E-Coat DD1008 (USA)

DataDot Technology (DataDot Technology USA)

Chemwatch Hazard Alert Code: **1**

Chemwatch: **5562-67**
Version No: **2.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **10/07/2022**
Print Date: **10/07/2022**
L.GHS.AUS.EN

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

Product Identifier

Product name	E-Coat DD1008 (USA)
Chemical Name	Not Applicable
Synonyms	Not Available
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Used as a clear base coating.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**DataDot Technology (DataDot Technology USA)**
Address	C/O Polsinelli, 1000 2nd Avenue, Suite 3500 Seattle WA 98104 United States
Telephone	509 251-8614
Fax	Not Available
Website	Not Available
Email	Not Available

Emergency telephone number

Association / Organisation	**DataDot Technology (DataDot Technology USA)**	**CHEMWATCH EMERGENCY RESPONSE**
Emergency telephone numbers	509 251-8614	+61 1800 951 288
Other emergency telephone numbers	Not Available	+61 3 9573 3188

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

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Chemwatch Hazard Ratings

	Min	Max
Flammability	0	
Toxicity	1	
Body Contact	1	
Reactivity	0	
Chronic	0	

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

Poisons Schedule	Not Applicable
Classification [1]	Not Applicable

Label elements

Hazard pictogram(s)	Not Applicable
Signal word	**Not Applicable**

Hazard statement(s)

Not Applicable

Precautionary statement(s) Prevention

Page 1 continued...

Not Applicable

Precautionary statement(s) Response

Not Applicable

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

Not Applicable

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available	30-60	acrylic polymer
25265-77-4	3.8	2,2,4-trimethyl-1,3-pentanediol monoisobutyrate
57-55-6	3.2	propylene glycol
34590-94-8	2.6	dipropylene glycol monomethyl ether
143-22-6	<0.2	butyl alcohol propoxylated
2682-20-4	<0.002	2-methyl-4-isothiazolin-3-one
2634-33-5	<0.002	1,2-benzisothiazoline-3-one
112-34-5	<0.075	diethylene glycol monobutyl ether
886-50-0	<0.03	terbutryn
26530-20-1	<0.03	2-octyl-4-isothiazolin-3-one
9005-00-9	<0.05	polyethylene glycol (10) stearyl ether
556-67-2	<0.01	octamethylcyclotetrasiloxane
68186-36-7	<0.3	tridecyl alcohol, ethoxylated, phosphated, potassium salt
24938-91-8	<0.3	tridecyl alcohol, ethoxylated
78330-21-9	0.299	alcohols C11-14-iso-, C13-rich, ethoxylated
7128-64-5	<0.05	2,5-bis(5-tert-butyl-2-benzoxazolyl)thiophene
2530-83-8	<0.1	gamma-glycidoxypropyltrimethoxysilane
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available*	

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	▸ 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.

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.

Continued...

	▸ 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 poisonous fumes. 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. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ **DO NOT** allow material to contact humans, exposed food or food utensils. ▸ 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	▸ 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

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Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	propylene glycol	Propane-1,2-diol total: (vapour & particulates)	150 ppm / 474 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	propylene glycol	Propane-1,2-diol: particulates only	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	dipropylene glycol monomethyl ether	(2-Methoxymethylethoxy) propanol	50 ppm / 308 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
2,2,4-trimethyl-1,3-pentanediol monoisobutyrate	13 mg/m3	140 mg/m3	840 mg/m3
propylene glycol	30 mg/m3	1,300 mg/m3	7,900 mg/m3
dipropylene glycol monomethyl ether	150 ppm	1700* ppm	9900** ppm
butyl alcohol propoxylated	27 mg/m3	300 mg/m3	1,800 mg/m3
diethylene glycol monobutyl ether	30 ppm	33 ppm	200 ppm
polyethylene glycol (10) stearyl ether	5.7 mg/m3	63 mg/m3	380 mg/m3
octamethylcyclotetrasiloxane	30 ppm	68 ppm	130 ppm
gamma-glycidoxypropyltrimethoxysilane	9.3 mg/m3	100 mg/m3	230 mg/m3

Ingredient	Original IDLH	Revised IDLH
acrylic polymer	Not Available	Not Available
2,2,4-trimethyl-1,3-pentanediol monoisobutyrate	Not Available	Not Available
propylene glycol	Not Available	Not Available
dipropylene glycol monomethyl ether	600 ppm	Not Available
butyl alcohol propoxylated	Not Available	Not Available
2-methyl-4-isothiazolin-3-one	Not Available	Not Available
1,2-benzisothiazoline-3-one	Not Available	Not Available
diethylene glycol monobutyl ether	Not Available	Not Available
terbutryn	Not Available	Not Available
2-octyl-4-isothiazolin-3-one	Not Available	Not Available
polyethylene glycol (10) stearyl ether	Not Available	Not Available
octamethylcyclotetrasiloxane	Not Available	Not Available
tridecyl alcohol, ethoxylated, phosphated, potassium salt	Not Available	Not Available
tridecyl alcohol, ethoxylated	Not Available	Not Available
alcohols C11-14-iso-, C13-rich, ethoxylated	Not Available	Not Available
2,5-bis(5-tert-butyl-2-benzoxazolyl)thiophene	Not Available	Not Available
gamma-glycidoxypropyltrimethoxysilane	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
butyl alcohol propoxylated	C	> 1 to ≤ 10 parts per million (ppm)
2-methyl-4-isothiazolin-3-one	D	> 0.01 to ≤ 0.1 mg/m³
1,2-benzisothiazoline-3-one	E	≤ 0.01 mg/m³
diethylene glycol monobutyl ether	E	≤ 0.1 ppm
terbutryn	E	≤ 0.01 mg/m³
2-octyl-4-isothiazolin-3-one	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
polyethylene glycol (10) stearyl ether	E	≤ 0.01 mg/m³
octamethylcyclotetrasiloxane	E	≤ 0.1 ppm
tridecyl alcohol, ethoxylated, phosphated, potassium salt	E	≤ 0.01 mg/m³
tridecyl alcohol, ethoxylated	E	≤ 0.1 ppm
alcohols C11-14-iso-, C13-rich, ethoxylated	E	≤ 0.1 ppm
gamma-glycidoxypropyltrimethoxysilane	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.*	

MATERIAL DATA

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 special circumstances. If risk of overexposure exists, wear approved respirator. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. Provide adequate ventilation in warehouses and enclosed 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.

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.

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	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:
E-Coat DD1008 (USA)

Material	CPI
PE/EVAL/PE	A

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

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required. Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 5 x ES	A-AUS / Class 1 P3	-	A-PAPR-AUS / Class 1 P3
up to 25 x ES	Air-line*	A-2 P3	A-PAPR-2 P3
up to 50 x ES	-	A-3 P3	-
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)

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Milky liquid with mild odour; mixes with water.

Physical state	Liquid	**Relative density (Water = 1)**	1.0-1.1
Odour	Slight	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Applicable
pH (as supplied)	8-9	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	~100	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Applicable	**Taste**	Not Available

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Evaporation rate	Not Applicable	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Applicable	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Applicable	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Applicable	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (Not Available%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.
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	The liquid may be miscible with fats or oils and may degrease the skin, producing a skin reaction described as non-allergic contact dermatitis. The material is unlikely to produce an irritant dermatitis as described in EC Directives . Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
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	Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

E-Coat DD1008 (USA)	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
acrylic polymer	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
2,2,4-trimethyl-1,3-pentanediol monoisobutyrate	**TOXICITY**	**IRRITATION**
	dermal (guinea pig) LD50: >19 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; >3200 mg/kg[2]	Eyes - Moderate irritant *
		Skin - Slight irritant *
		Skin (rabbit): mild ***
		Skin: no adverse effect observed (not irritating)[1]
propylene glycol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 11890 mg/kg[2]	Eye (rabbit): 100 mg - mild
	Inhalation(Rat) LC50; >44.9 mg/L4h[2]	Eye (rabbit): 500 mg/24h - mild
	Oral (Rat) LD50; 20000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin(human):104 mg/3d Intermit Mod
		Skin(human):500 mg/7days mild

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		Skin: no adverse effect observed (not irritating)[1]
dipropylene glycol monomethyl ether	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 9500 mg/kg[2]	Eye (human): 8 mg - mild
	Oral (Rat) LD50; 5135 mg/kg[2]	Eye (rabbit): 500 mg/24hr - mild
		Skin (rabbit): 238 mg - mild
		Skin (rabbit): 500 mg (open)-mild
butyl alcohol propoxylated	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 13340 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
	Inhalation(Rat) LC50; 0.147 mg/L4h[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rabbit) LD50; 1770 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
2-methyl-4-isothiazolin-3-one	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: 242 mg/kg[1]	Eye: adverse effect observed (irreversible damage)[1]
	Inhalation(Rat) LC50; 0.1 mg/l4h[1]	Skin: adverse effect observed (corrosive)[1]
	Oral (Rat) LD50; 120 mg/kg[1]	
1,2-benzisothiazoline-3-one	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: adverse effect observed (irreversible damage)[1]
	Oral (Rat) LD50; 454 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
diethylene glycol monobutyl ether	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 4120 mg/kg[2]	Eye (rabbit): 20 mg/24h moderate
	Oral (Rat) LD50; 5660 mg/kg[2]	Eye (rabbit): 5 mg - SEVERE
terbutryn	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[2]	Eye (rabbit): 76 mg - moderate
	Inhalation(Rat) LC50; >8 mg/L4h[2]	Skin (rabbit): 380 mg open - mild
	Oral (Rat) LD50; 2045 mg/kg[2]	
2-octyl-4-isothiazolin-3-one	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 311 mg/kg[2]	Eye (rabbit): 0.5% non irritant
	Oral (Rat) LD50; 248 mg/kg[2]	Eye (rabbit): 45% conc CORROSIVE
		Eye (rabbit): 5% conc moderate
		Eye(rabbit):100 mg SEVERE
		Eye: adverse effect observed (irreversible damage)[1]
		Skin (rabbit): 45% conc SEVERE
		Skin (rabbit): 500 mg/24 hours
		Skin: adverse effect observed (corrosive)[1]
		Skin: adverse effect observed (irritating)[1]
polyethylene glycol (10) stearyl ether	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >3000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50; >1.6 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; 1900 mg/kg[2]	
octamethylcyclotetrasiloxane	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 754.3 mg/kg[2]	Eye (rabbit): 500 mg/24h - mild
	Inhalation(Rat) LC50; 36 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; 1540 mg/kg[2]	Skin (rabbit): 500 mg/24h - mild
		Skin: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
tridecyl alcohol, ethoxylated, phosphated, potassium salt	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
tridecyl alcohol, ethoxylated	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; 7400 mg/kg[2]	Skin (rabbit): 2000 mg/4w mild

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alcohols C11-14-iso-, C13-rich, ethoxylated	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; 500 mg/kg[2]	Not Available
2,5-bis(5-tert-butyl-2-benzoxazolyl)thiophene	**TOXICITY**	**IRRITATION**
	Inhalation(Rat) LC50; >1.82 mg/L4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; >10000 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
gamma-glycidoxypropyltrimethoxysilane	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 4247.9 mg/kg[2]	Not Available
	Inhalation(Rat) LC50; >5.3 mg/l4h[1]	
	Oral (Rat) LD50; 7010 mg/kg[2]	
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*	

2,2,4-TRIMETHYL-1,3-PENTANEDIOL MONOISOBUTYRATE	Not a skin sensitiser (guinea pig, Magnusson-Kligman) *** Ames Test: negative *** Micronucleus, mouse: negative *** Not mutagenic *** No effects on fertility or foetal development seen in the rat *** * [SWIFT] ** [Eastman] *** [Perstop]
PROPYLENE GLYCOL	The acute oral toxicity of propylene glycol is very low, and large quantities are required to cause perceptible health damage in humans. Serious toxicity generally occurs only at plasma concentrations over 1 g/L, which requires extremely high intake over a relatively short period of time. It would be nearly impossible to reach toxic levels by consuming foods or supplements, which contain at most 1 g/kg of PG. Cases of propylene glycol poisoning are usually related to either inappropriate intravenous administration or accidental ingestion of large quantities by children.The potential for long-term oral toxicity is also low. Because of its low chronic oral toxicity, propylene glycol was classified by the U. S. Food and Drug Administration as "generally recognized as safe" (GRAS) for use as a direct food additive. Prolonged contact with propylene glycol is essentially non-irritating to the skin. Undiluted propylene glycol is minimally irritating to the eye, and can produce slight transient conjunctivitis (the eye recovers after the exposure is removed). Exposure to mists may cause eye irritation, as well as upper respiratory tract irritation. Inhalation of the propylene glycol vapours appears to present no significant hazard in ordinary applications. However, limited human experience indicates that inhalation of propylene glycol mists could be irritating to some individuals It is therefore recommended that propylene glycol not be used in applications where inhalation exposure or human eye contact with the spray mists of these materials is likely, such as fogs for theatrical productions or antifreeze solutions for emergency eye wash stations. Propylene glycol is metabolised in the human body into pyruvic acid (a normal part of the glucose-metabolism process, readily converted to energy), acetic acid (handled by ethanol-metabolism), lactic acid (a normal acid generally abundant during digestion), and propionaldehyde (a potentially hazardous substance). Propylene glycol shows no evidence of being a carcinogen or of being genotoxic. Research has suggested that individuals who cannot tolerate propylene glycol probably experience a special form of irritation, but that they only rarely develop allergic contact dermatitis. Other investigators believe that the incidence of allergic contact dermatitis to propylene glycol may be greater than 2% in patients with eczema. 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, 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.
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

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	metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body. As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces. As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating None are skin sensitisers. In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested). Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members. One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health. In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity. The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.
BUTYL ALCOHOL PROPOXYLATED	In general, the toxicity of the PPGs Butyl Ether decreased with increasing molecular weight; for example, PPG-40 Butyl Ether was less toxic than PPG-2 Butyl Ether. Mutagenicity data were not found on the PPGs Butyl Ether. However, an ether of molecular weight 800 Da (~PPG-13 Butyl Ether) was non-carcinogenic when fed to rats for 2 years. Because the PPGs Butyl Ethers undergo metabolic degradation; i.e., the butyl group are removed and oxidized, the PPG chains are split into random length fragments, the genotoxicity of the component chemicals, propylene glycol (PG) and n- Butyl Alcohol, were also considered. Both PG and n-Butyl Alcohol were non-mutagenic in mammalian and microbial assays. PG was non-carcinogenic in a 2-year feeding study using rats and in a lifetime dermal study using mice. These studies effectively eliminated the need for genotoxicity data on the PPG Butyl Ethers. There was concern about the irritancy potential of PPG-2 Butyl Ether. In animal irritation studies, the ingredient caused minor, transient erythema and desquamation; in addition, erythema, edema, ecchymosis, necrosis, and other changes were observed during an acute percutaneous study. PPG-2 Butyl Ether also caused minor to moderate conjunctival irritation and minor corneal injury. It was concluded that the PPG Butyl Ethers were safe for use in cosmetics when formulated to avoid irritation. The dermal LD50 of PPG-3 Butyl Ether was 2 g/kg in rats and rabbits, and the dermal LD50 of Buteth-3 in rats was 3.5 g/kg. The oral LD50 of PPG-3 Butyl Ether and of Buteth-3 in rats was 2 g/kg and 6.6 g/kg, respectively. Polypropyleneglycol butyl ethers (not defined) had a dermal and an oral LD50 of 2 g/kg and 0.3-2 g/kg bw, respectively,in mice.Buteth-3 (1000 mg/kg/day) was not toxic to rabbits in a 21-day dermal study; erythema, desquamation,and fissuring were observed In short-term oral toxicity studies in rats, PPG-3 Butyl Ether had a NOAEL of 1000 mg/kg bw; polypropylene glycol butyl ethers had a NOEL of 100 mg/kg bw/day for clinical observations, higher absolute and relative liver weights, and an increased incidence of liver and thyroid gland hypertrophy; and 1-(2-butoxy-1-methylethoxy)propan-2-ol had a NOAEL of 100 mg/kg/day based on very slight to slight hepatocellular hypertrophy with no corresponding increases in liver weights in low-dose males. In a 90-day oral toxicity study, administration of up to 1000 mg/kg bw/day PPG-3 Butyl Ether to rats in drinking water produced treatment-related increases in absolute and relative liver and kidney weights. The NOAELs in rats and mice exposed to=3000 ppm methoxyisopropanol via inhalation for 2 yrs were 1000 ppm (based on slight body wt decreases in males and females) and 300 ppm (based on altered hepatocellular foci in males), respectively.Dermal application of propylene glycol butyl ether was not embryotoxic or teratogenic to rabbits (=100 mg/kg bw/day applied on days 7-18 of. gestation) or rats (=1.0 ml/kg bw/day applied on days 6-16 of gestation). 1-(2-Butoxy-1-methyl-ethoxy)propan-2-ol (applied on days 6-16 or 6-15 of gestation) also was not embryotoxic or teratogenic in rats. No test-article related adverse developmental or reproductive effects were observed in rats dosed by gavage with up to 1000 mg/kg Buteth-3 or 1-(2-butoxy-1-methylethoxy)propan-2-ol or up to 500 mg/kg bw/day polypropylene glycol butyl ethers. In inhalation studies, exposure of rats to =1.0 mg/l air PPG-3 Methyl Ether did not have any teratogenic or reproductive effects. Exposure to 1000 and 3000 ppm methoxyisopropanol produced some adverse effects in a two-generation study in rats; adverse effects were not observed with 300 ppm. PPG-3 Butyl Ether was not genotoxic in vitro in the Ames test or in vivo in a mouse micronucleus assay. Propylene glycol butyl ether was not genotoxic in an Ames test or a mammalian chromosomal aberration assay in rat lymphocytes, andneither propylene glycol butyl ether or 1-(2-butoxy-1-methylethoxy)propan-2-ol were genotoxic in a mammalian cellmutation assay in CHO cell. In inhalation carcinogenicity studies, mice and rats were exposed by whole body exposure to =3000 ppm methoxyisopropan-ol for 2 yrs. An increase in S-phase DNA synthesis and in MFO activity in the liver was observed in high-dose male mice and rats. Renal epithelial tumors were not observed, and the NOEL for carcinogenicity was 3000 ppm for mice and rats. Undiluted PPG-3 Butyl Ether was not irritating to rabbit skin or eyes, and it was not an irritant or sensitizer in guinea pigs. Polypropylene glycol butyl ethers were classified as non-corrosive in an EpiDermTM study
2-METHYL-4-ISOTHIAZOLIN-3-ONE	Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies with similar materials using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies. The European Union has reclassified several formaldehyde-releasing agents (FRAs) such as methylenedimorpholine (MBM), oxazolidine (MBO) and hydroxypropylamine (HPT) as category 1B carcinogens. Previously, formaldehyde itself was classed as a carcinogen – but formaldehyde-releasing agents were not. This is no longer the case. Based on this regulation, formulations for

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	which the maximum theoretical concentration of releasable formaldehyde is more than > 1000 ppm (>0.1%), have to be labelled as carcinogenic. Water mix metalworking fluids are subject to contamination by bacteria and fungi, and the control of this is an essential part of good fluid maintenance. The use of preservatives both within the formulation and tank-side treatment plays a significant contribution in the protection of potentially harmful microbes that could cause health problems for workers. A large proportion of bactericides on the market today are classed as formaldehyde releasing biocides which means that under specific conditions they release small amounts of formaldehyde – this is their mode of action in the presence of bacteria. Although they are effective as a biocide their use may become restricted or unfavourable due to potential changes in legislation. A decision by the ECHA (European Chemicals Agency) was made to re-classify formaldehyde as a category 1b H350 carcinogen and category 2 mutagen in June 2015. It has also been proposed by the ECHA Risk Assessment Committee (RAC) that formaldehyde release biocides should be classified the same as formaldehyde because formaldehyde is released when these substances come into contact under favorable conditions (i.e. interaction with microorganisms). Formaldehyde generators (releasers) are often used as preservatives (antimicrobials, biocides, microbiocides). Formaldehyde may be generated following hydrolysis. The most widely used antimicrobial compounds function by releasing formaldehyde once inside the microbe cell. Some release detectable levels of formaldehyde into the air space, above working solutions, especially when pH has dropped. Many countries are placing regulatory pressure on suppliers and users to replace formaldehyde generators. Formaldehyde generators are a diverse group of chemicals that can be recognised by a small, easily detachable formaldehyde moiety, prepared by reacting an amino alcohol with formaldehyde ("formaldehyde-condensates"), There is concern that when formaldehyde-releasing preservatives are present in a formulation that also includes amines, such as triethanolamine (TEA), diethanolamine (DEA), or monoethanolamine (MEA), nitrosamines can be formed,; nitrosamines are carcinogenic substances that can potentially penetrate skin. One widely-discussed hypothesis states that formaldehyde-condensate biocides, such as triazines and oxazolidines, may cause an imbalance in the microbial flora of in-use metalworking fluids (MWFs).The hypothesis further asserts that this putative microbial imbalance favours the proliferation of certain nontuberculosis mycobacteria (NTM) in MWFs and that the subsequent inhalation of NTM-containing aerosols can cause hypersensitivity pneumonitis (HP), also known as extrinsic allergic alveolitis, in a small percentage of susceptible workers. Symptoms of HP include flu-like illness accompanied by chronic dyspnea, i.e., difficult or laboured respiration According to Annex VI of the Cosmetic Directive 76/768/EC, the maximum authorised concentration of free formaldehyde is 0.2% (2000 ppm). In addition, the provisions of Annex VI state that, *All finished products containing formaldehyde or substances in this Annex and which release formaldehyde must be labelled with the warning "contains formaldehyde" where the concentration of formaldehyde in the finished product exceeds 0.05%.* Formaldehyde-releasing preservatives have the ability to release formaldehyde in very small amounts over time. The use of formaldehyde-releasing preservatives ensures that the actual level of free formaldehyde in the products is always very low but at the same time sufficient to ensure absence of microbial growth. The formaldehyde reacts most rapidly with organic and inorganic anions, amino and sulfide groups and electron-rich groups to disrupt metabolic processes, eventually causing death of the organism. **NOTE:** Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA. Considered to be a minor sensitiser in Kathon CG (1) (1). Bruze etal - Contact Dermatitis 20: 219-39, 1989
1,2-BENZISOTHIAZOLINE-3-ONE	The predominant fate of the thiazole ring is oxidative ring scission catalysed by cytochrome P450 (CYP) and formation of the corresponding alpha-dicarbonyl metabolites and thioamide derivatives. The well-established toxicity associated with thioamides and thioureas has led to the speculation that thiazole toxicity is attributed to ring scission yielding the corresponding thioamide metabolite. Ring opening has also been observed in benzothiazoles. For instance, benzothiazole itself is converted to S-methylmercaptoaniline. **Acute toxicity** data show that 1,2-benzisothiazoline-3-one (BIT) is moderately toxic by the oral and dermal routes but that this chemical is a severe eye irritant. Irritation to the skin from acute data show only mild skin irritation , but repeated dermal application indicated a more significant skin irritation response. The neurotoxicity observed in the rat acute oral toxicity study (piloerection and upward curvature of the spine at 300 mg/kg and above; decreased activity, prostration, decreased abdominal muscle tone, reduced righting reflex, and decreased rate and depth of breathing at 900 mg/kg) and the acute dermal toxicity study (upward curvature of the spine was observed in increased incidence, but this was absent after day 5 post-dose at a dose of 2000 mg/kg) were felt to be at exposures in excess of those expected from the use pattern of this pesticide and that such effects would not be observed at estimated exposure doses. **Subchronic oral toxicity** studies showed systemic effects after repeated oral administration including decreased body weight, increased incidence of forestomach hyperplasia, and non-glandular stomach lesions in rats. In dogs, the effects occurred at lower doses than in rats, and included alterations in blood chemistry (decreased plasma albumin, total protein, and alanine aminotransferase) and increased absolute liver weight. **Developmental toxicity** studies were conducted in rats with maternal effects including decreased body weight gain, decreased food consumption, and clinical toxicity signs (audible breathing, haircoat staining of the anogenital region, dry brown material around the nasal area) as well as increased mortality. Developmental effects consisted of increases in skeletal abnormalities (extra sites of ossification of skull bones, unossified sternebrae) but not external or visceral abnormalities. **Reproductive toxicity:** In a two- generation reproduction study, parental toxicity was observed at 500 ppm and was characterized by lesions in the stomach. In pups, toxic effects were reported at 1000 ppm and consisted of preputial separation in males and impaired growth and survival in both sexes. The reproduction study did not show evidence of increased susceptibility of offspring.
DIETHYLENE GLYCOL MONOBUTYL ETHER	For diethylene glycol monoalkyl ethers and their acetates: This category includes diethylene glycol ethyl ether (DGEE), diethylene glycol propyl ether (DGPE) diethylene glycol butyl ether (DGBE) and diethylene glycol hexyl ether (DGHE) and their acetates. **Acute toxicity:** There are adequate oral, inhalation and/or dermal toxicity studies on the category members. Oral LD50 values in rats for all category members are all > 3000 mg/kg bw, with values generally decreasing with increasing molecular weight. Four to eight hour acute inhalation toxicity studies were conducted for all category members except DGPE in rats at the highest vapour concentrations achievable. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 2000 mg/kg bw (DGHE) to 15000 mg/kg bw (DGEEA). Signs of acute toxicity in rodents are consistent with non-specific CNS depression typical of organic solvents in general. All category members are slightly irritating to skin and slightly to moderately irritating to eyes (with the exception of DGHE, which is highly irritating to eyes). Sensitisation tests with DGEE, DGEEA, DGPE, DGBE and DGBEA in animals and/or humans were negative. **Repeat dose toxicity**: Valid oral studies conducted using DGEE, DGPE, DGBEA, DGHE and the supporting chemical DGBE ranged in duration from 30 days to 2 years. Effects predominantly included kidney and liver toxicity, absolute and/or relative changes in organ weights, and some changes in haematological parameters. All effects were seen at doses greater than 800-1000 mg/kg bw/day from oral or dermal studies; no systemic effects were observed in inhalation studies with less than continuous exposure regimens. **Mutagenicity:** DGEE, DGEEA, DGBE, DGBEA and DGHE generally tested negative for mutagenicity in *S. typhimurium* strains TA98, TA100, TA1535, TA1537 and TA1538 and DGBEA tested negative in E. coli WP2uvrA, with and without metabolic activation. *In vitro* cytogenicity and sister chromatid exchange assays with DGBE and DGHE in Chinese Hamster Ovary Cells with and without metabolic activation and *in vivo* micronucleus or cytogenicity tests with DGEE, DGBE and DGHE in rats and mice were negative, indicating that these diethylene glycol ethers are not likely to be genotoxic. **Reproductive and developmental toxicity:** Reliable reproductive toxicity studies on DGEE, DGBE and DGHE show no effect on fertility at the highest oral doses tested (4,400 mg/kg/day for DGEE in the mouse and 1,000 mg/kg/day for DGBE and DGHE in the

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	rat). The dermal NOAEL for reproductive toxicity in rats administered DGBE also was the highest dose tested (2,000 mg/kg/day). Although decreased sperm motility was noted in F1 mice treated with 4,400 mg/kg/day DGEE in drinking water for 14 weeks, sperm concentrations and morphology, histopathology of the testes and fertility were not affected. Results of the majority of adequate repeated dose toxicity studies in which reproductive organs were examined indicate that DGPE and DGBEA do not cause toxicity to reproductive organs (including the testes). Test material-related testicular toxicity was not noted in the majority of the studies with DGEE or DGEEA. Results of the developmental toxicity studies conducted with DGEE, DGBE and DGHE are almost exclusively negative. In these studies, effects on the foetus are generally not observed (even at concentrations that produced maternal toxicity). Exposure to 102 ppm (560 mg/m3) DGEE by inhalation (maximal achievable vapour concentration) or 1385 mg/kg/day DGEE by the dermal route during gestation did not cause maternal or developmental toxicity in the rat. Maternal toxicity and teratogenesis were not observed in rabbits receiving up to 1000 mg/kg/day DGBE by the dermal route during gestation; however a transient decrease in body weight was observed, which reversed by Day 21 In the mouse, the only concentration of DGEE tested (3500 mg/kg/day by gavage) caused maternal, but no foetal toxicity. Also, whereas oral administration of 2050 mg/kg/day DGBE (gavage) to the mouse and 1000 mg/kg/day DGHE (dietary) caused maternal toxicity, these doses had no effect on the developing foetus
TERBUTRYN	NOEL (90 days) for rats 600 mg/kg diet (50 mg/kg daily); (6 months) dogs 1000 mg/kg diet (10 mg/kg daily) * Toxicity Class WHO III; EPA III * ADI: 0.1 mg/kg/day NOEL: 10 mg/kg/day For terbutryn: **Acute Toxicity:** Terbutryn is slightly toxic. It affects the central nervous system in animals leading to incoordination, convulsions, or labored breathing . At extremely high dosages, the animals showed swelling and fluid in the lungs and central nervous system . Terbutryn is not a skin sensitiser . **Reproductive Effects:** A three generation reproduction study of rats showed that doses of 150 mg/kg/day of terbutryn caused decreased fertility indices in both male and female rats. . **Teratogenic Effects:** Above doses of 500 mg/kg/day, pregnant rats produced offspring with reduced weight and reduced bone formation in the front and rear paws. Pregnant rabbits exposed to doses of 75 mg/kg/day also had offspring with reduced bone formation . **Mutagenic Effects:** In tests of terbutryn, no mutagenic effects were observed . **Carcinogenic Effects:** In a two-year feeding study of rats, doses of 150 mg/kg of terbutryn caused cancerous tumor growth. However, there is no evidence of carcinogenicity in mice. Terbutryn has been classified as a possible human carcinogen by the U.S. EPA . **Organ Toxicity:** Long-term feeding at high doses of terbutryn can cause growth retardation, kidney damage, liver damage and a decreased number of white blood cells . **Fate in Humans and Animals:** When given orally to mammals, 73 to 85% of a terbutryn dose is eliminated in metabolised form in the faeces within 24 hours The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. ***[* The Pesticides Manual, Incorporating The Agrochemicals Handbook, 10th Edition, Editor Clive Tomlin, 1994, British Crop Protection Council]***
2-OCTYL-4-ISOTHIAZOLIN-3-ONE	ROHM & HAAS Data ADI: 0.03 mg/kg/day NOEL: 60 mg/kg/day
OCTAMETHYLCYCLOTETRASILOXANE	Does not cause skin sensitization Genotoxicity in vitro : Test Type: Bacterial reverse mutation assay (AMES) Result: negative Remarks: Based on test data Test Type: Mutagenicity (in vitro mammalian cytogenetic test) Result: negative Remarks: Based on test data Test Type: Chromosome aberration test in vitro Result: negative Remarks: Based on test data Test Type: In vitro sister chromatid exchange assay in mammalian cells Result: negative Remarks: Based on test data Test Type: DNA damage and repair, unscheduled DNA synthesis in mammalian cells (in vitro) Result: negative Remarks: Based on test data Genotoxicity in vivo : Test Type: Mammalian erythrocyte micronucleus test (in vivo cytogenetic assay) Species: Rat Application Route: inhalation (vapor) Result: negative Remarks: Based on test data Test Type: Rodent dominant lethal test (germ cell) (in vivo) Species: Rat Application Route: Ingestion Result: negative Remarks: Based on test data Germ cell mutagenicity - Assessment : Animal testing did not show any mutagenic effects Effects on fertility : Test Type: Two-generation reproduction toxicity study Species: Rat, male and female Application Route: inhalation (vapor) Symptoms: Effects on fertility. Remarks: Based on test data Effects on fetal development : Test Type: Prenatal development toxicity study (teratogenicity) Species: Rabbit Application Route: inhalation (vapor) Symptoms: No effects on fetal development. Remarks: Based on test data Reproductive toxicity - Assessment : Some evidence of adverse effects on sexual function and fertility, based on animal experiments. STOT-single exposure May cause damage to organs (Eyes, Central nervous system Routes of exposure: Ingestion Assessment: No significant health effects observed in animals at concentrations of 100 mg/kg bw or less. Routes of exposure: inhalation (vapor) Assessment: No significant health effects observed in animals at concentrations of 1 mg/l/6h/d or less. Routes of exposure: Skin contact Assessment: No significant health effects observed in animals at concentrations of 200 mg/kg bw or less. Results from a 2 year repeated vapor inhalation exposure study to rats of octamethylcyclotetrasiloxane (D4) indicate effects (benign uterine adenomas) in the uterus of female animals. This finding occurred at the highest exposure dose (700 ppm) only. Studies to date have not demonstrated if these effects occur through pathways that are relevant to humans. Repeated exposure in rats to D4 resulted in protoporphyrin accumulation in the liver. Without knowledge of the specific mechanism leading to the protoporphyrin accumulation the relevance of this finding to humans is unknown
TRIDECYL ALCOHOL, ETHOXYLATED, PHOSPHATED, POTASSIUM SALT	for alkyl alcohol alkoxylate phosphate (AAAPD) surfactants (alkyl or alcohol ether phosphates): **Acute toxicity:** This group of surfactants exhibits similar effects to the alcohol ether sulfates (AAASDs) (typically sodium lauryl ether sulfate - SLES - CAS RN 68891-38-3). They are likely to be skin/ eye irritants (R36/38) in their undiluted forms but not acutely toxic. The reported oral LD50 values were higher than 1600 mg/kg for the alkyl ether phosphates family described by CAS RN: 9046-01-9. No effects were found at any concentration tested dermally. Commercial products may contain excess phosphoric acid and may produce serious eye irritation (R41) or may even be classified as corrosive, acidic substances. **Subchronic toxicity:** Data for sulfate derivatives has been identified in the public domain**.** Subchronic 21-day repeat dose dietary studies showed low toxicity of compounds with carbon lengths of C12-15, C12-14 and C13-15 with sodium or ammonium alkyl ethoxylates with POE (polyoxyethylene) n=3. One study indicated that C16-18 POE n=18 had comparable low toxicity. No-observed-adverse-effect levels (NOAELs) range from 120 to 468 mg/kg/day, similar to a NOAEL from a 90-day rat gavage study with NaC12-14 POE n=2(CAS RN 68891-38-3), which was reported to be 225 mg/kg/day. In addition, another 90-day repeat dose dietary study with NaC12-15 POE n=3 (CAS RN 68424-50-0) resulted in low toxicity, with a NOAEL of greater than approximately 50 mg/kg/day (calculated based on dose of 1000 ppm in diet). Effects were usually related to hepatic hypertrophy, increased liver weight, and related increases in haematological endpoints related to liver enzyme induction. SLES was evaluated for effects on the reproduction and prenatal/postnatal development of the rat when administered orally via the drinking water through two successive generations. Based on this study an overall no-observed-adverse-effect level (NOAEL) for systemic effects was 0.1%, which was 86.6 mg/kg/day for the F0 generation, and 149.5 mg/kg/day for the F1 generation. The NOAEL of 86.6 mg/kg/day was selected as the toxicology endpoint for the chronic risk assessment for the sulfate derivatives **Genotoxicity:** Alcohol ether phosphates are unlikely to be genotoxic by analogy with their alcohol ether sulfate equivalents. **Carcinogenicity:** Chronic dietary studies conducted with rats on sulfate derivatives showed no incidence of cancer and no effects at the concentrations tested (lowest dose tested was ca 75 mg/kg/day).] **Reproductive and developmental toxicity:** Studies with sulfate derivatives showed little to no toxicity in dams or pups with the NOEL in a developmental toxicity study in rats with SLES at the limit dose of 1000 mg/kg/day and a reproductive NOAEL of 0.3% in drinking water (equivalent to 300 mg/kg/day), the highest dose tested in a two-generation reproduction study. In studies with phosphate derivatives, the reproductive/ developmental NOAEL for an OECD 422 study with CAS 681340-47-2 was

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	800 mg/kg/day, the highest dose tested, and for CAS RN 78330-24-2 the NOEL was 200 mg/kg/day. An NOAEL of 200 mg/kg/day was selected as the toxicological endpoint for he chronic risk assessment for phosphate derivatives by the US EPA. Both alcohol ether sulfates and phosphates have been evaluated in acute, subchronic, developmental and reproductive studies capable of detecting effects on endocrine mediated events. The results of these studies did not give any indication of a treatment-related effect on the oestrogen receptor or endocrine system**.** **Metabolic fate:** For compounds of comparable C16 carbon chain, the metabolites of the lower molecular weight ethoxylated (POE n=3) alcohol ether sulfate surfactants are readily absorbed and excreted primarily in the urine whereas the C16 surfactants with increased ethoxylation (POE n=9) are poorly absorbed and excreted primarily in the faeces There was also no evidence of hydrolysis of the sulfate group from C16 POE n= 3 and C16 POE n=9 or of metabolism of the ethoxylate portion of the molecule. With C11 POE n=3 and C12 POE n=3 metabolic studies in rats confirmed that the alkyl chain is extensively metabolised by beta- or omega oxidation leaving the ethoxysulfate, which is excreted directly. By analogy alcohol ether phosphate esters may initially undergo metabolism to generate the corresponding alkyl alcohol alkoxylate and POE (or POE/POP - polyoxypropylene) phosphate glycol; the dephosphoralyted metabolite should be hydrolysed to the POE (or POE/POP) polyalkoxylate glycols and linear branched saturated and unsaturated alkyl alcohol metabolites. The resultant alkyl alcohol metabolites would be oxidised in fatty acid oxidation pathways. The polyalkoxylate glycols may either be conjugated and excreted unchanged or hydrolysed/ oxidised to various degraded metabolites before bring conjugated and excreted **Sensitising potential:** Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture . On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.
ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED	* Ashland SDS The material may produce respiratory tract irritation. Symptoms of pulmonary irritation may include coughing, wheezing, laryngitis, shortness of breath, headache, nausea, and a burning sensation. Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence). The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties.
GAMMA-GLYCIDOXYPROPYLTRIMETHOXYSILANE	For alkoxysilanes: Low molecular weight alkoxysilanes (including alkyl orthosilicates) are a known concern for lung toxicity, due to inhalation of vapours or aerosols causing irreversible lung damage at low doses. Alkoxysilane groups that rapidly hydrolyse when in contact with water, result in metabolites that may only cause mild skin irritation. Although there appears to be signs of irritation under different test conditions, based on the available information, the alkoxysilanes cannot be readily classified as a skin irritant. The trimethoxysilane group of chemicals have previously been associated with occupational eye irritation in exposed workers who experienced severe inflammation of the cornea . Based on the collective information, these substances are likely to be severe irritants to the eyes. Methoxysilanes are generally reported to possess higher reactivity and toxicity compared to ethoxysilanes; some methoxysilanes appear to be carcinogenic .In the US, alkoxysilanes with alkoxy groups greater than C2 are classified as moderate concern. Based on available information on methoxysilanes, the possibility that this family causes skin sensitisation cannot be ruled out. Amine-functional methoxysilanes have previously been implicated as a cause of occupational contact dermatitis, often as a result of repeated skin exposure with workers involved in the manufacture or use of the resins containing the chemical during fibreglass production. For gamma-glycidopropyltrimethoxysilane (GPTMS) GPTMS is subject to rapid hydrolysis, and the observed toxicity is expected to be due primarily to methanol and silanetriols. GPTMS has been tested for acute toxicity by the oral, dermal, and inhalation routes of exposure. Reported acute oral LD50s in rats range from 7010 to 16900 mg/kg bw and > 5 ml/kg bw to 22.6 ml/kg bw. The dermal LD50s are 6800 mg/kg bw and 4.0 ml/kg bw. The 4-hour inhalation LC50 was greater than 2.7 mg/L in one study and greater than 5.3 mg/L in another study. GPTMS is mildly irritating to the skin and eyes and is not a known skin sensitiser in humans or in animals. Following inhalation exposures of rats to target aerosol concentrations of 0, 75, 225 and 750 mg/m3 (actual concentrations were 0, 77, 226, 707 mg/m3 (males) and 0, 73, 226, 734 mg/m3 (females)), GPTMS in 9 repeated exposures administered over two weeks, 6 animals in the high dose group died or were sacrificed from three to five days after initiation of the study. These animals had signs of inanition but no acute tissue toxicity. At both the mid and high doses, rats exhibited some clinical signs including a dose-related decrease in body weight. Under the conditions of this study, the No Observed Adverse Effect Concentration is 225 mg/m3. Repeated exposure of rats by gavage to GPTMS doses of 40, 400 and 1000 mg/kg bw/day for 5 days/week for 4 weeks resulted in no test substance-related organ weights effects or gross or microscopic pathological changes. Under the conditions of this study, the NOAEL for the test substance was found to be 1000 mg/kg bw/day. **Genotoxicity:** GPTMS did not induce chromosomal damage in mouse bone marrow cells by gavage at doses of 500, 1670 and 5000 mg/kg bw/day, or when administered by intraperitoneal (i.p.) injection at 1600 mg/kg bw/day. However, chromosomal damage was induced in mouse bone marrow cells when administered by i.p. in water at doses of 500, 1000 and 2000 mg/kg bw/day. GPTMS induced gene mutations in bacteria. GPTMS induced gene mutations in mouse lymphoma L1578Y TK cells but did not induce forward mutations in CHO cells. GPTMS induced SCE in vitro. There are no in vivo gene mutation data. **Carcinogenicity:** GPTMS was not considered tumourigenic when applied to the clipped skin of mice (25 ul dose of 25% GPTMS in acetone) three times per week for approximately 78 weeks. Note that there was only one dose level, and this dose was relatively low. **Reproductive toxicity:** In a one-generation reproduction toxicity study in rats, no reproductive effects were observed at any of the doses tested (250, 500, or 1000 mg/kg bw/day). At 1000 mg/kg bw/day, treatment with GPTMS resulted in the following signs in parental animals: discomfort after dosing (noted for females from early/mid gestation onwards), decreased body weight gain (males), increased mean relative liver and kidney weights (noted for males and females), and histopathological effects on livers and kidneys (males). Based on these data, a NOAEL for parental animals was established at 500 mg/kg bw/day. A NOAEL for reproductive effects was established at 1000 mg/kg bw/day. **Developmental toxicity:** Three developmental studies have been conducted using GPTMS. In a rabbit study, the maternal NOAEL was 200 mg/kg bw/day and the developmental NOAEL was 400 mg/kg bw/day (the highest dose tested). In a rat study, the NOAELs for both maternal and developmental toxicity were also at the highest dose tested (1000 mg/kg bw/day). In another rat study, developmental effects were observed at the maternally toxic dose of 3000 mg/kg bw/day (again, the highest dose tested).

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	Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit many common characteristics with respect to animal toxicology. One such oxirane is ethyloxirane; data presented here may be taken as representative. for 1,2-butylene oxide (ethyloxirane): Ethyloxirane increased the incidence of tumours of the respiratory system in male and female rats exposed via inhalation. Significant increases in nasal papillary adenomas and combined alveolar/bronchiolar adenomas and carcinomas were observed in male rats exposed to 1200 mg/m3 ethyloxirane via inhalation for 103 weeks. There was also a significant positive trend in the incidence of combined alveolar/bronchiolar adenomas and carcinomas. Nasal papillary adenomas were also observed in 2/50 high-dose female rats with none occurring in control or low-dose animals. In mice exposed chronically via inhalation, one male mouse developed a squamous cell papilloma in the nasal cavity (300 mg/m3) but other tumours were not observed. Tumours were not observed in mice exposed chronically via dermal exposure. When trichloroethylene containing 0.8% ethyloxirane was administered orally to mice for up to 35 weeks, followed by 0.4% from weeks 40 to 69, squamous-cell carcinomas of the forestomach occurred in 3/49 males (p=0.029, age-adjusted) and 1/48 females at week 106. Trichloroethylene administered alone did not induce these tumours and they were not observed in control animals . Two structurally related substances, oxirane (ethylene oxide) and methyloxirane (propylene oxide), which are also direct-acting alkylating agents, have been classified as carcinogenic
ACRYLIC POLYMER & 2-METHYL-4-ISOTHIAZOLIN-3-ONE & 1,2-BENZISOTHIAZOLINE-3-ONE & TRIDECYL ALCOHOL, ETHOXYLATED, PHOSPHATED, POTASSIUM SALT & 2,5-BIS(5-TERT-BUTYL-2-BENZOXAZOLYL)THIOPHENE	No significant acute toxicological data identified in literature search.
2,2,4-TRIMETHYL-1,3-PENTANEDIOL MONOISOBUTYRATE & DIPROPYLENE GLYCOL MONOMETHYL ETHER & 2-METHYL-4-ISOTHIAZOLIN-3-ONE & OCTAMETHYLCYCLOTETRASILOXANE	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
2,2,4-TRIMETHYL-1,3-PENTANEDIOL MONOISOBUTYRATE & DIPROPYLENE GLYCOL MONOMETHYL ETHER & 2-METHYL-4-ISOTHIAZOLIN-3-ONE & TERBUTRYN & OCTAMETHYLCYCLOTETRASILOXANE & TRIDECYL ALCOHOL, ETHOXYLATED	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.
PROPYLENE GLYCOL & ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
DIPROPYLENE GLYCOL MONOMETHYL ETHER & 2-METHYL-4-ISOTHIAZOLIN-3-ONE & 2-OCTYL-4-ISOTHIAZOLIN-3-ONE & ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. 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. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
BUTYL ALCOHOL PROPOXYLATED & ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED	Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture . On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing. Allergic Contact Dermatitis—Formation, Structural Requirements,and Reactivity of Skin Sensitizers. Ann-Therese Karlberg et al; Chem. Res. Toxicol.2008,21,53-69 Polyethylene glycols (PEGs) have a wide variety of PEG-derived mixtures due to their readily linkable terminal primary hydroxyl groups in combination with many possible compounds and complexes such as ethers, fatty acids, castor oils, amines, propylene glycols, among other derivatives. PEGs and their derivatives are broadly utilized in cosmetic products as surfactants, emulsifiers, cleansing agents, humectants, and skin conditioners. PEGs and PEG derivatives were generally regulated as safe for use in cosmetics, with the conditions that impurities and by-products, such as ethylene oxides and 1,4-dioxane, which are known carcinogenic materials, should be removed before they are mixed in cosmetic formulations. Most PEGs are commonly available commercially as mixtures of different oligomer sizes in broadly- or narrowly-defined molecular weight (MW) ranges. For instance, PEG-10,000 typically designates a mixture of PEG molecules (n = 195 to 265) having an average MW of 10,000. PEG is also known as polyethylene oxide (PEO) or polyoxyethylene (POE), with the three names being chemical synonyms. However, PEGs mainly refer to oligomers and polymers with molecular masses below 20,000 g/mol, while PEOs are polymers with molecular masses above 20,000 g/mol, and POEs are polymers of any molecular mass. Relatively small molecular weight PEGs are produced by the chemical reaction between ethylene oxide and water or ethylene glycol (or other ethylene glycol oligomers), as catalyzed by acidic or basic catalysts. To produce PEO or high-molecular weight PEGs, synthesis is performed by suspension polymerization. It is necessary to hold the growing polymer chain in solution during the course of the poly-condensation process. The reaction is catalyzed by magnesium-, aluminum-, or calcium-organoelement compounds. To prevent coagulation of polymer chains in the solution, chelating additives such as dimethylglyoxime are used Safety Evaluation of Polyethyene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of Toxicology http://doi.org/10.5487/TR.2015.31.2.105 For high boiling ethylene glycol ethers (typically triethylene- and tetraethylene glycol ethers): **Skin absorption:** Available skin absorption data for triethylene glycol ether (TGBE), triethylene glycol methyl ether (TGME), and triethylene glycol ethylene ether (TGEE) suggest that the rate of absorption in skin of these three glycol ethers is 22 to 34 micrograms/cm2/hr, with the methyl ether having the highest permeation constant and the butyl ether having the lowest. The rates of absorption of TGBE, TGEE and TGME are at least 100-fold less than EGME, EGEE, and EGBE, their ethylene glycol monoalkyl ether counterparts, which have absorption rates that range from 214 to 2890 micrograms/ cm2/hr . Therefore, an increase in either

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	the chain length of the alkyl substituent or the number of ethylene glycol moieties appears to lead to a decreased rate of percutaneous absorption. However, since the ratio of the change in values of the ethylene glycol to the diethylene glycol series is larger than that of the diethylene glycol to triethylene glycol series , the effect of the length of the chain and number of ethylene glycol moieties on absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetraethylene glycol methyl; ether (TetraME) and tetraethylene glycol butyl ether (TetraBE) are expected to be less permeable to skin than TGME and TGBE, the differences in permeation between these molecules may only be slight. **Metabolism:** The main metabolic pathway for metabolism of ethylene glycol monoalkyl ethers (EGME, EGEE, and EGBE) is oxidation via alcohol and aldehyde dehydrogenases (ALD/ADH) that leads to the formation of an alkoxy acids. Alkoxy acids are the only toxicologically significant metabolites of glycol ethers that have been detected *in vivo.* The principal metabolite of TGME is believed to be 2-[2-(2-methoxyethoxy)ethoxy] acetic acid . Although ethylene glycol, a known kidney toxicant, has been identified as an impurity or a minor metabolite of glycol ethers in animal studies it does not appear to contribute to the toxicity of glycol ethers. The metabolites of category members are not likely to be metabolized to any large extent to toxic molecules such as ethylene glycol or the mono alkoxy acids because metabolic breakdown of the ether linkages also has to occur **Acute toxicity:** Category members generally display low acute toxicity by the oral, inhalation and dermal routes of exposure. Signs of toxicity in animals receiving lethal oral doses of TGBE included loss of righting reflex and flaccid muscle tone, coma, and heavy breathing. Animals administered lethal oral doses of TGEE exhibited lethargy, ataxia, blood in the urogenital area and piloerection before death. **Irritation:** The data indicate that the glycol ethers may cause mild to moderate skin irritation. TGEE and TGBE are highly irritating to the eyes. Other category members show low eye irritation. **Repeat dose toxicity**: Results of these studies suggest that repeated exposure to moderate to high doses of the glycol ethers in this category is required to produce systemic toxicity In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME. Testicular effects included spermatid giant cells, focal tubular hypospermatogenesis, and increased cytoplasmic vacuolisation . Due to a high incidence of similar spontaneous changes in normal New Zealand White rabbits , the testicular effects were considered not to be related to treatment . Thus, the NOAELs for TGME, TGEE and TGBE were established at 1000 mg/kg/day. Findings from this report were considered unremarkable. A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day . In this study, significantly-increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500 mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or haemolysed blood in the stomach These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity **Mutagenicity:** Mutagenicity studies have been conducted for several category members. All in vitro and in vivo studies were negative at concentrations up to 5,000 micrograms/plate and 5,000 mg/kg, respectively, indicating that the category members are not genotoxic at the concentrations used in these studies. The uniformly negative outcomes of various mutagenicity studies performed on category members lessen the concern for carcinogenicity. **Reproductive toxicity:** Although mating studies with either the category members or surrogates have not been performed, several of the repeated dose toxicity tests with the surrogates have included examination of reproductive organs. A lower molecular weight glycol ether, ethylene glycol methyl ether (EGME), has been shown to be a testicular toxicant. In addition, results of repeated dose toxicity tests with TGME clearly show testicular toxicity at an oral dose of 4,000 mg/kg/day four times greater that the limit dose of 1,000 mg/kg/day recommended for repeat dose studies. It should be noted that TGME is 350 times less potent for testicular effects than EGME. TGBE is not associated with testicular toxicity, TetraME is not likely to be metabolised by any large extent to 2-MAA (the toxic metabolite of EGME), and a mixture containing predominantly methylated glycol ethers in the C5-C11 range does not produce testicular toxicity (even when administered intravenously at 1,000 mg/kg/day). **Developmental toxicity**: The bulk of the evidence shows that effects on the foetus are not noted in treatments with . 1,000 mg/kg/day during gestation. At 1,250 to 1,650 mg/kg/day TGME (in the rat) and 1,500 mg/kg/day (in the rabbit), the developmental effects observed included skeletal variants and decreased body weight gain.
BUTYL ALCOHOL PROPOXYLATED & TRIDECYL ALCOHOL, ETHOXYLATED & ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED	Human beings have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents, and other cleaning products . Exposure to these chemicals can occur through ingestion, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that volumes well above a reasonable intake level would have to occur to produce any toxic response. Moreover, no fatal case of poisoning with alcohol ethoxylates has ever been reported. Multiple studies investigating the acute toxicity of alcohol ethoxylates have shown that the use of these compounds is of low concern in terms of oral and dermal toxicity . Clinical animal studies indicate these chemicals may produce gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhea, and lethargy. Similarly, slight to severe irritation of the skin or eye was generated when undiluted alcohol ethoxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotoxin, carcinogen, or mutagen (HERA 2007). No information was available on levels at which these effects might occur, though toxicity is thought to be substantially lower than that of nonylphenol ethoxylates. Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture . On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However,their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose allergic contact dermatitis (ACD) to these compounds by patch testing Overall, alcohol alkoxylates (AAs) are not expected to be systemically toxic, although some short chain ethylene glycol ethers, e.g. methyl and ethyl homologues are of concern for a range of adverse health effects. They include skin and eye irritation, liver and kidney damage, bone marrow and central nervous system (CNS) depression, testicular atrophy, developmental toxicity, and immunotoxicity. For higher propyl and butyl homologues, the toxicity involves haemolysis (anaemia) with secondary effects relating to haemosiderin accumulation in the spleen, liver and kidney, and compensatory haematopoiesis in the bone marrow. Systemic toxicity was shown to decrease with increasing alkyl chain lengths and/or alkoxylation degrees (ECETOC, 2005; US EPA, 2010). The chemicals ethylene glycol hexyl ether (with a longer alkyl chain length, CAS No. 112-25-4) and diethylene glycol butyl ether (with a higher ethoxylation degree, CAS No. 112-34-5) have no evidence of systemic effects including haemolysis.

Continued...

	Commercially available AAs are mixtures of homologues of varying carbon chain lengths and it is possible that some of the chemicals with an average alkyl chain length C >=6 may also contain shorter alkyl chains C <6. It is not practical to quantify the proportion of shorter C <6 chain lengths present in such chemicals, or these shorter chain lengths may not be present at all. The available data suggest a lack of systemic toxicity for the AE chemicals with potential short alkyl chain presence (NICNASa); therefore, the toxicity of the chemicals in this assessment is unlikely to be significantly affected by the presence of shorter chain alkyl groups.
BUTYL ALCOHOL PROPOXYLATED & POLYETHYLENE GLYCOL (10) STEARYL ETHER & TRIDECYL ALCOHOL, ETHOXYLATED & ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED	Alcohol ethoxylates are according to CESIO (2000) classified as Irritant or Harmful depending on the number of EO-units: EO < 5 gives Irritant (Xi) with R38 (Irritating to skin) and R41 (Risk of serious damage to eyes) EO > 5-15 gives Harmful (Xn) with R22 (Harmful if swallowed) - R38/41 EO > 15-20 gives Harmful (Xn) with R22-41 >20 EO is not classified (CESIO 2000) Oxo-AE, C13 EO10 and C13 EO15, are Irritating (Xi) with R36/38 (Irritating to eyes and skin) . AE are not included in Annex 1 of the list of dangerous substances of the Council Directive 67/548/EEC In general, alcohol ethoxylates (AE) are readily absorbed through the skin of guinea pigs and rats and through the gastrointestinal mucosa of rats. AE are quickly eliminated from the body through the urine, faeces, and expired air (CO2).Orally dosed AE was absorbed rapidly and extensively in rats, and more than 75% of the dose was absorbed. When applied to the skin of humans, the doses were absorbed slowly and incompletely (50% absorbed in 72 hours). Half of the absorbed surfactant was excreted promptly in the urine and smaller amounts of AE appeared in the faeces and expired air (CO2)). The metabolism of C12 AE yields PEG, carboxylic acids, and CO2 as metabolites. The LD50 values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity. The ability of nonionic surfactants to cause a swelling of the stratum corneum of guinea pig skin has been studied. The swelling mechanism of the skin involves a combination of ionic binding of the hydrophilic group as well as hydrophobic interactions of the alkyl chain with the substrate. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants. A substantial amount of toxicological data and information in vivo and in vitro demonstrates that there is no evidence for alcohol ethoxylates (AEs) being genotoxic, mutagenic or carcinogenic. No adverse reproductive or developmental effects were observed. The majority of available toxicity studies revealed NOAELs in excess of 100 mg/kg bw/d but the lowest NOAEL for an individual AE was established to be 50 mg/kg bw/day. This value was subsequently considered as a conservative, representative value in the risk assessment of AE. The effects were restricted to changes in organ weights with no histopathological organ changes with the exception of liver hypertrophy (indicative of an adaptive response to metabolism rather than a toxic effect). It is noteworthy that there was practically no difference in the NOAEL in oral studies of 90-day or 2 years of duration in rats. A comparison of the aggregate consumer exposure and the systemic NOAEL (taking into account an oral absorption value of 75%) results in a Margin of Exposure of 5,800. Taking into account the conservatism in the exposure assessment and the assigned systemic NOAEL, this margin of exposure is considered more than adequate to account for the inherent uncertainty and variability of the hazard database and inter and intra-species extrapolations. AEs are not contact sensitisers. Neat AE are irritating to eyes and skin. The irritation potential of aqueous solutions of AEs depends on concentrations. Local dermal effects due to direct or indirect skin contact in certain use scenarios where the products are diluted are not of concern as AEs are not expected to be irritating to the skin at in-use concentrations. Potential irritation of the respiratory tract is not a concern given the very low levels of airborne AE generated as a consequence of spray cleaner aerosols or laundry powder detergent dust. In summary, the human health risk assessment has demonstrated that the use of AE in household laundry and cleaning detergents is safe and does not cause concern with regard to consumer use.
2-METHYL-4-ISOTHIAZOLIN-3-ONE & 1,2-BENZISOTHIAZOLINE-3-ONE & 2-OCTYL-4-ISOTHIAZOLIN-3-ONE	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.
2-METHYL-4-ISOTHIAZOLIN-3-ONE & 1,2-BENZISOTHIAZOLINE-3-ONE	In light of potential adverse effects, and to ensure a harmonised risk assessment and management, the EU regulatory framework for biocides has been established with the objective of ensuring a high level of protection of human and animal health and the environment. To this aim, it is required that risk assessment of biocidal products is carried out before they can be placed on the market. A central element in the risk assessment of the biocidal products are the utilization instructions that defines the dosage, application method and amount of applications and thus the exposure of humans and the environment to the biocidal substance. Humans may be exposed to biocidal products in different ways in both occupational and domestic settings. Many biocidal products are intended for industrial sectors or professional uses only, whereas other biocidal products are commonly available for private use by non-professional users. In addition, potential exposure of non-users of biocidal products (i.e. the general public) may occur indirectly via the environment, for example through drinking water, the food chain, as well as through atmospheric and residential exposure. Particular attention should be paid to the exposure of vulnerable sub-populations, such as the elderly, pregnant women, and children. Also pets and other domestic animals can be exposed indirectly following the application of biocidal products. Furthermore, exposure to biocides may vary in terms of route (inhalation, dermal contact, and ingestion) and pathway (food, drinking water, residential, occupational) of exposure, level, frequency and duration.
DIETHYLENE GLYCOL MONOBUTYL ETHER & TRIDECYL ALCOHOL, ETHOXYLATED & ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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*

Continued...

SECTION 12 Ecological information

Toxicity

E-Coat DD1008 (USA)

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

acrylic polymer

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

2,2,4-trimethyl-1,3-pentanediol monoisobutyrate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	15mg/l	Not Available
EC50	48h	Crustacea	>19mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	3.28mg/l	1
LC50	96h	Fish	16mg/l	Not Available

propylene glycol

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	336h	Algae or other aquatic plants	<5300mg/l	1
EC50	72h	Algae or other aquatic plants	19300mg/l	2
EC50	48h	Crustacea	>114.4mg/L	4
LC50	96h	Fish	>10000mg/l	2
EC50	96h	Algae or other aquatic plants	19000mg/l	2

dipropylene glycol monomethyl ether

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

butyl alcohol propoxylated

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>500mg/l	1
EC50	48h	Crustacea	>500mg/l	1
NOEC(ECx)	72h	Algae or other aquatic plants	62.5mg/l	2
LC50	96h	Fish	1350mg/l	1
EC50	96h	Algae or other aquatic plants	744.74mg/l	2
NOEC(ECx)	96h	Algae or other aquatic plants	<15.9mg/l	2
EC50	72h	Algae or other aquatic plants	445mg/l	2
EC50	48h	Crustacea	>100mg/l	2
LC50	96h	Fish	564mg/l	2
EC50	96h	Algae or other aquatic plants	315mg/l	2
EC50(ECx)	48h	Crustacea	89-101mg/L	4
EC50	48h	Crustacea	89-101mg/L	4
LC50	96h	Fish	48-52mg/L	4

2-methyl-4-isothiazolin-3-one

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	0.189-0.257mg/L	4
NOEC(ECx)	96h	Algae or other aquatic plants	0.01mg/l	2
LC50	96h	Fish	0.081-0.122mg/L	4
EC50	96h	Algae or other aquatic plants	0.063mg/l	2

1,2-benzisothiazoline-3-one

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	0.097mg/L	4
EC50(ECx)	48h	Crustacea	0.097mg/L	4
LC50	96h	Fish	0.067-0.29mg/L	4

diethylene glycol monobutyl ether

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	1101mg/l	2
EC50	48h	Crustacea	>100mg/l	1
NOEC(ECx)	96h	Algae or other aquatic plants	>=100mg/l	1

Continued...

	LC50	96h	Fish	1300mg/l	2
	EC50	96h	Algae or other aquatic plants	>100mg/l	1

terbutryn

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	72h	Algae or other aquatic plants	0.002mg/L	4
EC50	72h	Algae or other aquatic plants	0.002mg/L	4
EC50	48h	Crustacea	2.408-3.646mg/L	4
LC50	96h	Fish	0.56-1.2mg/l	4
EC50	96h	Algae or other aquatic plants	0.003mg/L	5

2-octyl-4-isothiazolin-3-one

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	0.057-0.178mg/L	4
NOEC(ECx)	840h	Fish	0.009mg/L	4
LC50	96h	Fish	0.041-0.104mg/l	4
EC50	96h	Algae or other aquatic plants	0.15mg/l	2

polyethylene glycol (10) stearyl ether

Endpoint	Test Duration (hr)	Species	Value	Source
EC20(ECx)	72h	Algae or other aquatic plants	0.06mg/l	2
EC50	72h	Algae or other aquatic plants	>100mg/l	2
LC50	96h	Fish	>5.6mg/l	2

octamethylcyclotetrasiloxane

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	96h	Fish	0.204-3.483mg/l	4
LC50	96h	Fish	0.204>3.483mg/l	4

tridecyl alcohol, ethoxylated, phosphated, potassium salt

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

tridecyl alcohol, ethoxylated

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

alcohols C11-14-iso-, C13-rich, ethoxylated

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	1-10mg/l	Not Available

2,5-bis(5-tert-butyl-2-benzoxazolyl)thiophene

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	528h	Crustacea	>=10mg/l	2
EC50	72h	Algae or other aquatic plants	>100mg/l	2
LC50	96h	Fish	>100mg/l	2

gamma-glycidoxypropyltrimethoxysilane

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>420mg/l	2
EC50	48h	Crustacea	473mg/l	2
NOEC(ECx)	96h	Fish	1.5mg/l	2
LC50	96h	Fish	4.9mg/l	2
EC50	96h	Algae or other aquatic plants	250mg/l	2

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

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
2,2,4-trimethyl-1,3-pentanediol monoisobutyrate	LOW	LOW
propylene glycol	LOW	LOW
dipropylene glycol monomethyl ether	HIGH	HIGH
butyl alcohol propoxylated	LOW	LOW
2-methyl-4-isothiazolin-3-one	HIGH	HIGH
diethylene glycol monobutyl ether	LOW	LOW

Continued...

Ingredient	Persistence: Water/Soil	Persistence: Air
terbutryn	HIGH	HIGH
2-octyl-4-isothiazolin-3-one	HIGH	HIGH
polyethylene glycol (10) stearyl ether	HIGH	HIGH
octamethylcyclotetrasiloxane	HIGH	HIGH
2,5-bis(5-tert-butyl-2-benzoxazolyl)thiophene	HIGH	HIGH
gamma-glycidoxypropyltrimethoxysilane	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
2,2,4-trimethyl-1,3-pentanediol monoisobutyrate	LOW (LogKOW = 2.9966)
propylene glycol	LOW (BCF = 1)
dipropylene glycol monomethyl ether	LOW (BCF = 100)
butyl alcohol propoxylated	LOW (LogKOW = 1.2706)
2-methyl-4-isothiazolin-3-one	LOW (LogKOW = -0.8767)
diethylene glycol monobutyl ether	LOW (BCF = 0.46)
terbutryn	LOW (LogKOW = 2.8257)
2-octyl-4-isothiazolin-3-one	LOW (LogKOW = 2.561)
polyethylene glycol (10) stearyl ether	LOW (LogKOW = 2.2284)
octamethylcyclotetrasiloxane	HIGH (BCF = 12400)
2,5-bis(5-tert-butyl-2-benzoxazolyl)thiophene	LOW (LogKOW = 8.6112)
gamma-glycidoxypropyltrimethoxysilane	LOW (LogKOW = -0.9152)

Mobility in soil

Ingredient	Mobility
2,2,4-trimethyl-1,3-pentanediol monoisobutyrate	LOW (KOC = 22.28)
propylene glycol	HIGH (KOC = 1)
dipropylene glycol monomethyl ether	LOW (KOC = 10)
butyl alcohol propoxylated	LOW (KOC = 10)
2-methyl-4-isothiazolin-3-one	LOW (KOC = 27.88)
diethylene glycol monobutyl ether	LOW (KOC = 10)
terbutryn	LOW (KOC = 3590)
2-octyl-4-isothiazolin-3-one	LOW (KOC = 2120)
polyethylene glycol (10) stearyl ether	LOW (KOC = 10000000000)
octamethylcyclotetrasiloxane	LOW (KOC = 17960)
2,5-bis(5-tert-butyl-2-benzoxazolyl)thiophene	LOW (KOC = 236300000)
gamma-glycidoxypropyltrimethoxysilane	LOW (KOC = 90.22)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging 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 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.

Continued...

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

Not Applicable

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

Product name	Group
acrylic polymer	Not Available
2,2,4-trimethyl-1,3-pentanediol monoisobutyrate	Not Available
propylene glycol	Not Available
dipropylene glycol monomethyl ether	Not Available
butyl alcohol propoxylated	Not Available
2-methyl-4-isothiazolin-3-one	Not Available
1,2-benzisothiazoline-3-one	Not Available
diethylene glycol monobutyl ether	Not Available
terbutryn	Not Available
2-octyl-4-isothiazolin-3-one	Not Available
polyethylene glycol (10) stearyl ether	Not Available
octamethylcyclotetrasiloxane	Not Available
tridecyl alcohol, ethoxylated, phosphated, potassium salt	Not Available
tridecyl alcohol, ethoxylated	Not Available
alcohols C11-14-iso-, C13-rich, ethoxylated	Not Available
2,5-bis(5-tert-butyl-2-benzoxazolyl)thiophene	Not Available
gamma-glycidoxypropyltrimethoxysilane	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
acrylic polymer	Not Available
2,2,4-trimethyl-1,3-pentanediol monoisobutyrate	Not Available
propylene glycol	Not Available
dipropylene glycol monomethyl ether	Not Available
butyl alcohol propoxylated	Not Available
2-methyl-4-isothiazolin-3-one	Not Available
1,2-benzisothiazoline-3-one	Not Available
diethylene glycol monobutyl ether	Not Available
terbutryn	Not Available
2-octyl-4-isothiazolin-3-one	Not Available
polyethylene glycol (10) stearyl ether	Not Available
octamethylcyclotetrasiloxane	Not Available
tridecyl alcohol, ethoxylated, phosphated, potassium salt	Not Available
tridecyl alcohol, ethoxylated	Not Available
alcohols C11-14-iso-, C13-rich, ethoxylated	Not Available
2,5-bis(5-tert-butyl-2-benzoxazolyl)thiophene	Not Available

Continued...

Product name	Ship Type
gamma-glycidoxypropyltrimethoxysilane	Not Available

SECTION 15 Regulatory information

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

acrylic polymer is found on the following regulatory lists

Not Applicable

2,2,4-trimethyl-1,3-pentanediol monoisobutyrate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

propylene glycol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

dipropylene glycol monomethyl ether is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

butyl alcohol propoxylated is found on the following regulatory lists

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

2-methyl-4-isothiazolin-3-one is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

1,2-benzisothiazoline-3-one is found on the following regulatory lists

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

diethylene glycol monobutyl ether is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

terbutryn is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australian Inventory of Industrial Chemicals (AIIC)

2-octyl-4-isothiazolin-3-one is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

polyethylene glycol (10) stearyl ether is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
Australian Inventory of Industrial Chemicals (AIIC)

octamethylcyclotetrasiloxane is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Chemical Footprint Project - Chemicals of High Concern List
Australian Inventory of Industrial Chemicals (AIIC)

tridecyl alcohol, ethoxylated, phosphated, potassium salt is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

tridecyl alcohol, ethoxylated is found on the following regulatory lists

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

alcohols C11-14-iso-, C13-rich, ethoxylated is found on the following regulatory lists

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

2,5-bis(5-tert-butyl-2-benzoxazolyl)thiophene is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

gamma-glycidoxypropyltrimethoxysilane 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 (terbutryn)
Canada - NDSL	No (2,2,4-trimethyl-1,3-pentanediol monoisobutyrate; propylene glycol; dipropylene glycol monomethyl ether; butyl alcohol propoxylated; 2-methyl-4-isothiazolin-3-one; 1,2-benzisothiazoline-3-one; diethylene glycol monobutyl ether; terbutryn; 2-octyl-4-isothiazolin-3-one; polyethylene glycol (10) stearyl ether; octamethylcyclotetrasiloxane; tridecyl alcohol, ethoxylated, phosphated, potassium salt; tridecyl alcohol, ethoxylated; alcohols C11-14-iso-, C13-rich, ethoxylated; 2,5-bis(5-tert-butyl-2-benzoxazolyl)thiophene; gamma-glycidoxypropyltrimethoxysilane)

Continued...

National Inventory	Status
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (tridecyl alcohol, ethoxylated, phosphated, potassium salt; tridecyl alcohol, ethoxylated; alcohols C11-14-iso-, C13-rich, ethoxylated)
Japan - ENCS	No (terbutryn; tridecyl alcohol, ethoxylated, phosphated, potassium salt)
Korea - KECI	Yes
New Zealand - NZIoC	No (terbutryn)
Philippines - PICCS	No (terbutryn)
USA - TSCA	No (terbutryn)
Taiwan - TCSI	Yes
Mexico - INSQ	No (tridecyl alcohol, ethoxylated, phosphated, potassium salt; gamma-glycidoxypropyltrimethoxysilane)
Vietnam - NCI	Yes
Russia - FBEPH	No (terbutryn; tridecyl alcohol, ethoxylated, phosphated, potassium salt)
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	10/07/2022
Initial Date	10/07/2022

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
ES: Exposure Standard
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
AIIC: Australian Inventory of Industrial Chemicals
DSL: Domestic Substances List
NDSL: Non-Domestic Substances List
IECSC: Inventory of Existing Chemical Substance in China
EINECS: European INventory of Existing Commercial chemical Substances
ELINCS: European List of Notified Chemical Substances
NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory
KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals
PICCS: Philippine Inventory of Chemicals and Chemical Substances
TSCA: Toxic Substances Control Act
TCSI: Taiwan Chemical Substance Inventory
INSQ: Inventario Nacional de Sustancias Químicas
NCI: National Chemical Inventory
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances