Mirotone

Chemwatch: **5072-40**Version No: **11.1.1.1**

Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 3

Issue Date: 08/15/2014 Print Date: 04/18/2016 Initial Date: Not Available L.GHS.AUS.EN

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

Product Identifier

Product name	URAPOL 1012/1013/1014	
Synonyms	012/1013/1014, Product Code: 1012, 1013, 1014	
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	

Use according to manufacturer's directions.

The use of a quantity of material in an unventilated or confined space may result in increased exposure and an irritating atmosphere developing. Before starting consider control of exposure by mechanical ventilation.

Moisture cured polyurethane floor coating for interior timber.

Details of the supplier of the safety data sheet

Registered company name	Mirotone	
Address	21 Marigold Street NSW Revesby 2212 Australia	
Telephone	+61 2 9795 3700	
Fax	61 2 9771 3601	
Website	ww.mirotone.com, www.polycure.com.au	
Email	Not Available	

Emergency telephone number

Association / Organisation	lot Available	
Emergency telephone numbers	1800 039 008 (Aust)	
Other emergency telephone numbers	+61 3 9573 3112 (International)	

CHEMWATCH EMERGENCY RESPONSE

Primary Number	Alternative Number 1	Alternative Number 2
1800 039 008	+612 9186 1132	Not Available

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

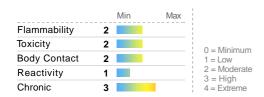
SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

CHEMWATCH HAZARD RATINGS

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Poisons Schedule	S5
Classification ^[1]	Flammable Liquid Category 3, Acute Toxicity (Oral) Category 4, Acute Toxicity (Dermal) Category 4, Acute Toxicity (Inhalation) Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Germ cell mutagenicity Category 2, Reproductive Toxicity Category 1B, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Specific target organ toxicity - repeated exposure Category 2, Acute Aquatic Hazard Category 2, Chronic Aquatic Hazard Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HSIS; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

Label elements

GHS label elements









SIGNAL WORD DANGER

Hazard statement(s)

` '		
H226	Flammable liquid and vapour.	
H302	Harmful if swallowed.	
H312	Harmful in contact with skin.	
H332	Harmful if inhaled.	
H315	Causes skin irritation.	
H319	Causes serious eye irritation.	
H341	Suspected of causing genetic defects.	
H360	May damage fertility or the unborn child.	
H336	May cause drowsiness or dizziness.	
H373	ay cause damage to organs.	
H411	oxic to aquatic life with long lasting effects.	
AUH066	Repeated exposure may cause skin dryness and cracking	

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.	
P210	Geep away from heat/sparks/open flames/hot surfaces No smoking.	
P260	Do not breathe dust/fume/gas/mist/vapours/spray.	
P271	Use only outdoors or in a well-ventilated area.	
P281	personal protective equipment as required.	
P240	ound/bond container and receiving equipment.	
P241	lse explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.	
P242	Use only non-sparking tools.	
P243	Take precautionary measures against static discharge.	
P270	Do not eat, drink or smoke when using this product.	
P273	Avoid release to the environment.	
P280	Wear protective gloves/protective clothing/eye protection/face protection.	

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/attention.	
P362	ke off contaminated clothing and wash before reuse.	
P363	Wash contaminated clothing before reuse.	
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam for extinction.	

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P305+P351+P338	IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue asing.			
P337+P313	riritation persists: Get medical advice/attention.			
P391	Collect spillage.			
P301+P312	WALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell.			
P302+P352	ON SKIN: Wash with plenty of soap and water.			
P303+P361+P353	F ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower.			
P304+P340	F INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.			
P330	Rinse mouth.			
P332+P313	If skin irritation occurs: Get medical advice/attention.			

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

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

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available	30-60	resin
67-63-0	10-30	isopropanol
71-36-3	10-30	<u>n-butanol</u>
1330-20-7	10-50	xylene
108-65-6	10-30	propylene glycol monomethyl ether acetate, alpha-isomer
123-86-4	10-30	n-butyl acetate
Not Available	0-10	filler
77-58-7	0-5	dibutyltin dilaurate
26471-62-5	NotSpec.	toluene diisocyanate

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.
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.

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 simple esters:

BASIC TREATMENT

Ingestion

- Establish a patent airway with suction where necessary.
- Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- Monitor and treat, where necessary, for pulmonary oedema.
- Monitor and treat, where necessary, for shock.
- DO NOT use emetics. Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.
- · Give activated charcoal.

ADVANCED TREATMENT

· Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.

- ▶ Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- ▶ Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Treat seizures with diazenam.
- Proparacaine hydrochloride should be used to assist eye irrigation.

EMERGENCY DEPARTMENT

- Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- Consult a toxicologist as necessary.

BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

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

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

Scanty animal data indicate that BAL may be useful against dialkyl but not trialkyl organotin compounds. D-penicillamine is thought to be inactive. GOSSELIN, SMITH & HODGE: Clinical Toxicology of Commercial Products, 5th Ed

Dimercaprol is suggested to be an effective antidote for dialkyltin poisoning and has been reported to prevent the accumulation of alpha-keto acids produced by dialkyltin compounds. It does not however appear to protect rats from the general toxic effects of triethyltin compounds. This may be due to the fact that dialkyltin compounds, at least up to dihexyl derivatives, react readily with sulfhydryl groups and trialkyltin compounds do not.

Surgical decompression was considered to be the only treatment that offered any benefit in human cases of cerebral oedema caused by trialkyl

Tin and Organotin Compounds: A Preliminary Review.

ENVIRONMENTAL HEALTH CRITERIA: World Health Organization Geneva 1980.

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SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

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

Do not use a water jet to fight fire.

Special hazards arising from the substrate or mixture

Fire	Incompatibility
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▶ 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. 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	 Liquid and vapour are flammable. Moderate fire hazard when exposed to heat or flame. Vapour forms an explosive mixture with air. Moderate explosion hazard when exposed to heat or flame. Vapour may travel a considerable distance to source of ignition. Heating may cause expansion or decomposition leading to violent rupture of containers. On combustion, may emit toxic fumes of carbon monoxide (CO).

Combustion products include: carbon dioxide (CO2)isocyanates and minor amounts of hydrogen cyanideother pyrolysis

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

products typical of burning organic material

•	
Minor Spills	 Remove all ignition sources. Clean up all spills immediately. Avoid breathing vapours and contact with skin and eyes. Control personal contact with the substance, by using protective equipment. Contain and absorb small quantities with vermiculite or other absorbent material.
	 Wipe up. Collect residues in a flammable waste container.
Major Spills	 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. 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

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Precautions for safe handling

Safe handling

Other information

- ▶ Avoid all personal contact, including inhalation.
- Wear protective clothing when risk of overexposure occurs.
- ▶ Use in a well-ventilated area.
- Prevent concentration in hollows and sumps.
- DO NOT enter confined spaces until atmosphere has been checked.
- ▶ Avoid smoking, naked lights or ignition sources.
- Avoid generation of static electricity.
- ► DO NOT use plastic buckets.
- ▶ Earth all lines and equipment.
- Use spark-free tools when handling. Avoid contact with incompatible materials.
- When handling, DO NOT eat, drink or smoke.
- ▶ Keep containers securely sealed when not in use.
- Avoid physical damage to containers.
- Always wash hands with soap and water after handling.
- Work clothes should be laundered separately.
- ▶ Use good occupational work practice.
- ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
- Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
- ▶ DO NOT allow clothing wet with material to stay in contact with skin
- ▶ Store in original containers in approved flammable liquid storage area.
- ▶ Store away from incompatible materials in a cool, dry, well-ventilated area.
- ▶ DO NOT store in pits, depressions, basements or areas where vapours may be trapped.
- ▶ No smoking, naked lights, heat or ignition sources.
- ▶ Storage areas should be clearly identified, well illuminated, clear of obstruction and accessible only to trained and authorised personnel - adequate security must be provided so that unauthorised personnel do not have access.
- Store according to applicable regulations for flammable materials for storage tanks, containers, piping, buildings, rooms, cabinets, allowable quantities and minimum storage distances.
- Use non-sparking ventilation systems, approved explosion proof equipment and intrinsically safe electrical systems.
- ▶ Have appropriate extinguishing capability in storage area (e.g. portable fire extinguishers dry chemical, foam or carbon dioxide) and flammable gas detectors.
- ▶ Keep adsorbents for leaks and spills readily available.
- ▶ Protect containers against physical damage and check regularly for leaks.
- ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

In addition, for tank storages (where appropriate):

- ▶ Store in grounded, properly designed and approved vessels and away from incompatible materials.
- For bulk storages, consider use of floating roof or nitrogen blanketed vessels; where venting to atmosphere is possible, equip storage tank vents with flame arrestors; inspect tank vents during winter conditions for vapour/ ice build-up.
- ▶ Storage tanks should be above ground and diked to hold entire contents.

Conditions for safe storage, including any incompatibilities

▶ 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

Suitable container

Avoid reaction with oxidising agents

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	isopropanol	Isopropyl alcohol	983 mg/m3 / 400 ppm	1230 mg/m3 / 500 ppm	Not Available	Not Available

Australia Exposure Standards	n-butanol	n-Butyl alcohol	Not Available	Not Available	152 mg/m3 / 50 ppm	Sk
Australia Exposure Standards	xylene	Xylene (o-, m-, p- isomers)	350 mg/m3 / 80 ppm	655 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxy- 2-propanol acetate	274 mg/m3 / 50 ppm	548 mg/m3 / 100 ppm	Not Available	Sk
Australia Exposure Standards	n-butyl acetate	n-Butyl acetate	713 mg/m3 / 150 ppm	950 mg/m3 / 200 ppm	Not Available	Not Available
Australia Exposure Standards	dibutyltin dilaurate	Tin, organic compounds (as Sn)	0.1 mg/m3	0.2 mg/m3	Not Available	Sk
Australia Exposure Standards	toluene diisocyanate	Isocyanates, all (as-NCO)	0.02 mg/m3	0.07 mg/m3	Not Available	Sen

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
isopropanol	Isopropyl alcohol	400 ppm	400 ppm	12000 ppm
n-butanol	Butyl alcohol, n-; (n-Butanol)	20 ppm	50 ppm	8000 ppm
xylene	Xylenes		Not Available	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Propylene glycol monomethyl ether acetate, alpha-isomer; (1-Methoxypropyl-2-acetate)	Not Available	Not Available	Not Available
n-butyl acetate	Butyl acetate, n-	Not Available	Not Available	Not Available
dibutyltin dilaurate	Dibutyltin dilaurate; (Dibutylbis(lauroyloxy)stannane)	1.1 mg/m3	3.3 mg/m3	5.2 mg/m3
toluene diisocyanate	Toluene diisocyanate (mixed isomers)	0.045 ppm	0.43 ppm	0.43 ppm
toluene diisocyanate	Toluene-2,4-diisocyanate; (TDI)	Not Available	Not Available	Not Available
toluene diisocyanate	Toluene-2,6-diisocyanate	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
resin	Not Available	Not Available
isopropanol	12,000 ppm	2,000 [LEL] ppm
n-butanol	8,000 ppm	1,400 [LEL] ppm
xylene	1,000 ppm	900 ppm
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available
n-butyl acetate	10,000 ppm	1,700 [LEL] ppm
filler	Not Available	Not Available
dibutyltin dilaurate	Unknown mg/m3 / Unknown ppm	25 mg/m3
toluene diisocyanate	Not Available	Not Available

MATERIAL DATA

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

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

The Odour Safety Factor (OSF) is defined as:

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

Classification into classes follows:

ClassOSF Description

- Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities
- 26-550As "A" for 50-90% of persons being distracted В
- 1-26 As "A" for less than 50% of persons being distracted
- 0.18-110-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached D
- Ε <0.18 As "D" for less than 10% of persons aware of being tested

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Odour Safety Factor(OSF) OSF=4

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.

Appropriate engineering controls

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











► Safety glasses with side shields.

- Chemical goggles.
- Eye and face protection

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 equivalent1

Skin protection

See Hand protection below

Hands/feet protection

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

Body protection

See Other protection below

Other protection

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

Thermal hazards

Not Available

Eyewash unit.

• Ensure there is ready access to a safety shower.

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	СРІ
##n-butyl	acetate
BUTYL	С
BUTYL/NEOPRENE	С
HYPALON	С
NAT+NEOPR+NITRILE	С
NATURAL RUBBER	С
NATURAL+NEOPRENE	С
NEOPRENE	С
NEOPRENE/NATURAL	С
NITRILE	С
NITRILE+PVC	С
PE	С
PE/EVAL/PE	С
PVA	С
PVC	С
PVDC/PE/PVDC	С
SARANEX-23	С
TEFLON	С
VITON	С
VITON/BUTYL	С
##toluene	diisocyanate

^{*} CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS / Class 1 P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	Air-line*	-	-
up to 100 x ES	-	A-3 P2	-
100+ x ES	-	Air-line**	-

* - Continuous-flow; ** - Continuous-flow or positive pressure demand 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)

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Clear colourless flammable liquid with a solvent odour; not miscible with water.		
Physical state	Liquid	Relative density (Water = 1)	0.95
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Applicable	Decomposition temperature	Not Available

Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	>23	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Flammable.	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Immiscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	>1	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	 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

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.

If exposure to highly concentrated solvent atmosphere is prolonged this may lead to narcosis, unconsciousness, even coma and possible death

Inhaled

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.

Ingestion

Effects on the nervous system characterise over-exposure to higher aliphatic alcohols. These include headache, muscle weakness, giddiness, ataxia, (loss of muscle coordination), confusion, delirium and coma. Gastrointestinal effects may include nausea, vomiting and diarrhoea. In the absence of effective treatment, respiratory arrest is the most common cause of death in animals acutely poisoned by the higher alcohols. Aspiration of liquid alcohols produces an especially toxic response as they are able to penetrate deeply in the lung where they are absorbed and may produce pulmonary injury. Those possessing lower viscosity elicit a greater response. The result is a high blood level and prompt death at doses otherwise tolerated by ingestion without aspiration. In general the secondary alcohols are less toxic than the corresponding primary isomers. As a general observation, alcohols are more powerful central nervous system depressants than their aliphatic analogues. In sequence of decreasing depressant potential, tertiary alcohols with multiple substituent OH groups are more potent than secondary alcohols, which, in turn, are more potent than primary alcohols. The potential for overall systemic toxicity increases with molecular weight (up to C7), principally because the water solubility is diminished and lipophilicity is increased.

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Within the homologous series of aliphatic alcohols, narcotic potency may increase even faster than lethality
Only scanty toxicity information is available about higher homologues of the aliphatic alcohol series (greater than C7) but
animal data establish that lethality does not continue to increase with increasing chain length. Aliphatic alcohols with 8 carbons
are less toxic than those immediately preceding them in the series. 10 -Carbon n-decyl alcohol has low toxicity as do the
solid fatty alcohols (e.g. lauryl, myristyl, cetyl and stearyl). However the rat aspiration test suggests that decyl and melted
dodecyl (lauryl) alcohols are dangerous if they enter the trachea. In the rat even a small quantity (0.2 ml) of these behaves
like a hydrocarbon solvent in causing death from pulmonary oedema.

Primary alcohols are metabolised to corresponding aldehydes and acids; a significant metabolic acidosis may occur. Secondary alcohols are converted to ketones, which are also central nervous system depressants and which, in he case of the higher homologues persist in the blood for many hours. Tertiary alcohols are metabolised slowly and incompletely so their toxic effects are generally persistent.

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.

Considered an unlikely route of entry in commercial/industrial environments. The liquid may produce gastrointestinal discomfort and may be harmful if swallowed. Ingestion may result in nausea, pain and vomiting. Vomit entering the lungs by aspiration may cause potentially lethal chemical pneumonitis

Skin Contact

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The material produces moderate skin irritation; evidence exists, or practical experience predicts, 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.

Exposure limits with "skin" notation indicate that vapour and liquid may be absorbed through intact skin. Absorption by skin may readily exceed vapour inhalation exposure. Symptoms for skin absorption are the same as for inhalation. Contact with eyes and mucous membranes may also contribute to overall exposure and may also invalidate the exposure standard. Open cuts, abraded or irritated skin should not be exposed to this material

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.

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

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.

Chronic

Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis). Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS] 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.

DURAPOL	TOXICITY	IRRITATION	
1012/1013/1014	Not Available	Not Available	
	TOXICITY	IRRITATION	
	Dermal (rabbit) LD50: 12792 mg/kg ^[1]	Eye (rabbit): 10 mg - moderate	
isopropanol	Inhalation (rat) LC50: 72.6 mg/L/4h ^[2]	Eye (rabbit): 100 mg - SEVERE	
	Oral (rat) LD50: 5000 mg/kg ^[2]	Eye (rabbit): 100mg/24hr-moderate	
		Skin (rabbit): 500 mg - mild	

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	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 3434.4 mg/kg ^[1]	Eye (human): 50 ppm - irritant
n-butanol	Inhalation (rat) LC50: 24 mg/L/4H ^[2]	Eye (rabbit): 1.6 mg-SEVERE
	Inhalation (rat) LC50: 8000 ppm/4hE ^[2]	Eye (rabbit): 24 mg/24h-SEVERE
	Oral (rat) LD50: 2292.3 mg/kg ^[1]	Skin (rabbit): 405 mg/24h-moderate
	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1700 mg/kg ^[2]	Eye (human): 200 ppm irritant
xylene	Inhalation (rat) LC50: 5000 ppm/4h ^[2]	Eye (rabbit): 5 mg/24h SEVERE
	Oral (rat) LD50: 4300 mg/kgt ^[2]	Eye (rabbit): 87 mg mild
		Skin (rabbit):500 mg/24h moderate
	TOXICITY	IRRITATION
propylene glycol	dermal (rat) LD50: >2000 mg/kg ^[1]	* [CCINFO]
monomethyl ether acetate, alpha-isomer	Inhalation (rat) LC50: 4345 ppm/6h ^[2]	Nil reported
, .	Oral (rat) LD50: >14.1 ml ^[1]	
	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >14080 mg/kg ^[1]	* [PPG]
n hutul aaatata	Inhalation (rat) LC50: 2000 ppm/4Hg ^[2]	Eye (human): 300 mg
n-butyl acetate	Inhalation (rat) LC50: 390 ppm/4h ^[2]	Eye (rabbit): 20 mg (open)-SEVERE
	Oral (rat) LD50: 10736 mg/kg ^[1]	Eye (rabbit): 20 mg/24h - moderate
		Skin (rabbit): 500 mg/24h-moderate
	TOXICITY	IRRITATION
dibtdiin dilaata	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 100 mg/24h -moderate
dibutyltin dilaurate	Inhalation (mouse) LC50: 0.15 mg/L/2H ^[2]	Skin (rabbit): 500 mg/24h - mild
	Oral (rat) LD50: >=33<=300 mg/kg ^[1]	
	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >12100 mg/kg ^[2]	Not Available
toluene diisocyanate	Inhalation (mouse) LC50: 14.1 ppm6 h ^[1]	
	Inhalation (mouse) LC50: 19 ppm6 h ^[1]	
	Oral (rat) LD50: >2000 mg/kg ^[1]	

DURAPOL 1012/1013/1014	None (No

Legend:

one) None: None None None None None

For isopropanol (IPA):

Acute toxicity: Isopropanol has a low order of acute toxicity. It is irritating to the eyes, but not to the skin. Very high vapor concentrations are irritating to the eyes, nose, and throat, and prolonged exposure may produce central nervous system depression and narcosis. Human volunteers reported that exposure to 400 ppm isopropanol vapors for 3 to 5 min. caused mild irritation of the eyes, nose and throat.

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

ISOPROPANOL

Although isopropanol produced little irritation when tested on the skin of human volunteers, there have been reports of isolated cases of dermal irritation and/or sensitization. The use of isopropanol as a sponge treatment for the control of fever has resulted in cases of intoxication, probably the result of both dermal absorption and inhalation. There have been a number of cases of poisoning reported due to the intentional ingestion of isopropanol, particularly among alcoholics or suicide victims. These ingestions typically result in a comatose condition. Pulmonary difficulty, nausea, vomiting, and headache accompanied by various degrees of central nervous system depression are typical. In the absence of shock, recovery

Repeat dose studies: The systemic (non-cancer) toxicity of repeated exposure to isopropanol has been evaluated in rats and mice by the inhalation and oral routes. The only adverse effects-in addition to clinical signs identified from these studies were to the kidney.

Reproductive toxicity: A recent two-generation reproductive study characterised the reproductive hazard for isopropanol associated with oral gavage exposure. This study found that the only reproductive parameter apparently affected by isopropanol exposure was a statistically significant decrease in male mating index of the F1 males. It is possible that the

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change in this reproductive parameter was treatment related and significant, although the mechanism of this effect could not be discerned from the results of the study. However, the lack of a significant effect of the female mating index in either generation, the absence of any adverse effect on litter size, and the lack of histopathological findings of the testes of the high-dose males suggest that the observed reduction in male mating index may not be biologically meaningful.

Developmental toxicity: The developmental toxicity of isopropanol has been characterized in rat and rabbit developmental toxicity studies. These studies indicate that isopropanol is not a selective developmental hazard. Isopropanol produced developmental toxicity in rats, but not in rabbits. In the rat, the developmental toxicity occurred only at maternally toxic doses and consisted of decreased foetal body weights, but no teratogenicity

Genotoxicity: All genotoxicity assays reported for isopropanol have been negative

Carcinogenicity: rodent inhalation studies were conduct to evaluate isopropanol for cancer potential. The only tumor rate increase seen was for interstitial (Leydig) cell tumors in the male rats. Interstitial cell tumors of the testis is typically the most frequently observed spontaneous tumor in aged male Fischer 344 rats. These studies demonstrate that isopropanol does not exhibit carcinogenic potential relevant to humans. Furthermore, there was no evidence from this study to indicate the development of carcinomas of the testes in the male rat, nor has isopropanol been found to be genotoxic. Thus, the testicular tumors seen in the isopropanol exposed male rats are considered of no significance in terms of human cancer risk assessment

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. 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.

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 dysonea, cough and mucus production.

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. for n-butanol

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 BAs 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.

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.

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.

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.

Reproductive toxicity: Several studies indicate that BA is not a reproductive toxicant.

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.

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.

Genotoxicity: An entire battery of negative in vitro tests and a negative in vivo micronucleus test indicate that BA is not genotoxic.

N-BUTANOL

Version No: 11.1.1.1 Print Date: 04/18/2016 **DURAPOL 1012/1013/1014** Carcinogenicity: Based upon the battery of negative mutagenicity and clastogenicity findings, BA presents a very small potential for carcinogenicity. 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. **XYLENE** 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. Reproductive effector in rats 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.

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.

PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER

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]

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] *Shin-Etsu SDS

N-BUTYL ACETATE

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 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.

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.

TOLUENE DIISOCYANATE

Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens).

Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. 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.

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

The material may produce severe 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) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration. for diisocyanates:

In general, there appears to be little or no difference between aromatic and aliphatic diisocyanates as toxicants. In addition, there are insufficient data available to make any major distinctions between polymeric (<1000 MW) and monomeric diisocyanates. Based on repeated dose studies in animals by the inhalation route, both aromatic and aliphatic diisocyanates appear to be of high concern for pulmonary toxicity at low exposure levels. Based upon a very limited data set, it appears that diisocyanate prepolymers exhibit the same respiratory tract effects as the monomers in repeated dose studies. There is also evidence that both aromatic and aliphatic diisocyanates are acutely toxic via the inhalation route. Most members of the diisocyanate category have not been tested for carcinogenic potential. Though the aromatic diisocyanates tested positive and the one aliphatic diisocyanate tested negative in one species, it is premature to make any generalizations about the carcinogenic potential of aromatic versus aliphatic diisocyanates. In the absence of more human data, it would be prudent at this time to assume that both aromatic and aliphatic diisocyanates are respiratory sensitisers. Diisocyanates are moderate to

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strong dermal sensitisers in animal studies. Skin irritation studies performed on rabbits and guinea pigs indicate no difference in the effects of aromatic versus aliphatic diisocyanates.

For monomers, effects on the respiratory tract (lungs and nasal cavities) were observed in animal studies at exposure concentrations of less than 0.005 mg/L. The experimental animal data available on prepolymeric diisocyanates show similar adverse effects at levels that range from 0.002 mg/L to 0.026 mg/L.

There is also evidence that both aromatic and aliphatic diisocyanates are acutely toxic via the inhalation route **Oncogenicity:** Most members of the diisocyanate category have not been tested for carcinogenic potential. Commercially available Poly-MDI was tested in a 2-year inhalation study in rats. The tested material contained 47% aromatic 4,4'-methylenediphenyl diisocyanate (MDI) and 53% higher molecular weight oligomers. Interim sacrifices at one year showed that males and females in the highest dose group (6 mg/m3) had treatment related histological changes in the nasal cavity, lungs and mediastinal lymph nodes. The incidence and severity of degeneration and basal cell hyperplasia of the olfactory epithelium and Bowman's gland hyperplasia were increased in males at the mid and high doses and in females at the high dose following the two year exposure period. Pulmonary adenomas were found in 6 males and 2 females, and pulmonary adenocarcinoma in one male in the high dose group. However, aliphatic hexamethylene diisocyanate (HDI) was found not to be carcinogenic in a two year repeated dose study in rats by the inhalation route. HDI has not been tested in mice by the inhalation route.

Though the oral route is not an expected route of exposure to humans, it should be noted that in two year repeated dose studies by the oral route, aromatic toluene diisocyanate (TDI) and 3,3'-dimethoxy-benzidine-4,4'-diisocyanate (dianisidine diisocyanate, DADI) were found to be carcinogenic in rodents. TDI induced a statistically significant increase in the incidence of liver tumors in rats and mice as well as dose-related hemangiosarcomas of the circulatory system and has been classified by the Agency as a B2 carcinogen. DADI was found to be carcinogenic in rats, but not in mice, with a statistically increase in the incidence of pancreatic tumors observed.

Respiratory and Dermal Sensitization: Based on the available toxicity data in animals and epidemiologic studies of humans, aromatic diisocyanates such as TDI and MDI are strong respiratory sensitisers. Aliphatic diisocyanates are generally not active in animal models for respiratory sensitization. However, HDI and possibly isophorone diisocyanate (IPDI), are reported to be associated with respiratory sensitization in humans. Symptoms resulting from occupational exposure to HDI include shortness of breath, increased bronchoconstriction reaction to histamine challenges, asthmatic reactions, wheezing and coughing. Two case reports of human exposure to IPDI by inhalation suggest IPDI is a respiratory sensitiser in humans. In view of the information from case reports in humans, it would be prudent at this time to assume that both aromatic and aliphatic diisocyanates are respiratory sensitisers. Studies in both human and mice using TDI, HDI, MDI and dicyclohexylmethane-4,4'-diisocyanate (HMDI) suggest cross-reactivity with the other diisocyanates, irrespective of whether the challenge compound was an aliphatic or aromatic diisocyanate. Diisocyanates are moderate to strong dermal sensitisers in animal studies. There seems to be little or no difference in the level of reactivity between aromatic and aliphatic diisocyanates.

Dermal Irritation: Skin irritation studies performed on rabbits and guinea pigs indicate no difference in the effects of aromatic versus aliphatic diisocyanates. The level of irritation ranged from slightly to severely irritating to the skin. One chemical, hydrogenated MDI (1,1-methylenebis-4-isocyanatocyclohexane), was found to be corrosive to the skin in guinea pigs.

Isocyanate vapours/mists are irritating to the upper respiratory tract and lungs; the response may be severe enough to produce bronchitis with wheezing, gasping and severe distress, even sudden loss of consciousness, and pulmonary oedema. Possible neurological symptoms arising from isocyanate exposure include headache, insomnia, euphoria, ataxia, anxiety neurosis, depression and paranoia. Gastrointestinal disturbances are characterised by nausea and vomiting. Pulmonary sensitisation may produce asthmatic reactions ranging from minor breathing difficulties to severe allergic attacks; this may occur following a single acute exposure or may develop without warning after a period of tolerance. A respiratory response may occur following minor skin contact. Skin sensitisation is possible and may result in allergic dermatitis responses including rash, itching, hives and swelling of extremities.

Isocyanate-containing vapours/ mists may cause inflammation of eyes and nasal passages.

Onset of symptoms may be immediate or delayed for several hours after exposure. Sensitised people can react to very low levels of airborne isocyanates. Unprotected or sensitised persons should not be allowed to work in situations allowing exposure to this material.

Acute Toxicity	✓	Carcinogenicity	0
Skin Irritation/Corrosion	~	Reproductivity	✓
Serious Eye Damage/Irritation	✓	STOT - Single Exposure	✓
Respiratory or Skin sensitisation	0	STOT - Repeated Exposure	✓
Mutagenicity	✓	Aspiration Hazard	0

I eaend:

- 🗶 Data available but does not fill the criteria for classification
- ✓ Data required to make classification available
- – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
isopropanol	EC50	384	Crustacea	42.389mg/L	3

isopropanol	EC50	96	Algae or other aquatic plants	993.232mg/L	3
sopropanol	LC50	96	Fish	183.844mg/L	3
sopropanol	NOEC	5760	Fish	0.02mg/L	4
isopropanol	EC50	48	Crustacea	12500mg/L	5
n-butanol	EC50	48	Crustacea	>500mg/L	1
n-butanol	LC50	96	Fish	88.462mg/L	3
n-butanol	BCF	24	Fish	921mg/L	4
n-butanol	EC50	504	Crustacea	18mg/L	2
n-butanol	NOEC	504	Crustacea	4.1mg/L	2
n-butanol	EC50	96	Algae or other aquatic plants	225mg/L	2
xylene	EC50	24	Crustacea	0.711mg/L	4
kylene	LC50	96	Fish	0.0013404mg/L	4
kylene	EC50	48	Crustacea	>3.4mg/L	2
kylene	EC50	72	Algae or other aquatic plants	4.6mg/L	2
xylene	NOEC	73	Algae or other aquatic plants	0.44mg/L	2
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	LC50	96	Fish	100mg/L	1
propylene glycol monomethyl ether acetate, alpha-isomer	NOEC	336	Fish	47.5mg/L	2
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	48	Crustacea	373mg/L	2
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	504	Crustacea	>100mg/L	2
n-butyl acetate	EC50	48	Crustacea	=32mg/L	1
n-butyl acetate	EC50	96	Algae or other aquatic plants	1.675mg/L	3
n-butyl acetate	EC50	96	Fish	18mg/L	2
n-butyl acetate	LC50	96	Fish	18mg/L	2
n-butyl acetate	NOEC	504	Crustacea	23mg/L	2
dibutyltin dilaurate	EC50	384	Crustacea	0.11177mg/L	3
dibutyltin dilaurate	LC50	96	Fish	0.00024mg/L	3
dibutyltin dilaurate	EC50	48	Crustacea	<0.463mg/L	2
dibutyltin dilaurate	NOEC	48	Crustacea	1.7mg/L	2
dibutyltin dilaurate	EC50	72	Algae or other aquatic plants	>1mg/L	2
oluene diisocyanate	LC50	96	Fish	ca.0.4mg/L	2
oluene diisocyanate	EC50	48	Crustacea	12.5mg/L	2
oluene diisocyanate	EC50	504	Crustacea	>=0.5mg/L	2
toluene diisocyanate	NOEC	504	Crustacea	0.5mg/L	2
toluene diisocyanate	EC50	96	Algae or other aquatic plants	3230mg/L	2

Legend:

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

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
isopropanol	LOW (Half-life = 14 days)	LOW (Half-life = 3 days)
n-butanol	LOW (Half-life = 54 days)	LOW (Half-life = 3.65 days)

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xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW	LOW
n-butyl acetate	LOW	LOW
dibutyltin dilaurate	HIGH	HIGH
toluene diisocyanate	LOW (Half-life = 1 days)	LOW (Half-life = 0.13 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
isopropanol	LOW (LogKOW = 0.05)
n-butanol	LOW (BCF = 0.64)
xylene	MEDIUM (BCF = 740)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW (LogKOW = 0.56)
n-butyl acetate	LOW (BCF = 14)
dibutyltin dilaurate	LOW (BCF = 110)
toluene diisocyanate	LOW (BCF = 5)

Mobility in soil

Ingredient	Mobility
isopropanol	HIGH (KOC = 1.06)
n-butanol	MEDIUM (KOC = 2.443)
propylene glycol monomethyl ether acetate, alpha-isomer	HIGH (KOC = 1.838)
n-butyl acetate	LOW (KOC = 20.86)
dibutyltin dilaurate	LOW (KOC = 64610000)
toluene diisocyanate	LOW (KOC = 9114)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

- ► Consult manufacturer for recycling options and recycle where possible .
- ► Consult State Land Waste Management Authority for disposal.
- ▶ Incinerate residue at an approved site.
- ▶ Recycle containers if possible, or dispose of in an authorised landfill.
- Product / Packaging
 disposal

 Containers may still present a chemical hazard/ danger when empty.

 Return to supplier for reuse/ recycling if possible.

Otherwise:

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

SECTION 14 TRANSPORT INFORMATION

Labels Required



Marine Pollutant



HAZCHEM •3Y Land transport (ADG) **UN** number 1263 Packing group PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED **UN proper shipping** MATERIAL (including paint thinning or reducing compound) **Environmental hazard** Not Applicable 3 Class Transport hazard class(es) Not Applicable Subrisk Special provisions 163 223 * Special precautions for user Limited quantity 5 L

Air transport (ICAO-IATA / DGR)

UN number	1263			
Packing group	III			
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)			
Environmental hazard	Not Applicable			
Transport hazard class(es)	ICAO/IATA Class	3		
	ICAO / IATA Subrisk	Not Applicable		
	ERG Code	3L		
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		
Packing group	III		
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	Marine Pollutant		
Transport hazard class(es)	IMDG Class 3 IMDG Subrisk 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

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

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 Inventory of Chemical Substances (AICS) Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists XYLENE(1330-20-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards Australia Inventory of Chemical Substances (AICS)

Australia Hazardous Substances Information System - Consolidated Lists International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

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

Australia Exposure Standards Australia Inventory of Chemical Substances (AICS)

Australia Hazardous Substances Information System - Consolidated Lists

N-BUTYL ACETATE(123-86-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards Australia Inventory of Chemical Substances (AICS)

Australia Hazardous Substances Information System - Consolidated Lists

DIBUTYLTIN DILAURATE(77-58-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards Australia Inventory of Chemical Substances (AICS)

Australia Hazardous Substances Information System - Consolidated Lists

TOLUENE DIISOCYANATE(26471-62-5) 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 Australia Hazardous Substances Information System - Consolidated Lists

by the IARC Monographs

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Υ
Canada - NDSL	N (propylene glycol monomethyl ether acetate, alpha-isomer; n-butanol; toluene diisocyanate; xylene; n-butyl acetate; dibutyltin dilaurate; isopropanol)
China - IECSC	Υ
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	Υ
Korea - KECI	Υ
New Zealand - NZIoC	Υ
Philippines - PICCS	Υ
USA - TSCA	Υ
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
propylene glycol monomethyl ether acetate, alpha-isomer	108-65-6, 142300-82-1, 84540-57-8
toluene diisocyanate	26471-62-5, 584-84-9, 91-08-7

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

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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