

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	<p>Eye Contact: Amine catalysts are alkaline in nature and their vapours are irritating to the eyes, even at low concentrations. Direct contact with the liquid amine may cause severe irritation and tissue injury, and the "burning" may lead to blindness. (Contact with solid products may result in mechanical irritation, pain, and corneal injury.) Exposed persons may experience excessive tearing, burning, conjunctivitis, and corneal swelling. The corneal swelling may manifest itself in visual disturbances such as blurred or "foggy" vision with a blue tint ("blue haze") and sometimes a halo phenomenon around lights. These symptoms are transient and usually disappear when exposure ceases. Some individuals may experience this effect even when exposed to concentrations below doses that ordinarily cause respiratory irritation.</p> <p>Ingestion: The oral toxicity of amine catalysts varies from moderately to very toxic. Some amines can cause severe irritation, ulceration, or burns of the mouth, throat, esophagus, and gastrointestinal tract. Material aspirated (due to vomiting) can damage the bronchial tubes and the lungs. Affected persons also may experience pain in the chest or abdomen, nausea, bleeding of the throat and the gastrointestinal tract, diarrhea, dizziness, drowsiness, thirst, circulatory collapse, coma, and even death.</p> <p>Polyurethane Amine Catalysts: Guidelines for Safe Handling and Disposal; Technical Bulletin June 2000 Alliance for Polyurethanes Industry</p>
Carboguard 635 Part B & ETHYLENEDIAMINE	<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>
Carboguard 635 Part B & CASTOR OIL, HYDROGENATED, MODIFIED & 2,4,6-TRIS((DIMETHYLAMINO)METHYL)PHENOL	<p>No significant acute toxicological data identified in literature search.</p>
Carboguard 635 Part B & 1,2,4-TRIMETHYL BENZENE	<p>For trimethylbenzenes: Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure. Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal. Following oral administration of the chemical to rats, 62.6% of the dose was recovered as urinary metabolites indicating substantial absorption. 1,2,4-Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. In the blood stream, approximately 85% of the chemical is bound to red blood cells. Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion. After a single oral dose to rats of 1200 mg/kg, urinary metabolites consisted of approximately 43.2% glycine, 6.6% glucuronic, and 12.9% sulfuric acid conjugates. The two principle metabolites excreted by rabbits after oral administration of 438 mg/kg/day for 5 days were 2,4-dimethylbenzoic acid and 3,4-dimethylhippuric acid. The major routes of excretion of 1,2,4-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. 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. 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>
Carboguard 635 Part B & ETHYLENEDIAMINE	<p>Allergic reactions involving the respiratory tract are usually due to interactions between IgE antibodies and allergens and occur rapidly. Allergic potential of the allergen and period of exposure often determine the severity of symptoms. Some people may be genetically more prone than others, and exposure to other irritants may aggravate symptoms. Allergy causing activity is due to interactions with proteins.</p>
Carboguard 635 Part B & ETHYLENEDIAMINE	<p>Attention should be paid to atopic diathesis, characterised by increased susceptibility to nasal inflammation, asthma and eczema.</p>
XYLENE & N-BUTANOL & ETHYLENEDIAMINE & 2,4,6-TRIS((DIMETHYLAMINO)METHYL)PHENOL	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>

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XYLENE & N-BUTANOL	The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.
N-BUTANOL & ETHYLENEDIAMINE & 1,2,4-TRIMETHYL BENZENE & 2,4,6-TRIS[(DIMETHYLAMINO)METHYL]PHENOL	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
ETHYLENEDIAMINE & 2,4,6-TRIS[(DIMETHYLAMINO)METHYL]PHENOL	The material may cause severe skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin. Repeated exposures may produce severe ulceration.

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

Legend: ✗ – Data available but does not fill the criteria for classification
 ✓ – Data available to make classification
 ☐ – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
xylene	LC50	96	Fish	2.6mg/L	2
xylene	EC50	48	Crustacea	>3.4mg/L	2
xylene	EC50	72	Algae or other aquatic plants	4.6mg/L	2
xylene	EC50	24	Crustacea	0.711mg/L	4
xylene	NOEC	73	Algae or other aquatic plants	0.44mg/L	2
n-butanol	LC50	96	Fish	88.462mg/L	3
n-butanol	EC50	48	Crustacea	>500mg/L	1
n-butanol	EC50	96	Algae or other aquatic plants	225mg/L	2
n-butanol	BCF	24	Fish	921mg/L	4
n-butanol	EC50	384	Crustacea	20.661mg/L	3
n-butanol	NOEC	48	Crustacea	415mg/L	2
ethylenediamine	LC50	96	Fish	115.7mg/L	4
ethylenediamine	EC50	48	Crustacea	3mg/L	1
ethylenediamine	EC50	96	Algae or other aquatic plants	61mg/L	1
ethylenediamine	EC0	24	Crustacea	1.2mg/L	1
ethylenediamine	NOEC	504	Crustacea	0.16mg/L	4
1,2,4-trimethyl benzene	LC50	96	Fish	1.318mg/L	3
1,2,4-trimethyl benzene	EC50	48	Crustacea	ca.6.14mg/L	1
1,2,4-trimethyl benzene	EC50	96	Algae or other aquatic plants	2.154mg/L	3
1,2,4-trimethyl benzene	EC50	384	Crustacea	0.328mg/L	3
2,4,6-tris[(dimethylamino)methyl]phenol	LC50	96	Fish	223.143mg/L	3
2,4,6-tris[(dimethylamino)methyl]phenol	EC50	96	Algae or other aquatic plants	34.812mg/L	3
2,4,6-tris[(dimethylamino)methyl]phenol	EC50	96	Algae or other aquatic plants	1616.048mg/L	3

Legend:

Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

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).

Continued...

Aquatic Fate: 1,2,4-Trimethylbenzene volatilizes rapidly from surface waters with volatilization half-life from a model river calculated to be 3.4 hours. Biodegradation of 1,2,4-trimethylbenzene has been noted in both seawater and ground water. Various strains of *Pseudomonas* can biodegrade 1,2,4-trimethylbenzene.

Terrestrial Fate: 1,2,4-Trimethylbenzene also volatilizes from soils however; moderate adsorption to soils and sediments may occur. Volatilization is the major route of removal of 1,2,4-trimethylbenzene from soils; although, biodegradation may also occur. Due to the high volatility of the chemical it is unlikely to accumulate in soil or surface water to toxic concentrations.

Ecotoxicity: No significant bioaccumulation has been noted. 1,2,4-Trimethylbenzene is moderately toxic to fathead minnow and slightly toxic to dungeness crab. 1,2,4-Trimethylbenzene has moderate acute toxicity to aquatic organisms. No stress was observed in rainbow trout, sea lamprey and *Daphnia magna* water fleas. The high concentrations required to induce toxicity in laboratory animals are not likely to be reached in the environment.

For Aromatic Substances Series:

Environmental Fate: Large, molecularly complex polycyclic aromatic hydrocarbons, or PAHs, are persistent in the environment longer than smaller PAHs.

Atmospheric Fate: PAHs are "semi-volatile substances" which can move between the atmosphere and the Earth's surface in repeated, temperature-driven cycles of deposition and volatilization.

Terrestrial Fate: BTEX compounds have the potential to move through soil and contaminate ground water, and their vapors are highly flammable and explosive.

Ecotoxicity - Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. The order of most toxic to least in a study using grass shrimp and brown shrimp was dimethylnaphthalenes > methylnaphthalenes > naphthalenes. 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 Silica:

Environmental Fate: Most documentation on the fate of silica in the environment concerns dissolved silica, in the aquatic environment, regardless of origin, (man-made or natural), or structure, (crystalline or amorphous).

Terrestrial Fate: Silicon makes up 25.7% of the Earth's crust, by weight, and is the second most abundant element, being exceeded only by oxygen. Silicon is not found free in nature, but occurs chiefly as the oxide and as silicates. Once released into the environment, no distinction can be made between the initial forms of silica.

Aquatic Fate: At normal environmental pH, dissolved silica exists exclusively as monosilicic acid. At pH 9.4, amorphous silica is highly soluble in water. Crystalline silica, in the form of quartz, has low solubility in water. Silicic acid plays an important role in the biological/geological/chemical cycle of silicon, especially in the ocean. Marine organisms such as diatoms, silicoflagellates and radiolarians use silicic acid in their skeletal structures and their skeletal remains leave silica in sea sediment

Ecotoxicity: Silicon is important to plant and animal life and is practically non-toxic to fish including zebrafish, and *Daphnia magna* water fleas.

For Inorganic Sulfate:

Environmental Fate - Sulfates can produce a laxative effect at concentrations of 1000 - 1200 mg/liter, but no increase in diarrhea, dehydration or weight loss. The presence of sulfate in drinking-water can also result in a noticeable taste. Sulfate may also contribute to the corrosion of distribution systems. No health-based guideline value for sulfate in drinking water is proposed.

Atmospheric Fate: Sulfates are removed from the air by both dry and wet deposition processes. Wet deposition processes including rain-out (a process that occurs within the clouds) and washout (removal by precipitation below the clouds) which contribute to the removal of sulfate from the atmosphere.

Terrestrial Fate: Soil - In soil, the inorganic sulfates can adsorb to soil particles or leach into surface water and groundwater. Plants - Sodium sulfate is not very toxic to terrestrial plants however; sulfates can be taken up by plants and be incorporated into the parenchyma of the plant. Some plants (e.g. corn and *Kochia Scoparia*) are capable of accumulating sulfate to concentrations that are potentially toxic to ruminants. Jack pine are the most sensitive plant species.

Aquatic Fate: Sulfate in water can also be reduced by sulfate bacteria (*Thiobacilli*) which use them as a source of energy. In anaerobic environments sulfate is biologically reduced to (hydrogen) sulfide by sulfate reducing bacteria, or incorporated into living organisms as source of sulfur. Sodium sulfate is not reactive in aqueous solution at room temperature. Sodium sulfate will completely dissolve, ionize and distribute across the entire planetary "aquasphere". Some sulfates may eventually be deposited with the majority of sulfates participating in the sulfur cycle in which natural and industrial sodium sulfates are not distinguishable.

Ecotoxicity: Significant bioconcentration or bioaccumulation is not expected. Algae are the most sensitive to sodium sulfate and toxicity occurs in bacteria from 2500mg/L. Sulfates are not acutely toxic to fish or invertebrates. *Daphnia magna* water fleas and fathead minnow appear to be the least sensitive species. Activated sludge showed a very low sensitivity to sodium sulfate. Overall it can be concluded that sodium sulfate has no acute adverse effect on aquatic and sediment dwelling organisms. No data were found for long term toxicity.

For Barium and its Compounds:

Environmental Fate: Barium is a highly reactive metal occurring naturally only in a combined state, primarily as inorganic complexes. Conditions such as pH, oxidation-reduction potential, cation exchange capacity, and the presence of sulfate, carbonate, and the presence of metal oxides will affect the partitioning of barium and its compounds in the environment. The element is released to environmental by both natural processes and man-made sources. Most barium released to the environment from industrial sources is in forms that do not become widely dispersed.

Atmospheric Fate: In the atmosphere, barium is likely to be present in particulate form. Barium compounds will be removed from the atmosphere via wet/dry deposition. The substance may change to different forms of barium in the air.

Terrestrial Fate: Soil - Barium will leach from geological formations to groundwater and will adsorb to soil. Barium is not very mobile in most soil systems and will form soluble complexes with fulvic/humic acids. Transportation rates of barium in soil are dependent on the characteristics of soil material. In soils with high positive ion exchange capacity, (e.g., fine textured mineral soils or soils with high organic matter content), barium mobility will be limited by adsorption. Soils high in calcium carbonate leave barium carbonate residues, which limit mobility. Barium produces barium sulfate residues in the presence of sulfates. Barium is more mobile, and is more likely to be leached, from soils in the presence of chloride and under acidic conditions. Barium binds with fatty acids, (e.g., in acidic landfill leachate), and will be much more mobile in soils containing fatty acids. Plants - Barium is not expected to concentrate in plants, relative to amounts found in soils; however, there are some plants, (beans, forage plants, Brazil nuts, and mushrooms), which accumulate barium.

Aquatic Fate: Barium will adsorb to sediment/suspended particulate matter. Precipitation of barium sulfate salts is accelerated where rivers enter the ocean. Sedimentation of suspended solids removes a large portion of the barium content from surface waters. Barium in sediments is found largely in the form of barium sulfate, (barite).

Ecotoxicity: Barium concentration will increase as it moves up the food chain in both land and aquatic species. In aquatic media, barium is likely to precipitate out of solution as an insoluble salt, (i.e. barium sulfate/barium sulfite). The uptake of barium by fish and marine organisms is also an important removal mechanism. Barium may concentrate in marine plants by a factor of 400-4,000 times the level present in the water. The substance may concentrate in marine animals, plankton, and brown algae.

For Xylenes:

log Koc : 2.05-3.08; Koc : 25.4-204; Half-life (hr) air : 0.24-42; Half-life (hr) H₂O surface water : 24-672; Half-life (hr) H₂O ground : 336-8640; Half-life (hr) soil : 52-672; Henry's Pa m³ /mol : 637-879; Henry's atm m³ /mol - 7.68E-03; BOD 5 if unstated - 1.4,1%; COD - 2.56,13% ThOD - 3.125 : BCF : 23; log BCF : 1.17-2.41.

Environmental Fate: Most xylenes released to the environment will occur in the atmosphere and volatilisation is the dominant environmental fate process. Soil - Xylenes are expected to have moderate mobility in soil evaporating rapidly from soil surfaces. The extent of the degradation is expected to depend on its concentration, residence time in the soil, the nature of the soil, and whether resident microbial populations have been acclimated. Xylene can remain below the soil surface for several days and may travel through the soil profile and enter groundwater. Soil and water microbes may transform it into other, less harmful compounds, although this happens slowly. It is not clear how long xylene remains trapped deep underground in soil or groundwater, but it may be months or years.

Atmospheric Fate: Xylene evaporates quickly into the air from surface soil and water and can remain in the air for several days until it is broken down by sunlight into other less harmful chemicals. In the ambient atmosphere, xylenes are expected to exist solely in the vapour phase. Xylenes are degraded in the atmosphere with an estimated atmospheric lifetime of about 0.5 to 2 days. Xylene may contribute to photochemical smog formation. p-Xylene has a moderately high photochemical reactivity under smog conditions, higher than the other xylene isomers. The photooxidation of p-xylene results in the production of carbon monoxide, formaldehyde, glyoxal, methylglyoxal, 3-methylbenzyl nitrate, m-tolaldehyde, 4-nitro-3-xylene, 5-nitro-3-xylene, 2,6-dimethyl-p-benzoquinone, 2,4-dimethylphenol, 6-nitro-2,4-dimethylphenol, 2,6-dimethylphenol, and 4-nitro-2,6-dimethylphenol.

Aquatic Fate: p-xylene may adsorb to suspended solids and sediment in water and is expected to volatilise from water surfaces. Estimated volatilisation half-lives for a model river and model lake are 3 hours and 4 days, respectively. Measurements taken from goldfish, eels and clams indicate that bioconcentration in aquatic organisms is low. Photo-oxidation in the presence of humic acids may play an important role in the abiotic degradation of p-xylene. p-Xylene is biodegradable and has been observed to degrade in pond water however; it is unclear if it degrades in surface waters. p-Xylene has been observed to degrade in anaerobic and aerobic groundwater; however, it is known to persist for many years in groundwater, at least at sites where the concentration might have been quite high. **Ecotoxicity:** Xylenes are slightly toxic to fathead minnow, rainbow trout and bluegill and not acutely toxic to water fleas. For *Photobacterium phosphoreum* EC50 (24 h): 0.0084 mg/L. and *Gammarus lacustris* LC50 (48 h): 0.6 mg/L.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
n-butanol	LOW (Half-life = 54 days)	LOW (Half-life = 3.65 days)
ethylenediamine	LOW	LOW
1,2,4-trimethyl benzene	LOW (Half-life = 56 days)	LOW (Half-life = 0.67 days)

2,4,6-tris[(dimethylamino)methyl]phenol	HIGH	HIGH
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Bioaccumulative potential

Ingredient	Bioaccumulation
xylene	MEDIUM (BCF = 740)
n-butanol	LOW (BCF = 0.64)
ethylenediamine	LOW (BCF = 0.07)
1,2,4-trimethyl benzene	LOW (BCF = 275)
2,4,6-tris[(dimethylamino)methyl]phenol	LOW (LogKOW = 0.773)

Mobility in soil

Ingredient	Mobility
n-butanol	MEDIUM (KOC = 2.443)
ethylenediamine	LOW (KOC = 24.72)
1,2,4-trimethyl benzene	LOW (KOC = 717.6)
2,4,6-tris[(dimethylamino)methyl]phenol	LOW (KOC = 15130)

SECTION 13 DISPOSAL CONSIDERATIONS**Waste treatment methods**

Product / Packaging disposal	<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 licenced to accept chemical and / or pharmaceutical wastes or Incineration in a licenced 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
HAZCHEM	•3Y

Land transport (ADG)

UN number	1263
UN proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
Transport hazard class(es)	Class : 3 Subrisk : Not Applicable
Packing group	III
Environmental hazard	Not Applicable

Carboguard 635 Part B

Special precautions for user	Special provisions	163 223 367
	Limited quantity	5 L

Air transport (ICAO-IATA / DGR)

UN number	1263	
UN proper shipping name	Paint (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base); Paint related material (including paint thinning or reducing compounds)	
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 A72 A192
	Cargo Only Packing Instructions	366
	Cargo Only Maximum Qty / Pack	220 L
	Passenger and Cargo Packing Instructions	355
	Passenger and Cargo Maximum Qty / Pack	60 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y344
	Passenger and Cargo Limited Maximum Qty / Pack	10 L

Sea transport (IMDG-Code / GGVSee)

UN number	1263	
UN proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac solutions, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)	
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	163 223 367 955
	Limited Quantities	5 L

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

Not Applicable

SECTION 15 REGULATORY INFORMATION**Safety, health and environmental regulations / legislation specific for the substance or mixture****CASTOR OIL, HYDROGENATED, MODIFIED(55353-47-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Not Applicable

XYLENE(1330-20-7) IS FOUND ON THE FOLLOWING REGULATORY LISTSAustralia Exposure Standards
Australia Hazardous Substances Information System - Consolidated ListsAustralia Inventory of Chemical Substances (AICS)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs**N-BUTANOL(71-36-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS**Australia Exposure Standards
Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

ETHYLENEDIAMINE(107-15-3) IS FOUND ON THE FOLLOWING REGULATORY LISTSAustralia Exposure Standards
Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

SILICA CRYSTALLINE - QUARTZ(14808-60-7) IS FOUND ON THE FOLLOWING REGULATORY LISTSAustralia Exposure Standards
Australia Hazardous Substances Information System - Consolidated ListsAustralia Inventory of Chemical Substances (AICS)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs**1,2,4-TRIMETHYL BENZENE(95-63-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

2,4,6-TRIS[(DIMETHYLAMINO)METHYL]PHENOL(90-72-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Continued...

Carboguard 635 Part B

Australia Hazardous Substances Information System - Consolidated Lists		Australia Inventory of Chemical Substances (AICS)
National Inventory	Status	
Australia - AICS	N (castor oil, hydrogenated, modified)	
Canada - DSL	N (castor oil, hydrogenated, modified)	
Canada - NDSL	N (n-butanol; xylene; 2,4,6-tris[(dimethylamino)methyl]phenol; ethylenediamine; silica crystalline - quartz; castor oil, hydrogenated, modified; 1,2,4-trimethyl benzene)	
China - IECSC	N (castor oil, hydrogenated, modified)	
Europe - EINEC / ELINCS / NLP	N (castor oil, hydrogenated, modified)	
Japan - ENCS	N (castor oil, hydrogenated, modified)	
Korea - KECI	N (castor oil, hydrogenated, modified)	
New Zealand - NZIoC	Y	
Philippines - PICCS	N (castor oil, hydrogenated, modified)	
USA - TSCA	N (castor oil, hydrogenated, modified)	
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing (see specific ingredients in brackets)	

SECTION 16 OTHER INFORMATION

Other information

Ingredients with multiple cas numbers

Name	CAS No
silica crystalline - quartz	14808-60-7, 122304-48-7, 122304-49-8, 12425-26-2, 1317-79-9, 70594-95-5, 87347-84-0

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

PC – TWA: Permissible Concentration-Time Weighted Average
 PC – STEL: Permissible Concentration-Short Term Exposure Limit
 IARC: International Agency for Research on Cancer
 ACGIH: American Conference of Governmental Industrial Hygienists
 STEL: Short Term Exposure Limit
 TEEL: Temporary Emergency Exposure Limit
 IDLH: Immediately Dangerous to Life or Health Concentrations
 OSF: Odour Safety Factor
 NOAEL :No Observed Adverse Effect Level
 LOAEL: Lowest Observed Adverse Effect Level
 TLV: Threshold Limit Value
 LOD: Limit Of Detection
 OTV: Odour Threshold Value
 BCF: BioConcentration Factors
 BEI: Biological Exposure Index

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