

ASIA PAINT SINGAPORE

Chemwatch: **5194-13** Version No: **2.1.1.1** Safety Data Sheet Chemwatch Hazard Alert Code: 2

Issue Date: 01/01/2025 Print Date: 01/01/2025 Initial Date: Not Available S.GHS.SGP.EN

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

Product Identifier

Product name	Ekolac 2200 2-Component PU Varnish (Base)
Synonyms	Not Available
Other means of identification	Not Available

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

Relevant identified uses

Single-component waterborne polyurethane finish.

Details of the supplier of the safety data sheet

Registered company name	ASIA PAINT SINGAPORE
Address	20 Tuas Ave 8 639235 Singapore
Telephone	+65 65 463 955
Fax	+65 65 463 855
Website	www.asiapaintsingapore.com
Email	sales@asiapaintsingapore.com

Emergency telephone number

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

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

CHEMWATCH HAZARD RATINGS

	Min	Max	
Flammability	0		
Toxicity	1		0 = Minimum
Body Contact	2		1 = Low 2 = Moderate
Reactivity	1 📃		3 = High
Chronic	0	1	4 = Extreme

GHS Classification Not Applicable

Label elements		
GHS label elements	Not Applicable	
SIGNAL WORD	NOT APPLICABLE	
Hazard statement(s)		

Not Applicable

Precautionary statement(s) Prevention

Precautionary statement(s) Response

Precautionary statement(s) Storage

Precautionary statement(s) Disposal

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
57-55-6	<5	propylene glycol
111-76-2	<5	ethylene glycol monobutyl ether
Not Available	<5	film forming aid
	balance	Ingredients determined not to be hazardous

The specific chemical identity and/or exact percentage (concentration) of composition has been withheld as a trade secret.

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	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, aerosols or combustion products are inhaled remove from contaminated area. Other measures are usually unnecessary.
Ingestion	If swallowed do NOT induce vomiting. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. Observe the patient carefully. Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. Seek medical advice.

Indication of any immediate medical attention and special treatment needed

For acute or short term repeated exposures to ethylene glycol:

- Early treatment of ingestion is important. Ensure emesis is satisfactory.
- Test and correct for metabolic acidosis and hypocalcaemia.
- Apply sustained diuresis when possible with hypertonic mannitol.
- Evaluate renal status and begin haemodialysis if indicated. [I.L.O]
- Rapid absorption is an indication that emesis or lavage is effective only in the first few hours. Cathartics and charcoal are generally not effective.
- Correct acidosis, fluid/electrolyte balance and respiratory depression in the usual manner. Systemic acidosis (below 7.2) can be treated with intravenous sodium bicarbonate solution.
- Ethanol therapy prolongs the half-life of ethylene glycol and reduces the formation of toxic metabolites. ۶
- Pyridoxine and thiamine are cofactors for ethylene glycol metabolism and should be given (50 to 100 mg respectively) intramuscularly, four times per day for 2 days.
- Magnesium is also a cofactor and should be replenished. The status of 4-methylpyrazole, in the treatment regime, is still uncertain. For clearance of the material and its metabolites, haemodialysis is much superior to peritoneal dialysis.

[Ellenhorn and Barceloux: Medical Toxicology]

It has been suggested that there is a need for establishing a new biological exposure limit before a workshift that is clearly below 100 mmol ethoxy-acetic acids per mole creatinine in morning urine of people occupationally exposed to ethylene glycol ethers. This arises from the finding that an increase in urinary stones may be associated with such exposures. Laitinen J., et al: Occupational & Environmental Medicine 1996; 53, 595-600

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

		 There is no restriction on the type of extinguisher which may be used. Use extinguishing media suitable for surrounding area.
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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. Wear breathing apparatus plus protective gloves in the event of a fire. Prevent, by any means available, spillage from entering drains or water courses. Use fire fighting procedures suitable for surrounding area. DO NOT approach containers suspected to be hot. Cool fire exposed containers with water spray from a protected location. If safe to do so, remove containers from path of fire. Equipment should be thoroughly decontaminated after use.	
Fire/Explosion Hazard	 Non combustible. Not considered to be a significant fire risk. Expansion or decomposition on heating may lead to violent rupture of containers. 	

Decomposes on heating and may produce toxic fumes of carbon monoxide (CO).

May emit acrid smoke.

Decomposition may produce toxic fumes of; carbon dioxide (CO2), other pyrolysis products typical of burning organic materialMay emit poisonous fumes. May emit corrosive fumes.

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Minor Spills	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 spill with sand, earth, inert material or vermiculite. Wipe up. Place in a suitable, labelled container for waste disposal.
Major Spills	Moderate hazard. Clear area of personnel and move upwind. Alert Fire Brigade and tell them location and nature of hazard. Wear breathing apparatus plus protective gloves. Prevent, by any means available, spillage from entering drains or water course. Stop leak if safe to do so. Contain spill with sand, earth or vermiculite. Collect recoverable product into labelled containers for recycling. Neutralise/decontaminate residue (see Section 13 for specific agent). Collect solid residues and seal in labelled drums for disposal. Wash area and prevent runoff into drains. After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. If contamination of drains or waterways occurs, advise emergency services.
	Personal Protective Equipment advice is contained in Section 8 of the SDS.

SECTION 7 HANDLING AND STORAGE

Precautions for safe handlin	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. Avoid contact with moisture. 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. Launder contaminated clothing before re-use. 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 are maintained.		
Other information	Store in original containers. Keep containers securely sealed. Store in a cool, dry, well-ventilated area. Store away from incompatible materials and foodstuff containers. Protect containers against physical damage and check regularly for leaks. Observe manufacturer's storage and handling recommendations contained within this SDS.		

Conditions for safe storage, including any incompatibilities

Suitable container	Polyethylene or polypropylene container. Packing as recommended by manufacturer. Check all containers are clearly labelled and free from leaks.	
Storage incompatibility	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
Singapore Permissible Exposure Limits of Toxic Substances	ethylene glycol monobutyl ether	2-Butoxyethanol (EGBE)	121 mg/m3 / 25 ppm	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
propylene glycol	Propylene glycol; (1,2-Propanediol)	30 mg/m3	1300 mg/m3	7900 mg/m3
ethylene glycol monobutyl ether	Butoxyethanol, 2-; (Glycol ether EB)	20 ppm	20 ppm	700 ppm

Ingredient	Original IDLH	Revised IDLH
propylene glycol	Not Available	Not Available
ethylene glycol monobutyl ether	700 ppm	700 [Unch] ppm
film forming aid	Not Available	Not Available

Exposure controls			
Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the haz effective in protecting workers and will typically be independent of worker interactions to provide this I The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Cc Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensis An approved self contained breathing apparatus (SCBA) may be required in some situations. Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contain Type of Contaminant: solvent, vapours, degreasing etc., evaporating from tank (in still air). aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, acid furmes, pickling (released at low velocity into zone of active generation) direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas dis zone of rapid air motion) grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial veloa air motion). Within each range the appropriate value depends on: Lower end of the range 1: Room air currents minimal or favourable to capture 2: Contaminants of low toxicity or of nuisance value only. 3: Intermittent, low production. 4: Large hood or large air mass in motion Simple theory shows that air velocity falls rapidly with	high level of protection. a risk. the worker and ventilation that stra properly. The design of a ventilation pure adequate protection. workplace possess varying "escap- minant. welding, spray drift, plating charge (active generation into city into zone of very high rapid Upper end of the range 1: Disturbing room air currents 2: Contaminants of high toxicit 3: High production, heavy use 4: Small hood-local control onl raction pipe. Velocity generally dec should be adjusted, accordingly, at minimum of 1-2 m/s (200-400 (/mini- post), producing performance deficit	tegically "adds" and system must match uate protection. De" velocities which, in Air Speed: 0.25-0.5 m/s (50-100 f/min.) 0.5-1 m/s (100-200 f/min.) 1-2.5 m/s (200-500 f/min.) 2.5-10 m/s (500-2000 f/min.) 2.5-10 m/s (500-2000 f/min.) 2.5-10 m/s (500-2000 f/min.)
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 irrita lenses or restrictions on use, should be created for each workplace or task. This should include a chemicals in use and an account of injury experience. Medical and first-aid personnel should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove or at the first signs of eye redness or irritation - lens should be removed in a clean environment only Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] 	a review of lens absorption and ac trained in their removal and suitab contact lens as soon as practicable.	lsorption for the class of le equipment should be Lens should be removed
Skin protection	See Hand protection below		
Hands/feet protection	 Wear chemical protective gloves, e.g. PVC. Wear safety footwear or safety gumboots, e.g. Rubber NOTE: The material may produce skin sensitisation in predisposed individuals. Care must be taken, wh all possible skin contact. Contaminated leather items, such as shoes, belts and watch-bands should be removed an The selection of suitable gloves does not only depend on the material, but also on further marks of q the chemical is a preparation of several substances, the resistance of the glove material can not be of to the application. The exact break through time for substances has to be obtained from the manufacturer of the protect choice. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gleve 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 when prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or 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 2161.10.1 or national equivalent) is recommended.	Id destroyed. uality which vary from manufacture calculated in advance and has the ive gloves and has to be observed oves include: national equivalent). or higher (breakthrough time greate	er to manufacturer. Where refore to be checked prior when making a final er than 240 minutes

	Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. Contaminated gloves should be replaced. 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. P.V.C. apron. Barrier cream. Skin cleansing cream. Eye wash unit.
Thermal hazards	Not Available

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

The effect(s) of the following substance(s) are taken into account in the computer*generated* selection:

Ekolac 2200 2-Component PU Varnish (Base)

Material	CPI
BUTYL	С
NAT+NEOPR+NITRILE	С
NATURAL RUBBER	С
NEOPRENE	С
NITRILE	С
PE/EVAL/PE	С
PVA	С
PVC	С
SARANEX-23	С
##ethylene glycol monobutyl	ether

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Clear or coloured liquid; mixes with water. Appearance Physical state Liquid Relative density (Water = 1) ~1 Partition coefficient Odour Not Available Not Available n-octanol / water Auto-ignition temperature Odour threshold Not Available Not Applicable (°C) Decomposition pH (as supplied) Not Available Not Available temperature Melting point / freezing Not Available Viscosity (cSt) Not Available point (°C) Initial boiling point and Not Available Molecular weight (g/mol) Not Applicable boiling range (°C) Flash point (°C) Not Applicable Not Available Taste Evaporation rate Explosive properties Not Available Not Available Flammability Not Applicable **Oxidising properties** Not Available Surface Tension (dyn/cm or Upper Explosive Limit (%) Not Applicable Not Available mN/m) Lower Explosive Limit (%) Not Applicable Volatile Component (%vol) 6-10 Vapour pressure (kPa) Not Available Not Available Gas group Solubility in water (g/L) Miscible pH as a solution (1%) Not Available

VOC g/L

Not Available

SECTION 10 STABILITY AND REACTIVITY

Not Available

Vapour density (Air = 1)

Respiratory protection

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

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 5 x ES	A-AUS / Class 1 P2	-	A-PAPR-AUS / Class 1 P2
up to 25 x ES	Air-line*	A-2 P2	A-PAPR-2 P2
up to 50 x ES	-	A-3 P2	-
50+ x ES	-	Air-line**	-

^ - Full-face

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

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

Inhaled	The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.		
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Considered an unlikely route of entry in commercial/industrial environments		
Skin Contact	The material is not thought to produce adverse health effects or skin irritation following contact (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream, through, for example, cuts, abrasions or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.		
Eye	Although the liquid is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).		
Chronic	Substance accumulation, in the human body, may occur and may cause There is limited evidence that, skin contact with this product is more likely to population. There is some evidence from animal testing that exposure to this materi Based on experience with similar materials, there is a possibility that exposure toxic effects.	cause a sensitisation reaction in some persons compared to the general	
Ekolac 2200 2-Component	ΤΟΧΙΟΙΤΥ	IRRITATION	
PU Varnish (Base)	Not Available	Not Available	
	тохісіту	IRRITATION	
	Dermal (rabbit) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 100 mg - mild	
propylene glycol	Oral (rat) LD50: 20000 mg/kgd ^[2]	Eye (rabbit): 500 mg/24h - mild	
		Skin(human):104 mg/3d Intermit Mod	
		Skin(human):500 mg/7days mild	
	тохісіту	IRRITATION	
ethylene glycol monobutyl	dermal (rat) LD50: >2000 mg/kg ^[1]	* [Union Carbide]	
ether	Inhalation (rat) LC50: 450 ppm/4H ^[2]	Eye (rabbit): 100 mg SEVERE	
	Oral (rat) LD50: 250 mg/kg ^[2]	Eye (rabbit): 100 mg/24h-moderate	
		Skin (rabbit): 500 mg, open; mild	
Legend:	 Value obtained from Europe ECHA Registered Substances - Acute toxicity extracted from RTECS - Register of Toxic Effect of chemical Substances 	(2.* Value obtained from manufacturer's SDS. Unless otherwise specified data	
PROPYLENE GLYCOL	scaling and thickening of the skin. The acute oral toxicity of propylene glycol is very low, and large quantities generally occurs only at plasma concentrations over 1 g/L, which requires impossible to reach toxic levels by consuming foods or supplements, which related to either inappropriate intravenous administration or accidental inge also low. Because of its low chronic oral toxicity, propylene glycol was class safe" (GRAS) for use as a direct food additive. Prolonged contact with propylene glycol is essentially non-irritating to the s produce slight transient conjunctivitis (the eye recovers after the exposure i respiratory tract irritation. Inhalation of the propylene glycol vapours appear human experience indicates that inhalation of propylene glycol vapours appear human experience indicates that inhalation for emergency eye wash statii Propylene glycol is metabolised in the human body into pyruvic acid (a no acid (handled by ethanol-metabolism), lactic acid (a normal acid generally substance). Propylene glycol shows no evidence of being a carcinogen or of being Research has suggested that individuals who cannot tolerate propylene glycen spiratory.	s removed). Exposure to mists may cause eye irritation, as well as upper s to present no significant hazard in ordinary applications. However, limited be irritating to some individuals It is therefore recommended that propylene contact with the spray mists of these materials is likely, such as fogs for ons. rmal part of the glucose-metabolism process, readily converted to energy), acetic abundant during digestion), and propionaldehyde (a potentially hazardous	

	One study strongly suggests a connection between airborne concentrations of propylene glycol in houses and development of asthma and allergic
	reactions, such as rhinitis or hives in children Another study suggested that the concentrations of PGEs (counted as the sum of propylene glycol and glycol ethers) in indoor air, particularly bedroom air, the literature is the sum of the sum
	is linked to increased risk of developing numerous respiratory and immune disorders in children, including asthma, hay fever, eczema, and allergies, with increased risk ranging from 50% to 180%. This concentration has been linked to use of water-based paints and water-based system cleansers.
	Patients with vulvodynia and interstitial cystitis may be especially sensitive to propylene glycol. Women suffering with yeast infections may also notice that some over the counter creams can cause intense burning. Post menopausal women who require the use of an eostrogen cream may notice that brand
	name creams made with propylene glycol often create extreme, uncomfortable burning along the vulva and perianal area. Additionally, some electronic cigarette users who inhale propylene glycol vapor may experience dryness of the throat or shortness of breath. As an alternative, some suppliers will put
	Vegetable Glycerin in the "e-liquid" for those who are allergic (or have bad reactions) to propylene glycol. Adverse responses to intravenous administration of drugs which use PG as an excipient have been seen in a number of people, particularly with large
	dosages thereof. Responses may include "hypotension, bradycardia QRS and T abnormalities on the ECG, arrhythmia, cardiac arrest, serum
	hyperosmolality, lactic acidosis, and haemolysis". A high percentage (12% to 42%) of directly-injected propylene glycol is eliminated/secreted in urine unaltered depending on dosage, with the remainder appearing in its glucuronide-form. The speed of renal filtration decreases as dosage increases, which
	may be due to propylene glycol's mild anesthetic / CNS-depressant -properties as an alcohol. In one case, intravenous administration of propylene glycol- suspended nitroglycerin to an elderly man may have induced coma and acidosis.
	Propylene glycol is an approved food additive for dog food under the category of animal feed and is generally recognized as safe for dogs with an LD50 of
	9 mL/kg. The LD50 is higher for most laboratory animals (20 mL/kg) Similarly, propylene glycol is an approved food additive for human food as well. The exception is that it is prohibited for use in food for cats due to links to
	Heinz body anemia.
	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 on contact skin redness, swelling, the production of vesicles,
	scaling and thickening of the skin. For ethylene glycol monoalkyl ethers and their acetates (EGMAEs):
	Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates.
	EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are
	transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers.
	Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour
	concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 >
	2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category
	members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the
	exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and
	butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed
	in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE <i>in vitro</i> than those of rats.
	Repeat dose toxicity: The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more
	sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA <i>in vitro</i> and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans,
	pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA <i>in vitro.</i> Mutagenicity: In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in S.
	typhimunium strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. In
	vitro cytogenicity and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and in vivo micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.
	Carcinogenicity: In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available,
ETHYLENE GLYCOL MONOBUTYL ETHER	there was no significant hazard for human carcinogenicity
	Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity
	studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes).
	Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531,
	1062, or 2125 mg/m3 and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m3), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m3), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m3) indicate that the members of the category are not teratogenic.
	The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m3 (rabbit-EGPE), 100 ppm or 425 mg/m3 (rat-EGPE), 50 ppm or 241 mg/m3 (rat EGBE) and 100 ppm or 483 mg/m3 (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m3 (rat and rabbit-EGHE).
	Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetoxicity in the form of poorly ossified or
	unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species.
	At least one researcher has stated that the reproductive effects were less than that of other monoalkyl ethers of ethylene glycol. Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility.
	Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions
	(1). The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that
	2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of erythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-
	year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration
	dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation
	produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration -dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular
	carcinoma.
	1: NTP Toxicology Program Technical report Series 484, March 2000. For ethylene glycol:
	Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body

respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol.

dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glycoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glycoxylate; glycoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic

acid can generate CO2, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO2, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested. Respiratory Effects. Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases) Cardiovascular Effects. Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12-24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol. Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases. Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown Gastrointestinal Effects. Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition. Musculoskeletal Effects. Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tendemess and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia. Hepatic Effects. Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. Renal Effects. Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, anuria, and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near normal renal function can return with adequate supportive therapy. Metabolic Effects. One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate). Neurological Effects: Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion. Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months. Reproductive Effects: Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration. Developmental Effects: The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embyrotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight. Cancer: No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol. Genotoxic Effects: Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available in vivo and in vitro laboratory studies provide consistently negative genotoxicity results for ethylene glycol. NOTE: Changes in kidney, liver, spleen and lungs are observed in animals exposed to high concentrations of this substance by all routes. ** ASCC (NZ) SDS

Acute Toxicity 0 Carcinogenicity \bigcirc \bigcirc \odot Skin Irritation/Corrosion Reproductivity Serious Eye \bigcirc \bigcirc STOT - Single Exposure Damage/Irritation Respiratory or Skin \odot STOT - Repeated Exposure \bigcirc sensitisation \odot \odot Mutagenicity Aspiration Hazard Data required to make classification available Legend:

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 \mathbf{X} – Data available but does not fill the criteria for classification

🚫 – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

DO NOT discharge into sewer or waterways

Persistence and degradability

Ingredient

Persistence: Water/Soil

Persistence: Air

propylene glycol	LOW	LOW
ethylene glycol monobutyl ether	LOW (Half-life = 56 days)	LOW (Half-life = 1.37 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
propylene glycol	LOW (BCF = 1)
ethylene glycol monobutyl ether	LOW (BCF = 2.51)
Mobility in soil	

Ingredient	Mobility
propylene glycol	HIGH (KOC = 1)
ethylene glycol monobutyl ether	HIGH (KOC = 1)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: Reduction Reuse Recycling Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Product / Packaging Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. disposal DO NOT allow wash water from cleaning or process equipment to enter drains. It may be necessary to collect all wash water for treatment before disposal. In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. Where in doubt contact the responsible authority. Recycle wherever possible. Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. > Dispose of by: burial in a land-fill specifically licenced to accept chemical and / or pharmaceutical wastes or incineration in a licenced apparatus (after admixture with suitable combustible material). Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant NO

Land transport (UN): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

SECTION 15 REGULATORY INFORMATION

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

PROPYLENE GLYCOL(57-55-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Not Applicable

ETHYLENE GLYCOL MONOBUTYL ETHER(111-76-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

Singapore Permissible Exposure Limits of Toxic Substances

National Inventory	Status
Australia - AICS	Υ
Canada - DSL	Υ
Canada - NDSL	N (propylene glycol; ethylene glycol monobutyl ether)
China - IECSC	Υ
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	Υ
Korea - KECI	Υ
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y

Continued...

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

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

A list of reference resources used to assist the committee may be found at:

www.chemwatch.net

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.

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. TEL (+61 3) 9572 4700.

