CUTEK Interior

Chemisys Manufacturing Pty Ltd

Chemwatch Hazard Alert Code: **2**

Version No: **1.25**
Safety Data Sheet according to WHS and ADG requirements

Issue Date: **09/02/2021**
Print Date: **09/02/2021**
L.GHS.AUS.EN

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

Product Identifier

Product name	CUTEK Interior
Chemical Name	Not Applicable
Synonyms	Not Available
Other means of identification	Not Available

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

Relevant identified uses	Interior matt coating for wood.

Details of the supplier of the safety data sheet

Registered company name	**Chemisys Manufacturing Pty Ltd**
Address	72 Chetwynd St QLD 4129 Australia
Telephone	+617 3188 5242
Fax	+617 3073 3919
Website	www.cutek.com.au
Email	admin@chemisys.com

Emergency telephone number

Association / Organisation	**Chemisys Manufacturing Pty Ltd**
Emergency telephone numbers	+617 3188 5246
Other emergency telephone numbers	131 126

SECTION 2 Hazards identification

Classification of the substance or mixture

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

ChemWatch Hazard Ratings

	Min	Max
Flammability	0	
Toxicity	0	
Body Contact	2	
Reactivity	0	
Chronic	2	

0 = Minimum
1 = Low
2 = Moderate
3 = High
4 = Extreme

Poisons Schedule	Not Applicable
Classification [1]	Eye Irritation Category 2A, Skin Corrosion/Irritation Category 2, Chronic Aquatic Hazard Category 3

Page 1 continued...

Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

Hazard pictogram(s)	
Signal word	**Warning**

Hazard statement(s)

H319	Causes serious eye irritation.
H315	Causes skin irritation.
H412	Harmful to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection/hearing protection/...

Precautionary statement(s) Response

P321	Specific treatment (see ... on this label).
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.
P302+P352	IF ON SKIN: Wash with plenty of water.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
34590-94-8	<10	dipropylene glycol monomethyl ether
78-51-3	<10	tris(2-butoxyethyl)phosphate
41556-26-7	<1	bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate
82919-37-7	<1	methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate
Not Available	<1	Fluorosurfactant

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.

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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	▸ Immediately give a glass of water. ▸ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.
Atropine sulfate, usually in doses of 600 microgram may be given intravenously, intramuscularly, or subcutaneously to control the muscarinic effects of choline esterase inhibitors. Supportive treatment may be required.
MARTINDALE: The Extra Pharmacopoeia, Twenty-ninth Edition
While other antimuscarinic agents (e.g., scopolamine) can counteract the effects of cholinesterase inhibitors, their inherent toxic effects in patients who do not have cholinesterase inhibitor poisoning have led to their rejection in favor of atropine. Glycopyrrolate in doses of 1-2 mg, I.V., (0.025 mg/kg in children) has been suggested as an alternative to atropine, and is said to have fewer CNS side effects. However, its use has not been extensively evaluated.
Atropine works by competitively occupying muscarinic receptor sites, thus reducing the effects of excessive acetylcholine on these sites brought about by cholinesterase inhibition.
Atropine is not thought to have significant effect on nicotinic receptors, and thus does not counteract fasciculations, weakness, or flaccid paralysis. Thus, even when given sufficient doses of atropine, patients may need artificial ventilation, sometimes for weeks.
A number of authors have recommended the “atropine challenge” as an aid to diagnosis.
When given to a normal person who has not been exposed to cholinesterase inhibitors, a 2 mg dose of atropine (0.025-0.050/kg in pediatric cases) causes:

- A dry mouth.
- An increase in heart rate of about 35 beats/minute (which is usually not noticed by the recipient) within 3-5 minutes of an I.V. dose, and a maximal increase in heart rate of about 35-45 beats/minute with I.M. or autoinjector administration, respectively, within about 35-45 minutes (the longer being with I.M. injection).
- Blurred near-vision.
- Dry, hot skin.
- Mydriasis (pupillary dilation).

Most of these effects will dissipate within 4-6 hours, except blurred near-vision which may persist for 24 hours.
It has been suggested that when these physiological changes do not occur with this dose (sometimes referred to as an atropine challenge), this is indicative of cholinesterase inhibitor toxicity.
Cautions

- If miosis (pupillary constriction) is due to direct conjunctival vapor exposure, it is relatively unresponsive to parenteral atropine. Although, it does respond to topical administration).
- In 2-13% of cases of cholinesterase inhibitor toxicity, mydriasis (pupillary dilation) --- rather than miosis (pupillary constriction), and tachycardia --- rather than bradycardia (3-77% of cases),.may be a presenting signs.
- One author points out that this strategy has never been empirically tested and may not be very sensitive or specific (Parenteral atropine is not generally recommended for those whose sole manifestation of toxicity is miosis (pupillary constriction).
- Some cases of mild to moderate poisonings may improve with these doses of atropine. Thus, signs of atropinization do not always exclude the presence of cholinesterase inhibitor toxicity.

In approximate order of preference, the following routes of administration can be used for the administration of atropine

1. Intravenous: bolus, followed by I.V. drip. .
1. Intraosseous: (American Heart Association 2005) bolus, followed by continuous infusion.
1. Military MARK I atropine autoinjector: Although intravenous injection is the preferred route of administration, use of the autoinjector may be more practical in the field, where it can be rapidly administered even through clothing.) Blood levels are achieved more rapidly than by other forms of IM injection. Note that each MARK I kit contains an atropine autoinjector, containing 2 mg of atropine plus another autoinjector containing 600 mg of 2-PAM. Paediatric atropine autoinjector syringes are available in 0.5 mg and 1 mg sizes.
1. Intramuscular: Research for this Case Study did not turn up any comparisons of intramuscular with inhalation routes of atropine administration.
1. Inhalation: by nebulised inhalation or via the intratracheal route. The intratracheal route can be used, but absorption is notably less complete and less reliable than the intravenous or intraosseous routes, which are preferred. The optimal intratracheal dose is unknown, but is typically administered in an amount 2-2½ times the intravenous dose. The American Heart Association recommends that the dose be diluted in 5-10 ml water or normal saline. American Heart Association 2005; American Heart Association 2005)
1. Oral: use has been reported after I.V. administration became unnecessary.
1. Ophthalmic: Anticholinergic eye drops (e.g., atropine or homatropine) have been recommended for severe eye pain caused by miosis (pupillary constriction), and secondary reflex nausea and vomiting, but may result in blurred vision. However, one author questions whether there is enough evidence to recommend this practice.

Tachycardia should not be used as an end-point, because it sometimes is a nicotinic manifestation of toxicity.
Resolution of miosis [Miosis has been defined as pupillary diameter of <3 mm in the dark, along with sluggish or absent response to light] should not be used as an end-point, because:

- Miosis (pupillary constriction) from systemic exposure may be a late finding.
- When miosis pupillary constriction) is present, it may be resistant to systemic atropine therapy.
- Miosis (pupillary constriction) may reflect only localized ophthalmic exposure to vapor without systemic effects.
- Pupils are of normal size in a significant minority of poisoned patients (20% in one series).
- Toxic patients may present with mydriasis (pupillary dilation) due to occasional dominance of nicotinic effects from cholinesterase inhibitors.

Case Studies in Environmental Medicine (CSEM) Cholinesterase Inhibitors Including Insecticides and Chemical Warfare Nerve Agents Part 4: The Cholinergic Toxidrome; Section 11: Management of the Cholinergic Toxidrome Management Strategy 3: Medications Atropine Agency for Toxic Substance and Disease Registry ATSDR (USA)

Continued...

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.

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.

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 a significant fire risk, however containers may burn. May emit corrosive fumes.
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	- 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 handling

Safe handling	- Avoid all personal contact, including inhalation. - Wear protective clothing when risk of exposure occurs. - Use in a well-ventilated area.

Continued...

	‣ 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. ‣ **DO NOT** allow clothing wet with material to stay in contact with skin
Other information	

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	None known

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X — Must not be stored together
0 — May be stored together with specific preventions
+ — May be stored together

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	dipropylene glycol monomethyl ether	(2-Methoxymethylethoxy) propanol	50 ppm / 308 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
dipropylene glycol monomethyl ether	Dipropylene glycol methyl ether	150 ppm	1700* ppm	9900** ppm
tris(2-butoxyethyl)phosphate	Butoxyethanol phosphate, 2-	9 mg/m3	99 mg/m3	590 mg/m3

Ingredient	Original IDLH	Revised IDLH
dipropylene glycol monomethyl ether	600 ppm	Not Available
tris(2-butoxyethyl)phosphate	Not Available	Not Available
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	Not Available	Not Available
methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
tris(2-butoxyethyl)phosphate	E	≤ 0.1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

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Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	D	> 0.1 to ≤ 1 ppm
methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	D	> 0.1 to ≤ 1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

for dipropylene glycol monomethyl ether:
The TLV-TWA and STEL recommendations were thought to be sufficiently low to prevent objectionable irritation and provide a considerable safety factor against CNS impairment. In view of the large dose required to cause weight loss and narcosis in rabbits the skin notation is being reviewed.
Probable minimum concentration that may cause minor nasal irritation is about 35 ppm.
Probable minimum concentration that may cause tolerable eye, throat, and respiratory irritation is about 75 ppm.
Lowest concentration at which vapour is rated tolerable 80 ppm.
Based on these criteria it is possible that an occasional person may find the vapour of dipropylene glycol monomethyl ether intolerable at the recommended 100 ppm TLV.
Dermal absorption of the substance under specific experimental conditions led to narcotic effects and consequent deaths. However, only slight narcotic effects were seen after several hours exposure of rats to
aerosols which wet the fur of animals. Rabbits tolerated dermal application of 3.0 ml/kg per day without effects. A skin designation is thought to be unnecessary by the MAK committee, in contrast with others.

Exposure controls

Appropriate engineering controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "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.

General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in specific circumstances. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 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.

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Personal protection	
Eye and face protection	‣ Safety glasses with side shields. ‣ Chemical goggles. ‣ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	‣ Wear chemical protective gloves, e.g. PVC. ‣ Wear safety footwear or safety gumboots, e.g. Rubber The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Body protection	See Other protection below
Other protection	‣ Overalls. ‣ P.V.C apron. ‣ Barrier cream. ‣ Skin cleansing cream. ‣ Eye wash unit.

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:

Continued...

CUTEK Interior

Material	CPI
BUTYL	C
CPE	C
NATURAL RUBBER	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
SARANEX-23	C
SARANEX-23 2-PLY	C
TEFLON	C
VITON	C
VITON/CHLOROBUTYL	C
VITON/NEOPRENE	C

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	White

Physical state	Liquid	**Relative density (Water = 1)**	1.03
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Continued...

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 adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.
Ingestion	The material has **NOT** been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.
Eye	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Long-term exposure to the product is not thought to produce chronic effects adverse to health (as classified by EC Directives using animal models); nevertheless exposure by all routes should be minimised as a matter of course. Repeated or prolonged exposures to cholinesterase inhibitors produce symptoms similar to acute effects. In addition workers exposed repeatedly to these substances may exhibit impaired memory and loss of concentration, severe depression and acute psychosis, irritability, confusion, apathy, emotional lability, speech difficulties, headache, spatial disorientation, delayed reaction times, sleepwalking, drowsiness or insomnia. An influenza-like condition with nausea, weakness, anorexia and malaise has been described. There is a growing body of evidence from epidemiological studies and from experimental laboratory studies that short-term exposure to some cholinesterase-inhibiting insecticides may produce behavioural or neuro-chemical changes lasting for days or months, presumably outlasting the cholinesterase inhibition. Although the number of adverse effects following humans poisonings subsides, there are still effects in some workers months after cholinesterase activity returns to normal. These long-lasting effects include blurred vision, headache, weakness, and anorexia. The neurochemistry of animals exposed to chlorpyrifos or fenthion is reported to be altered permanently after a single exposure. These effects may be more severe in developing animals where both acetyl- and butyrylcholinesterase may play an integral part in the development of the nervous system. *Padilla S., The Neurotoxicity of Cholinesterase-Inhibiting Insecticides: Past and Present Evidence Demonstrating Persistent Effects. Inhalation Toxicology 7:903-907, 1995*

CUTEK Interior	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

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dipropylene glycol monomethyl ether	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 9.50 mg/kg[1]	Eye (human): 8 mg - mild
	Oral(Rat) LD50; 5.130 mg/kg[1]	Eye (rabbit): 500 mg/24hr - mild
		Skin (rabbit): 238 mg - mild
		Skin (rabbit): 500 mg (open)-mild
tris(2-butoxyethyl)phosphate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2.04 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
	Inhalation(Rat) LC50; >6.4 mg/l4hrs[2]	Skin (rabbit): 500 mg/24h mild
	Oral(Rat) LD50; <5000 mg/kg[1]	Skin: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	**TOXICITY**	**IRRITATION**
	Oral(Rat) LD50; =2369-3920 mg/kg[2]	Not Available
methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*	

DIPROPYLENE GLYCOL MONOMETHYL ETHER	for propylene glycol ethers (PGEs): Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM). Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids. Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product. Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body. As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces. As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating None are skin sensitisers. In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested). Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43

Continued...

	ppm). In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members. One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health. In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity. The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.
TRIS(2-BUTOXYETHYL)PHOSPHATE	Oral (rat) LD50: 3000-9490 mg/kg Eye (rabbit): 500 mg/24h mild Inhalation (rat) LC50: 5->6.4 mg/l 4 hrs for tris(2-butoxyethyl)phosphate (TBEP): TBEP has been found associated with particulate matter in the air of offices. The acute systemic mammalian toxicity and irritation potential are low. Several subchronic studies in laboratory animals have shown that the liver is the target organ for TBEP toxicity. One study in male Sprague-Dawley rats suggested that TBEP might cause focal myocarditis. Neurotoxic effects in rats after single doses of TBEP are inconsistent. In rats repeatedly given high doses by gavage, TBEP decreased nerve conduction velocity and increased the refractory period. The increased refractory period and the decreased conduction velocity were dose-related in females, but in males the maximum effect appears to have been reached by the low dose, suggesting that the magnitude of the maximum attainable neurophysiological changes is modest. Most of the treated animals showed the presence of some degenerative myelin sheaths accompanied by axonal swelling and an advanced stage of degeneration, indicated by the presence of lamellated electron-dense inclusions in unmyelinated nerve fibres In a 14-week oral toxicity study with TBEP, Wistar rats (5 weeks old, male and female, 15 rats/group) were given a diet containing 0, 0.3, 3 or 30 g TBEP/kg. Suppression of body weight gain was observed in both sexes at 30 g/kg. Serum cholinesterase activity was significantly decreased in both sexes at 3 and 30 g/kg, and serum gamma-glutamyl transferase activity was significantly increased in both sexes at 30 g/kg. Examination of the liver in both sexes revealed moderate periportal hepatocyte swelling in male rats at 30 g/kg after 14 weeks of exposure but this change was not found in male rats given 3 g/kg or less. The no-observed-effect level (NOEL) of TBEP in the diet was 0.3 g/kg diet (for males 20 mg/kg body weight per day and for females 22 mg/kg body weight per day. The NOAEL of this study to be 3 g/kg diet. In a gavage study, groups of 12 male and 12 female Sprague-Dawley rats were administered 0, 0.25 or 0.5 ml/kg body weight undiluted TBEP on 5 days/week for 18 weeks. During the first week, two high-dose females showed muscular weakness and ataxia which had disappeared by the end of the fourth week. After about 7 weeks, nearly all animals exhibited some signs of toxicity, which seemed to be treatment related. All treated animals appeared less active, and one female died during week 13. Breathing difficulties and ataxia were present in several males and females in both treatment groups, though the low-dose group was affected to a lesser extent. Tremors, piloerection, lacrimation and increased urination were observed in both males and females of the high-dose group. After the last dose, the clinical signs observed in the high-dose group decreased in intensity. High-dose females had significantly elevated level of serum gammaglutamyltransferase. Red cell acetylcholinesterase (AchE) activity was significantly reduced in males at both doses. TBEP did not cause delayed neurotoxicity in hens but did inhibit brain and plasma cholinesterases. Based on an 18-week repeated dose study in rats, the no-observed-effect level (NOEL) for liver effects was reported to be 15 mg/kg body weight per day, while the lowest-observed-effect level (LOEL) was 150 mg/kg body weight per day. The long-term toxicity and carcinogenicity of TBEP have not been studied. Bacterial and mammalian cell tests for gene mutation gave negative results, but no tests for chromosomal damage have been reported. Teratogenicity was not observed in one study in rats. Other aspects of reproductive toxicity have not been reported. A Repeat Human Insult Patch Test indicated no skin sensitization and minimal skin irritation.
METHYL 1,2,2,6,6-PENTAMETHYL-4-PIPERIDYL SEBACATE	No significant acute toxicological data identified in literature search.
DIPROPYLENE GLYCOL MONOMETHYL ETHER & TRIS(2-BUTOXYETHYL)PHOSPHATE	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.

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	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
BIS(1,2,2,6,6-PENTAMETHYL-4-PIPERIDYL)SEBACATE & METHYL 1,2,2,6,6-PENTAMETHYL-4-PIPERIDYL SEBACATE	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.

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

Legend: ✗ *– Data either not available or does not fill the criteria for classification*
✓ *– Data available to make classification*

SECTION 12 Ecological information

Toxicity

CUTEK Interior

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

dipropylene glycol monomethyl ether

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	1000mg/L	2
EC50	48	Crustacea	1930mg/L	2
EC50	72	Algae or other aquatic plants	>969mg/L	2
NOEC	528	Crustacea	>=0.5mg/L	2

tris(2-butoxyethyl)phosphate

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	-10.4-12mg/L	4
EC50	48	Crustacea	53mg/L	2
EC50	72	Algae or other aquatic plants	33mg/L	2
BCF	168	Fish	1mg/L	4
EC0	48	Crustacea	10mg/L	2
NOEL	504	Not Available	0.0223686-mg/L	4

bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

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

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

Continued...

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
dipropylene glycol monomethyl ether	HIGH	HIGH
tris(2-butoxyethyl)phosphate	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
dipropylene glycol monomethyl ether	LOW (BCF = 100)
tris(2-butoxyethyl)phosphate	LOW (BCF = 5.8)

Mobility in soil

Ingredient	Mobility
dipropylene glycol monomethyl ether	LOW (KOC = 10)
tris(2-butoxyethyl)phosphate	LOW (KOC = 466200)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	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. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible. ▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▸ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible material). ▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG): 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

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

Continued...

Not Applicable

Transport in bulk in accordance with MARPOL Annex V and the IMSBC Code

Product name	Group
dipropylene glycol monomethyl ether	Not Available
tris(2-butoxyethyl)phosphate	Not Available
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	Not Available
methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	Not Available
Fluorosurfactant	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
dipropylene glycol monomethyl ether	Not Available
tris(2-butoxyethyl)phosphate	Not Available
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	Not Available
methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	Not Available
Fluorosurfactant	Not Available

SECTION 15 Regulatory information

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

dipropylene glycol monomethyl ether is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

tris(2-butoxyethyl)phosphate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australian Inventory of Industrial Chemicals (AIIC)

bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (Fluorosurfactant)
Canada - NDSL	No (dipropylene glycol monomethyl ether; tris(2-butoxyethyl)phosphate; bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate; methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate; Fluorosurfactant)
China - IECSC	No (Fluorosurfactant)
Europe - EINEC / ELINCS / NLP	No (Fluorosurfactant)
Japan - ENCS	No (Fluorosurfactant)
Korea - KECI	No (Fluorosurfactant)
New Zealand - NZIoC	Yes
Philippines - PICCS	No (Fluorosurfactant)

Continued...

National Inventory	Status
USA - TSCA	No (Fluorosurfactant)
Taiwan - TCSI	Yes
Mexico - INSQ	No (methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate; Fluorosurfactant)
Vietnam - NCI	Yes
Russia - ARIPS	No (methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate; Fluorosurfactant)
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 Other information

Revision Date	09/02/2021
Initial Date	02/03/2020

SDS Version Summary

Version	Issue Date	Sections Updated
0.25.1.1.1	09/02/2021	Ingredients, Physical Properties

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.
The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
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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end of SDS