Crystalite Thermoplastic Concrete Primer Concentrate

Crystalite Design

Chemwatch:	4861-86	Print Date:	03/10/2013
Version No:	2.1.1.1	Issue Date:	03/10/2013
Safety Data Sheet according to WHS and ADG requirements		S.GHS.AUS.EN	

SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name:	Crystalite Thermoplastic Concrete Primer Concentrate
Chemical Name:	Not Applicable
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)
Chemical formula:	Not Applicable
Other means of identification:	Not Available
CAS number:	Not Applicable

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

Relevant identified uses:	Use according to manufacturer's directions.

Details of the supplier of the safety data sheet

Registered company name:	Crystalite Design		
Address:	26-28 Frederick Kelly Street South West Rocks 2431 NSW Australia		
Telephone:	+61 2 6566 7766		
Fax:	Not Available		
Website:	www.crystalite.com.au		
Email:	ryan@crystalite.com.au		

Emergency telephone number

Association / Organisation:	Not Available		
Emergency telephone numbers:	+61 407 766 796 (Mon-Fri; 8am-6pm)		
Other emergency telephone numbers:	Not Available		

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Poisons Schedule:	S5

GHS Classification[1]:

Acute Toxicity (Inhalation) Category 4, STOT - SE (Narcosis) Category 3, Aspiration Hazard Category 1, Eye Irrit. 2, Acute Aquatic Hazard Category 2, Chronic Aquatic Hazard Category 2, Flammable Liquid Category 2, Acute Toxicity (Dermal) Category 4

Legend:*1. Classified by Chemwatch; 2. Classification drawn from HSIS; 3. Classification drawn from EC Directive 1272/2008 - Annex VI*

Label elements

GHS label elements

Signal word:	**DANGER**

Hazard statement(s):

AUH066	Repeated exposure may cause skin dryness and cracking
H225	Highly flammable liquid and vapour
H304	May be fatal if swallowed and enters airways
H312	Harmful in contact with skin
H319	Causes serious eye irritation
H332	Harmful if inhaled
H336	May cause drowsiness or dizziness
H401	Toxic to aquatic life
H411	Toxic to aquatic life with long lasting effects

Precautionary statement(s): Prevention

P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.

P233	Keep container tightly closed.
P240	Ground/bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use only non-sparking tools.
P243	Take precautionary measures against static discharge.
P261	Avoid breathing dust/fume/gas/mist/vapours/spray.
P264	Wash all exposed external body areas thoroughly after handling.
P271	Use only outdoors or in a well-ventilated area.
P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
Precautionary statement(s): Response	
P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider
P302+P352	IF ON SKIN: Wash with plenty of water and soap
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water/shower.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P322	Specific measures (see advice on this label).
P331	Do NOT induce vomiting.
P337+P313	If eye irritation persists: Get medical advice/attention.
P363	Wash contaminated clothing before reuse.
P370+P378	In case of fire: Use… to extinguish.
P391	Collect spillage.
Precautionary statement(s): Storage	
P403+P233	Store in a well-ventilated place. Keep container tightly closed.
P403+P235	Store in a well-ventilated place. Keep cool.
P405	Store locked up.
Precautionary statement(s): Disposal	
P501	Dispose of contents/container to authorised chemical landfill or if organic to high temperature incineration

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
67-64-1	20-40	acetone
64742-95-6.	20-40	naphtha petroleum, light aromatic solvent
64742-16-1	20-40	hydrocarbon resin, postpolymerised with maleic anhydride
1330-20-7	5-20	xylene
108-65-6	1-10	propylene glycol monomethyl ether acetate, alpha-isomer

SECTION 4 First aid measures

Description of first aid measures

Eye Contact:

If this product comes in contact with the eyes:

- Wash out immediately with fresh running water.
- Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.
- Seek medical attention without delay; if pain persists or recurs seek medical attention.
- Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

Skin Contact:

If skin contact occurs:

- Immediately remove all contaminated clothing, including footwear.
- Flush skin and hair with running water (and soap if available).
- Seek medical attention in event of irritation.

In case of burns:

- Immediately apply cold water to burn either by immersion or wrapping with saturated clean cloth.
- **DO NOT** remove or cut away clothing over burnt areas. **DO NOT** pull away clothing which has adhered to the skin as this can cause further injury.
- **DO NOT** break blister or remove solidified material.
- Quickly cover wound with dressing or clean cloth to help prevent infection and to ease pain.
- For large burns, sheets, towels or pillow slips are ideal; leave holes for eyes, nose and mouth.
- **DO NOT** apply ointments, oils, butter, etc. to a burn under any circumstances.
- Water may be given in small quantities if the person is conscious.
- Alcohol is not to be given under any circumstances.
- Reassure.
- Treat for shock by keeping the person warm and in a lying position.
- Seek medical aid and advise medical personnel in advance of the cause and extent of the injury and the estimated time of arrival of the patient.

Inhalation:

- If fumes or combustion products are inhaled remove from contaminated area.
- Lay patient down. Keep warm and rested.
- Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.
- Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.
- Transport to hospital, or doctor.

Ingestion:

- For advice, contact a Poisons Information Centre or a doctor at once.
- Urgent hospital treatment is likely to be needed.
- 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.
- Transport to hospital or doctor without delay.

Indication of any immediate medical attention and special treatment needed

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.
Treat symptomatically.
For acute or short term repeated exposures to acetone:

- Symptoms of acetone exposure approximate ethanol intoxication.
- About 20% is expired by the lungs and the rest is metabolised. Alveolar air half-life is about 4 hours following two hour inhalation at levels near the Exposure Standard; in overdose, saturable metabolism and limited clearance, prolong the elimination half-life to 25-30 hours.
- There are no known antidotes and treatment should involve the usual methods of decontamination followed by supportive care.

[Ellenhorn and Barceloux: Medical Toxicology]
Management:
Measurement of serum and urine acetone concentrations may be useful to monitor the severity of ingestion or inhalation.
Inhalation Management:

- Maintain a clear airway, give humidified oxygen and ventilate if necessary.
- If respiratory irritation occurs, assess respiratory function and, if necessary, perform chest X-rays to check for chemical pneumonitis.
- Consider the use of steroids to reduce the inflammatory response.
- Treat pulmonary oedema with PEEP or CPAP ventilation.

Dermal Management:

- Remove any remaining contaminated clothing, place in double sealed, clear bags, label and store in secure area away from patients and staff.
- Irrigate with copious amounts of water.
- An emollient may be required.

Eye Management:

- Irrigate thoroughly with running water or saline for 15 minutes.
- Stain with fluorescein and refer to an ophthalmologist if there is any uptake of the stain.

Oral Management:

- No **GASTRIC LAVAGE OR EMETIC**
- Encourage oral fluids.

Systemic Management:

- Monitor blood glucose and arterial pH.
- Ventilate if respiratory depression occurs.
- If patient unconscious, monitor renal function.
- Symptomatic and supportive care.

The Chemical Incident Management Handbook:
Guy's and St. Thomas' Hospital Trust, 2000
BIOLOGICAL EXPOSURE INDEX
These represent the determinants observed in specimens collected from a healthy worker exposed at the Exposure Standard (ES or TLV):

Determinant	Sampling Time	Index	Comments
Acetone in urine	End of shift	50 mg/L	NS

NS: Non-specific determinant; also observed after exposure to other material
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	End of shift	
	2 mg/min	Last 4 hrs of shift	

SECTION 5 Firefighting measures

Extinguishing media

- Alcohol stable foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.

Special hazards arising from the substrate or mixture

Fire Incompatibility:

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

Advice for firefighters

Fire Fighting:

- Alert Fire Brigade and tell them location and nature of hazard.
- May be violently or explosively reactive.
- Wear breathing apparatus plus protective gloves in the event of a fire.
- Prevent, by any means available, spillage from entering drains or water course.

Fire/Explosion Hazard:

- Liquid and vapour are highly flammable.
- Severe fire hazard when exposed to heat, flame and/or oxidisers.
- Vapour may travel a considerable distance to source of ignition.
- Heating may cause expansion or decomposition leading to violent rupture of containers.

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

Minor Spills:

- Remove all ignition sources.
- Clean up all spills immediately.
- Avoid breathing vapours and contact with skin and eyes.
- Control personal contact with the substance, by using protective equipment.

Major Spills:

- 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.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling

- **DO NOT** allow clothing wet with material to stay in contact with skin
- Avoid all personal contact, including inhalation.
- Wear protective clothing when risk of exposure occurs.
- Use in a well-ventilated area.
- Prevent concentration in hollows and sumps.

Other information

- Store in original containers in approved flame-proof area.
- No smoking, naked lights, heat or ignition sources.
- **DO NOT** store in pits, depressions, basements or areas where vapours may be trapped.
- Keep containers securely sealed.
- Store away from incompatible materials in a cool, dry well ventilated area.

Conditions for safe storage, including any incompatibilities

Suitable container:

- Packing as supplied by manufacturer.
- Plastic containers may only be used if approved for flammable liquid.
- Check that containers are clearly labelled and free from leaks.

Storage incompatibility:

- Avoid reaction with oxidising agents

Package Material Incompatibilities:

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	acetone	Acetone	1185 (mgm3) / 500 (ppm)	2375 (mgm3) / 1000 (ppm)	Not Available	Not Available
Australia Exposure Standards	xylene	Xylene (o-, m-, p- isomers)	350 (mgm3) / 80 (ppm)	655 (mgm3) / 150 (ppm)	Not Available	Not Available
Australia Exposure Standards	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxy-2-propanol acetate	274 (mgm3) / 50 (ppm)	548 (mgm3) / 100 (ppm)	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-0	TEEL-1	TEEL-2	TEEL-3
acetone	200(ppm)	200(ppm)	3200(ppm)	5700(ppm)
naphtha petroleum, light aromatic solvent	500(ppm)	750(ppm)	750(ppm)	750(ppm)
xylene	100(ppm)	130(ppm)	920(ppm)	2500(ppm)
propylene glycol monomethyl ether acetate, alpha-isomer	50(ppm)	150(ppm)	250(ppm)	600(ppm)

Ingredient	Original IDLH	Revised IDLH
acetone	20,000 / 5,000(ppm)	2,500 [LEL] / 1,500(ppm)
xylene	1,000(ppm)	900(ppm)

Exposure controls

Appropriate engineering controls

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

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)

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

Within each range the appropriate value depends on:

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

Hand protection:

- Wear chemical protective gloves, e.g. PVC.
- Wear safety footwear or safety gumboots, e.g. Rubber

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.
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.
- Contaminated gloves should be replaced.

Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

Body protection:

See Other protection below

Other protection:

- Overalls.
- PVC Apron.
- PVC protective suit may be required if exposure severe.
- Eyewash unit.

Thermal hazards:

Recommended material(s):	Respiratory protection:
1.PE/EVAL/PE 2.PVDC/PE/PVDC	Type AX-P Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance

Highly flammable liquid; does not mix with water.

Physical state	Liquid	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	<21	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available

Solubility in water (g/L)	Immiscible	**pH as a solution(1%)**	Not Available
Vapour density (Air = 1)	Not Available		

SECTION 10 Stability and reactivity

Reactivity:

See section 7

Chemical stability:

- 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:

Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful.
Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.
Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.
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.
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.
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
Inhalation hazard is increased at higher temperatures.
High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations.
Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.
Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination
Mice exposed at up to 3000 ppm PGMEA 6 hr/day for a total of 9 days during an 11-day period showed no pronounced effect on the weights of liver, kidneys, heart, spleen, thymus or testes. Histopathological examination revealed degeneration of the olfactory epithelium in mice exposed at 300 ppm for the same time. Rats, similarly failed to show changes in internal organs and did not show olfactory epithelium degeneration until 3000 ppm. The no-effect level in rats was 1000 ppm.
Headache, fatigue, lassitude, irritability and gastrointestinal disturbances (e.g., nausea, anorexia and flatulence) are the most common symptoms of xylene overexposure. Injury to the heart, liver, kidneys and nervous system has also been noted amongst workers. Transient memory loss, renal impairment, temporary confusion and some evidence of disturbance of liver function was reported in three workers overcome by gross exposure to xylene (10000 ppm). One worker died and autopsy revealed pulmonary congestion, oedema and focal alveolar haemorrhage. Volunteers inhaling xylene at 100 ppm for 5 to 6 hours showed changes in manual coordination reaction time and slight ataxia. Tolerance developed during the workweek but was lost over the weekend. Physical exercise may antagonise this effect. Xylene body burden in humans exposed to 100 or 200 ppm xylene in air depends on the amount of body fat with 4% to 8% of total absorbed xylene accumulating in adipose tissue.
Xylene is a central nervous system depressant. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.
Systemic effects of acetone inhalation exposure include central nervous system depression, light-headedness, incoherent speech, ataxia, stupor, hypotension, tachycardia, metabolic acidosis, hyperglycaemia and ketosis. Rarely, convulsions and tubular necrosis may be evident. Other symptoms of exposure may include restlessness, headache, vomiting, low blood-pressure and rapid and irregular pulse, eye and throat irritation, weakness of the legs and dizziness. Inhalation of high concentrations may produce dryness of the mouth and throat, nausea, uncoordinated movement, loss of coordinated speech, drowsiness and, in severe cases, coma. Inhalation of acetone vapours over long periods causes irritation of the respiratory tract, coughing and headache. Rats exposed to 52200 ppm vapour for 1 hour showed clear signs of narcosis; fatalities occurred at 126600 ppm.
Exposure to ketone vapours may produce nose, throat and mucous membrane irritation. High concentrations of vapour may produce central nervous system depression characterised by headache, vertigo, loss of coordination, narcosis and cardiorespiratory failure. Some ketones produce neurological disorders (polyneuropathy) characterised by bilateral symmetrical paresthesia and muscle weakness primarily in the legs and arms.

Ingestion:

Accidental ingestion of the material may be damaging to the health of the individual.
Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.
Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).
Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosal ulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea and vomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injury may produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Light aromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration into the lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage.

Skin Contact:

Skin contact with the material may be harmful; systemic effects may result following absorption.
The material may produce moderate skin irritation; limited evidence or practical experience suggests, that the material either:

- produces moderate inflammation of the skin in a substantial number of individuals following direct contact and/or
- produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period.

Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.
Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.
Repeated application of commercial grade PGMEA to the skin of rabbits for 2-weeks caused slight redness and very slight exfoliation.
Open cuts, abraded or irritated skin should not be exposed to this materialThe material may accentuate any pre-existing dermatitis condition
Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Aromatic hydrocarbons may produce skin irritation, vasodilation with erythema and changes in endothelial cell permeability. Systemic intoxication, resulting from contact with the light aromatics, is unlikely due to the slow rate of permeation. Branching of the side chain appears to increase percutaneous absorption.

Eye:

Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may also result. The aromatic fraction may produce irritation and lachrymation.
Undiluted propylene glycol monomethyl ether acetate (PGMEA) causes moderate discomfort, slight conjunctival redness and slight corneal injury in rabbits
The liquid may produce eye discomfort and is capable of causing temporary impairment of vision and/or transient eye inflammation, ulceration

Chronic:

Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.
On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.
Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.
There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.
Repeated exposure to higher concentrations of propylene glycol monomethyl ether acetate (PGMEA) (1000 ppm and above) causes mild liver and kidney damage in animals.
A minor component, 2-methoxy-1-propyl acetate (the beta-isomer) produced birth defects on inhalation exposure of pregnant rabbits at 545 ppm, but not at 145 or 36 ppm; maternal and embryo/foetal toxicity on inhalation exposure of pregnant rats at 2710 ppm, but not at 545 or 110 ppm; and no adverse effects on dermal exposure of pregnant rabbits at applied dosages of 1000 and 2000 mg/kg of body weight per day during the critical period or embryo/foetal development. In a further study, no developmental effects were seen following exposure of pregnant rats at air concentrations of commercial propylene glycol monomethyl ether acetate (containing 3-5% of the minor component) up to 4000 ppm; slight maternal effects were seen at 5000 ppm and greater.
Exposure of pregnant rats and rabbits to the parent glycol ether, propylene glycol monomethyl ether which contained comparable amounts of the primary isomer, 2-methoxy-1-propanol, did not produce teratogenic effects at concentrations up to 3000 ppm. Foetotoxic effects were seen in rat foetuses but not in rabbit foetuses at this concentration and maternal toxicity was noted in both species at this concentration
Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses. Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.
Prolonged or repeated contact with xylenes may cause defatting dermatitis with drying and cracking. Chronic inhalation of xylenes has been associated with central nervous system effects, loss of appetite, nausea, ringing in the ears, irritability, thirst anaemia, mucosal bleeding, enlarged liver and hyperplasia. Exposure may produce kidney and liver damage. In chronic occupational exposure, xylene (usually mix ed with other solvents) has produced irreversible damage to the central nervous system and ototoxicity (damages hearing and increases sensitivity to noise), probably due to neurotoxic mechanisms.
Industrial workers exposed to xylene with a maximum level of ethyl benzene of 0.06 mg/l (14 ppm) reported headaches and irritability and tired quickly. Functional nervous system disturbances were found in some workers employed for over 7 years whilst other workers had enlarged livers.
Xylene has been classed as a developmental toxin in some jurisdictions.
Small excess risks of spontaneous abortion and congenital malformation were reported amongst women exposed to xylene in the first trimester of pregnancy. In all cases, however, the women were also been exposed to other substances. Evaluation of workers chronically exposed to xylene has demonstrated lack of genotoxicity. Exposure to xylene has been associated with increased risks of haemopoietic malignancies but, again, simultaneous exposure to other substances (including benzene) complicates the picture. A long-term gavage study to mixed xylenes (containing 17% ethyl benzene) found no evidence of carcinogenic activity in rats and mice of either sex.
Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).
Workers exposed to 700 ppm acetone for 3 hours/day for 7-15 years showed inflammation of the respiratory tract, stomach and duodenum, attacks of giddiness and loss of strength. Exposure to acetone may enhance liver toxicity of chlorinated solvents.

TOXICITY	IRRITATION
Crystalite Thermoplastic Concrete Primer Concentrate	
Not Available	Not Available
acetone	
Dermal (rabbit) LD50: 20000 mg/kg	Eye (human): 500 ppm - irritant
Inhalation (rat) LC50: 50100 mg/m3/8 hr	Eye (rabbit): 20mg/24hr -moderate
Oral (rat) LD50: 5800 mg/kg	Eye (rabbit): 3.95 mg - SEVERE
	Skin (rabbit): 500 mg/24hr - mild
	Skin (rabbit):395mg (open) - mild
Not Available	Not Available
naphtha petroleum, light aromatic solvent	
Inhalation (rat) LC50: >3670 ppm/8 h *	
Oral (rat) LD50: >5000 mg/kg *	
Not Available	Not Available
hydrocarbon resin, postpolymerised with maleic anhydride	
Oral (-) LD50: 7000-10000 mg/kg	. [Manufacturer]
Not Available	Not Available
xylene	
Inhalation (rat) LC50: 5000 ppm/4h	Eye (human): 200 ppm irritant
Intraperitoneal (Mouse) LD50: 1548 mg/kg	Eye (rabbit): 5 mg/24h SEVERE
Intraperitoneal (Rat) LD50: 2459 mg/kg	Eye (rabbit): 87 mg mild
Oral (Mouse) LD50: 2119 mg/kg	Skin (rabbit):500 mg/24h moderate
Oral (rat) LD50: 4300 mg/kg	
Subcutaneous (Rat) LD50: 1700 mg/kg	

Not Available	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	
Dermal (rabbit) LD50: >5000 mg/kg*	* [CCINFO]
Inhalation (rat) LC50: 4345 ppm/6h	
Oral (rat) LD50: 8532 mg/kg	
Not Available	Not Available

Not available. Refer to individual constituents.

ACETONE

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
for acetone:
The acute toxicity of acetone is low. Acetone is not a skin irritant or sensitiser but is a defatting agent to the skin. Acetone is an eye irritant. The subchronic toxicity of acetone has been examined in mice and rats that were administered acetone in the drinking water and again in rats treated by oral gavage. Acetone-induced increases in relative kidney weight changes were observed in male and female rats used in the oral 13-week study. Acetone treatment caused increases in the relative liver weight in male and female rats that were not associated with histopathologic effects and the effects may have been associated with microsomal enzyme induction. Haematologic effects consistent with macrocytic anaemia were also noted in male rats along with hyperpigmentation in the spleen. The most notable findings in the mice were increased liver and decreased spleen weights. Overall, the no-observed-effect-levels in the drinking water study were 1% for male rats (900 mg/kg/d) and male mice (2258 mg/kg/d), 2% for female mice (5945 mg/kg/d), and 5% for female rats (3100 mg/kg/d). For developmental effects, a statistically significant reduction in foetal weight, and a slight, but statistically significant increase in the percent incidence of later resorptions were seen in mice at 15,665 mg/m3 and in rats at 26,100 mg/m3. The no-observable-effect level for developmental toxicity was determined to be 5220 mg/m3 for both rats and mice.
Teratogenic effects were not observed in rats and mice tested at 26,110 and 15,665 mg/m3, respectively. Lifetime dermal carcinogenicity studies in mice treated with up to 0.2 mL of acetone did not reveal any increase in organ tumor incidence relative to untreated control animals.
The scientific literature contains many different studies that have measured either the neurobehavioural performance or neurophysiological response of humans exposed to acetone. Effect levels ranging from about 600 to greater than 2375 mg/m3 have been reported. Neurobehavioral studies with acetone-exposed employees have recently shown that 8-hr exposures in excess of 2375 mg/m3 were not associated with any dose-related changes in response time, vigilance, or digit span scores. Clinical case studies, controlled human volunteer studies, animal research, and occupational field evaluations all indicate that the NOAEL for this effect is 2375 mg/m3 or greater.

NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT

for petroleum:
This product contains benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic.
This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss.
This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents
Carcinogenicity: Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans.
Mutagenicity: There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays.
Reproductive Toxicity: Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed.
Human Effects: Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials.
Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.

XYLENE

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
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.

PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER

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).
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).
This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.
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.
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.
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
None are skin sensitisers.
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).
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.
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.
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.
The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, 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 *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.
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.I]

Acute Toxicity:	Acute Toxicity (Inhalation) Category 4 Acute Toxicity (Dermal) Category 4	**Carcinogenicity:**	Not Applicable
Skin Irritation/Corrosion:	Not Applicable	**Reproductivity:**	Not Applicable
Serious Eye Damage/Irritation:	Eye Irrit. 2	**STOT - Single Exposure:**	STOT - SE (Narcosis) Category 3
Respiratory or Skin sensitisation:	Not Applicable	**STOT - Repeated Exposure:**	Not Applicable
Mutagenicity:	Not Applicable	**Aspiration Hazard:**	Aspiration Hazard Category 1

CMR STATUS

SKIN

propylene glycol monomethyl ether acetate, alpha-isomer	Australia Exposure Standards - Skin	Sk

SECTION 12 Ecological information

Toxicity

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
Not Available	Not Available	Not Available

Bioaccumulative potential

Ingredient	Bioaccumulation
Not Available	Not Available

Mobility in soil

Ingredient	Mobility
Not Available	Not Available

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal:

- Containers may still present a chemical hazard/ danger when empty.
- Return to supplier for reuse/ recycling if possible.

Otherwise:

- If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.
- Where possible retain label warnings and MSDS and observe all notices pertaining to the product.

SECTION 14 Transport information

Labels Required:

Marine Pollutant: NO

HAZCHEM: •3YE; •3Y

Land transport (ADG)

UN number	1263	**Packing group**	II
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) (see 3.2.5 for relevant [AUST.] entries)	**Environmental hazard**	No relevant data
Transport hazard class(es)	Class: 3 Subrisk:	**Special precautions for user**	Special provisions 163 * limited quantity 5 L

Air transport (ICAO-IATA / DGR)

UN number	1263	**Packing group**	II
UN proper shipping name	Paint (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base)	**Environmental hazard**	No relevant data
Transport hazard class(es)	ICAO/IATA Class: 3 ICAO / IATA Subrisk: ERG Code: 3L	**Special precautions for user**	Special provisions: A3A72 Cargo Only Packing Instructions: 364 Cargo Only Maximum Qty / Pack: 60 L Passenger and Cargo Packing Instructions: 353 Passenger and Cargo Maximum Qty / Pack: 5 L Passenger and Cargo Limited Quantity Packing Instructions: Y341 Passenger and Cargo Maximum Qty / Pack: 1 L

Sea transport (IMDG-Code / GGVSee)

UN number	1263	**Packing group**	II
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)	**Environmental hazard**	No relevant data
Transport hazard class(es)	IMDG Class: 3 IMDG Subrisk:	**Special precautions for user**	EMS Number: F-E,S-E Special provisions: 163 Limited Quantities: 5 L

Transport in bulk according to Annex II of MARPOL 73 / 78 and the IBC code

Source	Ingredient	Pollution Category	Residual Concentration - Outside Special Area (% w/w)	Residual Concentrati
IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances	acetone	Not Available	Not Available	Not Available
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	xylene	Not Available		

SECTION 15 Regulatory information

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

acetone(67-64-1) is found on the following regulatory lists

"Australia Inventory of Chemical Substances (AICS)","OECD List of High Production Volume (HPV) Chemicals","Australia High Volume Industrial Chemical List (HVICL)","Australia Exposure Standards","Australia Customs (Prohibited Exports) Regulations 1958 - Schedule 9 Precursor substances - Part 2","Australia FAISD Handbook - First Aid Instructions, Warning Statements, and General Safety Precautions","United Nations Consolidated List of Products Whose Consumption and/or Sale Have Been Banned, Withdrawn, Severely Restricted or Not Approved by Governments","Australia Hazardous Substances Information System - Consolidated Lists","Australia Crimes (Traffic in Narcotic Drugs and Psychotropic Substances) Act - Schedule 1 - United Nations Convention Against Illicit Traffic In Narcotic Drugs And Psychotropic Substances - Table II","IOFI Global Reference List of Chemically Defined Substances","Australia National Pollutant Inventory","GESAMP/EHS Composite List - GESAMP Hazard Profiles","IMO IBC Code Chapter 18: List of products to which the Code does not apply","IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances","FisherTransport Information","Sigma-AldrichTransport Information","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix F (Part 3)","FEMA Generally Recognized as Safe (GRAS) Flavoring Substances 23 - Examples of FEMA GRAS Substances with Non-Flavor Functions","International Fragrance Association (IFRA) Survey: Transparency List","Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2013 (English)","International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index","Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List","International Maritime Dangerous Goods Requirements (IMDG Code)","Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes","International Air Transport Association (IATA) Dangerous Goods Regulations","Australia - Victoria Occupational Health and Safety Regulations - Schedule 9: Materials at Major Hazard Facilities (And Their Threshold Quantity) Table 2","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)","IMO IBC Code Chapter 17: Summary of minimum requirements","Australia Illicit Drug Reagents/Essential Chemicals - Category III","United Nations List of Precursors and Chemicals Frequently used in the Illicit Manufacture of Narcotic Drugs and Psychotropic Substances Under International Control (Red List) - Table II","United Nations Convention Against Illicit Traffic in Narcotic Drugs and Psychotropic Substances - Table II","OSPAR National List of Candidates for Substitution – Norway"

naphtha petroleum, light aromatic solvent(64742-95-6.) is found on the following regulatory lists

"International Council of Chemical Associations (ICCA) - High Production Volume List","Australia Inventory of Chemical Substances (AICS)","OECD List of High Production Volume (HPV) Chemicals","Australia High Volume Industrial Chemical List (HVICL)","International Chemical Secretariat (ChemSec) SIN List (*Substitute It Now!)","Australia Hazardous Substances Information System - Consolidated Lists","Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2013 (English)","International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index","Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List","International Maritime Dangerous Goods Requirements (IMDG Code)","Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes","International Air Transport Association (IATA) Dangerous Goods Regulations"

hydrocarbon resin, postpolymerised with maleic anhydride(64742-16-1) is found on the following regulatory lists

"OECD List of High Production Volume (HPV) Chemicals","Australia High Volume Industrial Chemical List (HVICL)","Australia Inventory of Chemical Substances (AICS)"

xylene(1330-20-7) is found on the following regulatory lists

"International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs","International Council of Chemical Associations (ICCA) - High Production Volume List","Australia Inventory of Chemical Substances (AICS)","Australia High Volume Industrial Chemical List (HVICL)","Australia Hazardous Substances Information System - Consolidated Lists","FisherTransport Information","International Fragrance Association (IFRA) Survey: Transparency List","Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2013 (English)","International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index","Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List","International Maritime Dangerous Goods Requirements (IMDG Code)","Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes","International Air Transport Association (IATA) Dangerous Goods Regulations","OECD List of High Production Volume (HPV) Chemicals","OSPAR List of Chemicals for Priority Action","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7","IMO IBC Code Chapter 17: Summary of minimum requirements","GESAMP/EHS Composite List - GESAMP Hazard Profiles","Australia Exposure Standards","Australia FAISD Handbook - First Aid Instructions, Warning Statements, and General Safety Precautions","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix I","WHO Guidelines for Drinking-water Quality - Guideline values for chemicals that are of health significance in drinking-water","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6","Australia National Pollutant Inventory","IMO Provisional Categorization of Liquid Substances - List 3: (Trade-named) mixtures containing at least 99% by weight of components already assessed by IMO, presenting safety hazards","IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix F (Part 3)","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)","Australia - Australian Capital Territory - Environment Protection Regulation: Ambient environmental standards (Domestic water supply - organic compounds)","Australia Drinking Water Guideline Values For Physical and Chemical Characteristics","Australia - Australian Capital Territory - Environment Protection Regulation: Pollutants entering waterways taken to cause environmental harm - Domestic water supply quality","Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5"

propylene glycol monomethyl ether acetate, alpha-isomer(108-65-6) is found on the following regulatory lists

"International Council of Chemical Associations (ICCA) - High Production Volume List","Australia Inventory of Chemical Substances (AICS)","OECD List of High Production Volume (HPV) Chemicals","Australia High Volume Industrial Chemical List (HVICL)","Australia Exposure Standards","Australia Hazardous Substances Information System - Consolidated Lists","IMO IBC Code Chapter 17: Summary of minimum requirements","GESAMP/EHS Composite List - GESAMP Hazard Profiles","FisherTransport Information","Sigma-AldrichTransport Information","Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2013 (English)","International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index","Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List","International Maritime Dangerous Goods Requirements (IMDG Code)","Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes","International Air Transport Association (IATA) Dangerous Goods Regulations","Australia National Pollutant Inventory","OSPAR National List of Candidates for Substitution – Norway"

SECTION 16 Other information

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.
A list of reference resources used to assist the committee may be found at:
www.chemwatch.net/references

The (M)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.