SAFETY DATA SHEET (SDS)

DATA DOT

SECTION 1: IDENTIFICATION

Product Name:	Data Dot
Product Use:	Used as a clear base coating
Product Code(s):	DD-KIT001

Supplier:
Sym-Tech Inc.
P.O. Box 430, Stn A
Scarborough ON
M1K 5C3
1-800-363-5796

Emergency Tel.	1-613-996-6666 CANUTEC for 24HR emergency information

SECTION 2: HAZARD(s) IDENTIFICATION

Classification of Substance of mixture:

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

CHEMWATCH HAZARD RATING:

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

Poison Schedule:	Not applicable
Classification (1):	Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A

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

Label Elements:
Hazard Pictogram(s):

Signal Word: Warning

Hazard Statements:
H315: Causes skin irritation
H319: Causes serious eye irritation

Precautionary Statements Prevention:
P280: Wear protective gloves/protective clothing/eye protection/face protection.

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

Precautionary Statements Storage:
Not applicable

Precautionary Statements Disposal:
Not applicable

SECTION 3: COMPOSITION/INFORMATION ON HAZARDOUS INGREDIENTS

Subsatnces:
See section below for composition of Mixtures.

CAS #	% (weight)	Name
Not available	30-60	Acrylic polymer
34590-94-8	<10	Dipropylene glycol monomethyl ether
25265-77-4	<10	2.2.4-trimethyl-1.3-pentanediol monoisobutyrate
57-55-6	<10	Propylene glycol
143-22-6	<0.2	Butyl alcohol propoxylated
2682-20-4	<0.002	2-methyl-4-isothiazolin-3-one
2634-33-5	<0.02	1.2-benzisothiazoline-3-one
112-34-5	<0.075	Diethylene glycol monobutyl either
886-50-0	<003	terbutryn
26530-20-1	<0.03	Terbutryn
9005-00-9	<0.05	Polyethylene glycol (10) stearyl ether
556-67-2	<0.01	octamethylcycloterasiloxane
78330-21-9	<2.4	Alcohols C11-14-iso-. C13-rich. ethoxylated
68186-36-7	<0.03	Tridecyl alcohol, Ethoxylated, Phosphate, Potassium salt
24938-91-8	<0.03	Tridecyl alcohol ethoxylated
7128-64-5	<0.05	2.5-bis(5-tert-butyl-2-benzoxazolyl)thiophene
2530-83-8	<0.3	Gamma-glycidoxypropytrimethoxysilane

SECTION 4: FIRST-AID MEASURES

Inhalation:	If fumes, aerosols or combustion products are inhaled removed from contaminated area. Other measures are usually necessary.
Skin:	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.
Eyes:	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 reoccurs seek medical attention. Removal of contact lenses after an eye injury should be undertaken by a skilled professional.
Ingestion:	Immediately give a glass of water. First aid is not required. If in doubt, contact a POISON CONTROL CENTER or doctor/physician.
Indication of immediate medical attention and special treatment needed:	Treat symptomatically.

SECTION 5: FIRE-FIGHTING MEASURES

Fire and Explosive Properties:	Non-combustible. Not considered a significant fire risk, however containers may burn. May emit poisonous fumes and may emit corrosive fumes.
Extinguishing Media:	There is no restriction on the type of extinguisher which may be used. Use extinguishing media suitable for surrounding area.
Fire Fighting:	Alert Fire Department and tell them location and nature of hazard. Wear breathing apparatus plus protective gloves in the event of a fire. Prevent, by any means available, spillage 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.
Special Hazards arising from the substrate or mixture: **Fire Incompatibility**	None known.
HAZCHEM:	Not applicable.

SECTION 6: ACCIDENTAL RELEASE MEASURES

Minor Spill:	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 Department and tell them location and nature of hazard. Wear breathing apparatus plus protective gloves. Prevent, by any means available, spillage from entering drain 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. Neutralize/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 cleanup operations, decontaminate and launder all protective clothing and equipment before storing and reusing. 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

Storage:	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.
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. Atmospheres 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.

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 oxidizing agents.

SECTION 8: EXPOSURE CONTROLS/PERSONAL PROTECTION

Control Parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

Ingredient Data:

Source	Ingredient	Material Name	TWA	STEL	Peak
Australia Exposure Standards	Dipropylene glycol monomethyl ether	(2-Methoxymethlethoxy) propanol	50 ppm/308 mg/m3	Not available	Not available
Australia Exposure Standards	Propylene glycol	Propane-1,2-diol total: (vapour & particulates)	150 ppm/474 mg/m3	Not available	Not available
Australia Exposure Standards	Propylene glycol	Propane-1,2-diol: particulates only	10 mg/m3	Not available	Not available

Emergency Limits:

Ingredient	Material Name	TEEL-1	Teel-2	TEEL-3
Dipropylene glycol monomethyl ether	Dipropylene glycol methyl ether	150 ppm	1700*ppm	9900**ppm
2,2,4-trimethyl-1,3-pentanediol monoisobutyrate	Trimethyl-1,3-pentanediol monoisobutyrate, 2,2,4-; 9 Texanol)	13 mg/m3	140 mg/m3	840 mg/m3
Propylene glycol	Polypropylene glycols	30 mg/m3	330 mg/m3	2,000 mg/m3
Propylene glycol	Propylene glycol; (1,2-Propanediol)	30 mg/m3	1,300 mg/m3	7,900 mg/m3
Butyl alcohol propoxylated	Butoxypolypropoxylene glycol	27 mg/m3	300 mg/m3	1,800 mg/m3
Diethylene glycol monobutyl ether	Butoxyethoxy ethanol, 2-2-; (Diethylene glycol monobutyl ether	30 ppm	33 ppm	200 ppm
Polyethylene glycol (10) stearyl ether	Poly(oxyethylene)(2) stearyl ether	5.7 mg/m3	63 mg/m3	380 mg/m3
Octamethylcyclotetrasiloxane	Octamethylcyclotetrasiloxane	30 ppm	68 ppm	130 ppm
Gamma-glycidoxypropyltrimethoxysilane	Glycidoxypropyltrimethoxysilane; (3-(2,3-Epoxypropoxy) propyltrimethoxysilane	9.3 mg/m3	100 mg/m3	230 mg/m3

Ingredient	Original IDHL	Revised IDHL
Acrylic polymer	Not available	Not available
Dipropylene glycol monomethyl ether	600 ppm	Not available
2,2,4-trimethyl-1,3-pentanediol monoisobutyrate	Not available	Not available
Propylene glycol	Not available	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
Alcohols C11-14-iso-, C13-rich, ethoxylated	Not available	Not available
Tridecyl alcohol, ethoxylated, phosphate, potassium salt	Not available	Not available
Tridecyl alcohol, 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	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
Polyethylene glycol (10)stearyl ether	E	≤ 0.01 mg/m3
Octamethylcyclotetrasiloxane	E	≤ 0.1 ppm
Alcogols C11-14-iso-, C13-rich, ethoxylated	E	≤ 0.1 ppm
Tridecyl alcohol, ethoxylated, phosphate, potassium salt	E	≤ 0.01 mg/m3
Tridecyl alcohol, 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 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 place or 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 control 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. Employees 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 (release 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 dust, 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 (release at high initial velocity into zone of very high rapid air motion)	2.5-10 m/s (5500-200 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 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 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 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 cannot be calculated in advance and has to be checked prior to the application. The exact break through time for substances, has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: Frequency and duration of product, 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 2161.10.0 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 2161.01.1 or national equivalent) is recommended. Some glove polymer types are less effected 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 rates 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 application, 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 permeated 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 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.

Thermal Hazards: Not applicable

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 DD 1008

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 choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory Protection
Type A Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:200 & 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	Power Air Respirator
Up to 5 x ES	A-AUS/Class 1 P3	-	A-PAPR-AUS/Class1 P3
Up to 25 x ES	Air-line	A-2 P3	A-PAPR-2 P3
Up to 50 x ES	-	A-3 P3	-
50 + 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 compound(below 65 deg°C)

SECTION 9: PHYSICAL AND CHEMICAL PROPERTIES

Appearance:	Milky liquid with mild odour; miscible with water
Physical State:	Liquid
Odour:	Not available
Odour Threshold:	Not available
Boiling Point(°C):	~100
Vapour Density (Air = 1):	Not available
% Volatile:	Not applicable
Vapour Pressure (kPa):	Not applicable
Solubility in Water (g/L):	Miscible
Melting Point/Freezing Point (°C):	Not available
pH (as supplied):	8-9
Flashpoint (°C):	Not applicable
Evaporation Rate:	Not applicable
Flammability:	Not applicable
Upper Explosive Limit (%):	Not applicable
Lower Explosive Limit (%):	Not applicable
Relative Density (Water = 1):	1.0-1.1
Partition Coefficient n-octanol/water:	Not available
Auto-Ignition Temperature:	Not applicable
Decomposition Temperature:	Not available
Viscosity (cSt):	Not available
Molecular Weight (g/mol):	Not applicable
Taste:	Not available
Explosive Properties:	Not available
Surface Tension (dyn/cm or mN/m):	Not available
Oxidising properties:	Not available
Volatile Component (%vol):	Not available
Gas Group:	Not available
pH as a Solution (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
Materials to Avoid:	See Section 7
Conditions to Avoid:	See Section 7
Hazardous Decomposition Products:	See Section 5

SECTION 11: TOXICOLOGICAL INFORMATION

Information on Toxicological Effects

Inhalation:	Limited evidence or practical experience suggest 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 neutralizing 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 materials has not been classified by EC Directives or other classification systems as harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g. liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.
Skin Contact:	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intracellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. 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 Contact:	Evidence exists, or practical experience suggests, that the material may cause irritation in a substantial number of individuals and/or is expected to produce significant ocular lesions which are present twenty-four hours or more after instillation into the eyes of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic:	Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health defects including organs or biochemical systems.

TOXICITY/IRRITATION:

Acrylic Polymer:	**TOXICITY** Not Available	**IRRITATION** Not available
Dipropylene glycol monomethyl ether	**TOXICITY** Oral (rat) LD50: 5135 mg/kg(2)	**IRRITATION** Eye (human): 8 mg – mild Eye (rabbit): 500 mg/24hr – mild Skin (rabbit): 238 mg – mild Skin (rabbit): 500 mg (open) - mild
2,2,4-trimethyl-1,3-petanediol monoisobutyrate	**TOXICITY** >16000 MG/KG(2) Dermal (rabbit) LD50:>16000 mg/kg(2) Inhalation (rat) LC50:>5.325 mg/l/6h(2) Inhalation (rat) LC50: 1600 mg/l***(2) Oral (rat) LD50: 3200 mg/kg(2)	**IRRITATION** Eye: no adverse effect observed (not irritating)(1) Eyes – Moderate irritant* Skin – Slight irritant* Skin (rabbit): mild*** Skin: no adverse effect observed (not irritating)(1)
Propylene Glycol	**TOXICITY** Dermal (rabbit) LD50:20800 mg/kg(2) Inhalation (rat) LC50:>44.9 mg/l/4h(2) Oral (dog) LD50:=20000 mg/kg(2) Oral (mouse) LD50:=22000 mg/kg(2) Oral (mouse) LD50:=23900 mg/kg(2) Oral (rabbit) LD50:=18000-19000 mg/kg(2) Oral (rabbit) LD50:=18500 mg/kg(2) Oral (rat) LD50:20000 mg/kg(2)	**IRRITATION** Eye (rabbit):100 mg - mild Eye (rabbit): 500 mg/24h – mild Eye: no adverse effect observed (not irritating)(1) Skin (human):104 mg/3d Intermit Mod Skin (human):500 mg/7days mild Skin: no adverse effects observed (not irritating)(1)
Butyl alcohol propoxylated	**TOXICITY** Dermal (rabbit) LD50:>20000 mg/kg(2) Dermal (rabbit) LD50:14100 mg/kg(2)	**IRRITATION** Eye: adverse effect observed (irritating)(1) Eye: no adverse effects observed (not irritating)(1)

	Dermal (rabbit) LD50:20000 mg/kg(2) Dermal (rabbit) LD50:3540 mg/kg(2) Inhalation (rat) LC50:0.147 mg/l/4h**(2) Oral (rat) LD50:=>300-2000 mg/kg(1) Oral (rat) LD50:=4000 mg/kg(2) Oral (rat) LD50:=5300 mg/kg(2) Oral (rat) LD50:=9100 mg/kg(2)	Skin: no adverse effects observed (not irritating)(1)
2-methyl-4-isothiazolin-3-one	**TOXICITY** Not available	**IRRITATION** Eye: adverse effect observed (irreversible damage)(1) Skin: adverse effect observed (corrosive)(1)
1,2-benzisothiazoline-3-one	**TOXICITY** Oral (rat) LD50:1020 mg/kg(2) Oral (rat) LD50:670 mg/kg(2) Oral (rat) LD50:784 mg/kg(2)	**IRRITATION** Eye: adverse effect observed (irreversible damage)(1) Skin: no adverse effects observed (not irritating)(1)
Diethylene glycol monobutyl ether	**TOXICITY** Dermal (rabbit) LD50:4120 mg/kg(2) Oral (guinea pig) LD50:=1720-2310 mg/kg(2) Oral (mouse) LD50:=5526 mg/kg(2) Oral (rabbit) LD50:=2200 mg/kg(2) Oral (rat) LD50:=4500 mg/kg(2) Oral (rat) LD50:=5080 mg/kg(2) Oral (rat) LD50:=5660 mg/kg(2)	**IRRITATION** Eye (rabbit): 20 mg/24h – moderate Eye (rabbit): 5mg – SEVERE
Terbutryn	**TOXICITY** Dermal (rat) LD50:>2000 mg/kg(2) Inhalation (rat) LC50:>8 mg/l/4he(2) Oral (rat) LD50:=2045 mg/kg(2)	**IRRITATION** Eye (rabbit): 76 mg – moderate Skin (rabbit): 380 mg open - mild
2-octyl-4-isothiazolin-3-one	**TOXICITY** Dermal (rabbit) LD50:690 mg/kg(2) Oral (rat) LD50:=550 mg/kg(2)	**IRRITATION** Eye (rabbit): 0.5% non irritant 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** Oral (rat) LD50:>2000 mg/kg(2) Oral (rat) LD50:1900 mg/kg(2) Oral (rat) LD50:2900 mg/kg(2)	**IRRITATION** Eye: no adverse effects observed (not irritating)(1) Skin: no adverse effects observed (not irritating)(1)
Octamethylcyclotetrasiloxane	**TOXICITY** 6000-7000 mg/kg(2) Dermal (rat) LD50:1770 mg/kg(2) Inhalation (rat) LC50:36 mg/l/4hd(2) Oral (rat) LD50:>2000 mg/kg(2) Oral (rat) LD50:1540 mg/kg(2)	**IRRITATION** Eye (rabbit): 500 mg/24h – mild Eye: no adverse effects observed (not irritating)(1) Skin (rabbit): 500 mg/24h - mild Skin: adverse effect observed (irritating)(1) Skin: no adverse effects observed (not irritating)(1)
Alcohols C11-14-iso-, C13-rich, ethoxylated	**TOXICITY** Oral (rat) LD50:500 mg/kg(2	**IRRITATION** Not available
Tridecyl alohol, ethoxylated, phosphate, potassium salt	**TOXICITY** Not available	**IRRITATION** Not available
Tridecyl alcohol, ethoxylated	**TOXICITY** Oral (rat) LD50:7400 mg/kg(2)	**IRRITATION** Skin (rabbit): 2000 mg/4w - mild
2,5-bis(5-tert-butyl-2-benzoxazoly)thiophene	**TOXICITY** Oral (rat) LD50:>10000 mg/kg(2)	**IRRITATION** Eye: no adverse effects observed (not irritating)(1) Skin: no adverse effects observed (not irritating)(1)
Gamma-glycidoxypropythrimethoxysilane	**TOXICITY** Dermal (rabbit) LD50:3970 mg/kg(2) Inhalation (rat) LC50:>5.3 mg/l/4h(2) Oral (rat) LD50:7010 mg/kg(2)	**IRRITATION** Not available

Legend: *1. Value obtained from Europe ECHA Registered Substances – Acute toxicity 2. *Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS – Register of Toxic Effects of Chemical Substances.*

Dipropylene Glycol Monomethyl Ether

For propylene glycol ethers (PGEs): Typical proprylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl 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 proplylene 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 favoured 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 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 date show that this commercial grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body.

As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is 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 value 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, PnB, the 4-hour LC50 was>651 ppm (>3,412 mg/m3), representing the highest practically attainable vapour level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to skin while the remaining category members are slightly to non-irritating. None are skin sensitizers.

In repeated close 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 LOAEL (increase 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 deceased 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 would not be expected to show teratogenic effects. At high doses where material toxicity occurs (e.g. significant body weight loss) an increases 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 genotoxic. In vitro, negative results have been in a number of assays for PnB, DPnB, DPMA and TPM. Positive results wre 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 inn rats and mice.

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

Not a skin sensitizer (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 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 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 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 PGE’s (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 asthmas, 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 cleaners.

Patients with vulvodynia and interstitial cystitis may be especially sensitive to propylene glycol. Woman suffering with yeast infections may also notice that some of the over the counter creams can cause intense burning. Post-menopausal woman who require the use of an estrogen 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 vapour 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 "hypotention, 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 included 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.

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. Mutenicity 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 Ether. 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 2g/kg and 6.6 g/kg, respectively. Polypropyleneglycol butyl ethers (not defined) had a dermal and 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 has a NOAEL of 1000 mg/kg bw; polypropylene glycol butyl ethers had a NOEL of 100 mg/kg bw/day for clinical observations, highrer absolute and relative Liver weights, and an increase 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 hepatocellular hypertrophy with no corresponding increase in liver weights in low-dose males. In a 90-day oral toxicity study, administration of up to 100 mg/kg bw/day PPG-3 Butyl Ether to rats and mice exposed to=3000 ppm methoxylsopropanol 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-(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 terogenic 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 Methyl 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, and neither propylene.

2-methyl-4-isothiazolin-3-one

Exposure to the material may result in a possible risk or irreversible effects. The material may produce mutagenic effects in men. This concern is raised, generally, on the basis of appropriate studies with similar material using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.

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 biocide 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 I 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 woman, 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.

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 supplies and users to replace formaldehyde generators. Formaldehyde generators are a diverse group of chemicals that can be recognized

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 triethanolgenic (TEA), diethanoiamine (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 mycrobacteria (NTM) in MWFs and that the subsequent inhalation of NTM-containing aerosols can cause hypersensitivity pneumonitis (HP), also known as extrinsic 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 76/68/EC, the maximum authorized 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 essay, or belongs to a family of chemicals producing damage or change to cellular DNA.
Considered to be a minor sensitizer in Kathon CG (1)(1). Bruze etal – Contact Dermatitis 20: 219-39, 1989.

1,2-benzisothiazoline-3-one

Acute Toxicity data shows that 1,2-benzisothiazoline-3-one (BIT) is moderately toxic by the oral and dermal routes but that the 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 altercations n 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 there (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 concentration 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 or organic solvents in general. All category members are 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, DGHE, DGBEA and the supporting chemical DGBE ranged in duration from 30 days to 2 years. Effects predominantly include 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 DGHE and DGBE in Chinese Hamster Ovary Cells with and without metabolic activation and in vivo micronucleous 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 (4400 mg/kg/day for DGEE in the mouse and 1000 mg/kg/day for DGBE and DGHE in the rat). The dermal NOAEL for reproductive toxicity in rats administered DGBE also was the highest dose tested (2000 mg/kg/day). Although decreases sperm motility was noted in F1 mice treated with 4400 mg/kg/day DGEE in drinking water for 14 weeks, sperm concentrations are morphology, histopathology of the tests 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 produce 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/kd/day NOEL: 10 mg/kg/day. For terbutryn: **Acute toxicity:** Tebutryn is slightly toxic. It affects the central nervous system in animals leading to incoordination, convulsions, or laboured breathing. At extremely high dosages, the animals showed swelling and fluid in the lungs and central nervous system. Terbutryn is not a skin sensitizer. **Reproductive effects:** A three generation reproduction study of rats showed that doses of 150 kg/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 at 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. **Fats 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 Agrochemical 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	Des not cause skin sensitization Genotoxicity in vitro: Test Type: Bacteria 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) Results: negative. Remarks: Based on test data Genotoxicity in vivo: Test Type: Mammalian erythrocyte micronucleus test (in vivo cytogenic assay) Species: Rat Application Route: inhalation (vapour) 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 Specie: Rat, male and female Application Route: inhalation (vapour) 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 (vapour) 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 (vapour) Assessment: No significant health effects observed in animals a 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 vapour 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 in humans is unknown.

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

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 chemical agent, by first removing or neutralizing the irritant and then repairing the damage. (Inflammation of the lungs may be 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 lings. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties.

Tridecyl alohol, ethoxylated, phosphate, 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 (AAADSs) (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 there 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 sulfates 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-effects 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 668424-50-0) resulted in Iow toxicity, with a NOEAL of greater than approximately 50 mg/kg/day)calculated based on dose of 1000 ppm in diet). Effects were usually related to hepatic hypertrophy, increase 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 (NOEAL) 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 NOEL 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 800 mg/kg/day, the highest does 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 the 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 result of these studies did not give any indication of treatment-related effects 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 sulfates 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 ethyoxylate 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 oxidization leaving the ethoxysulfate, which is excreted directly. Bu 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 dephosphorylated 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-docecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs are sensitizers that the pure nonoxidezed surfactant itself in 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 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, non-ionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effects, it is difficult to diagnose ACD to these compounds by patch testing.

Gamma-glycidoxypropythrimethoxysilane

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 metablolites that may only caise 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, alkoxysilane with alkoxy groups greater than C2 are classified as moderate concern. Based on available information on methoxysilanes, the possibility that this family causes skin sensitization 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 reins containing the chemical during fiberglass production.

For gamma-glycidoxypropythrimethoxysilan (GPTMS)
GPTMS is subject to rapid hydrolysis, and the observed toxicity is expected to be due primarily to methanol and silanetriois. GPTMS has been tested for acute toxicity by 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 sensitizer 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 dies or were sacrificed from three to five days after initiation of the study. These animals had signs in 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 Effects 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 substances 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 concluded 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 aslo 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). 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 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. Trichlorethylene 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 & Tridecyl Alcohol, Ethoxylated, Phosphated, Pottasium salt & 2,5-BIS(5-Tert-Butyl-2-Benzoxazolyl)Thiophene	No significant cute toxicological data identified in literature search.
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 ceases. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterized by dyspnea, cough and mucus production.
Dipropylene glycol monomethyl ether & 2,2,4-trimethyl-1,3-pentanediol monoisobutyrate & 2-methyl-4-isothiazolin-3-one & Octamethylcyclotet-rasiloxane	This material may be irritating to the eyes, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

Dipropylene glycol monomethyl ether & 2,2,4-trimethyl-1,3-pentanediol monoisobutyrate & 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.
Butyl alcohol propoxylated & 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 characterized by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
Butyl alcohol propoxylated & alcohols C11-14-ISO-, C13-rich, ethoxylated	Polyethers, for example, ethoxylated surfacants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of 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, non-ionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritation effects, 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 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 wit molecular masses below 20,000 g/mol, while PEOs are polymers with mmolecular 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 catylsts. 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 dimethylglyoximime are used. Safety Evaluation of Polyethyene Glycol (PEG) Componds 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 glucol 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 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-methoxyyethoxy)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 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 date 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 1000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (score as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME. Testicular effects included spermatid giant cells, focal tubular hypospermatogenisis, and increased cytoplasmic vacuolisation. Due to 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 1000, 2500 and 4000 mg/kg/day. In this study, significantly-increased red blood cells at 4000 mg/kg/day and significantly-increased urea concentrations in the urine at 2500 mg/kg/day were observed. A

few of the rats given 2500 or 4000 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 1000 or 2500 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, 1200, and 4000 mg/kg/day. Statistically significant changes in relative liver weight were observed at 1200 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 4000 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 I vitro and vivo studies were negative at concentrations up to 5000 micrograms/plate and 5000 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 4000 mg/kg/day four times greater that the limit dose of 1000 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 extents 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 1000 mg/kg/day).

Developmental toxicity: the bulk of the evidence shows that effects on the foetus are not noted in treatments with 1000 mg/kg/day during gestation. At 1250 to 1650 mg/kg/day TGME (in the rat) and 1500 mg/kg/day (in the rabbit), the developmental effects observed included skeletal variants and decreased body weight gain.

Butyl alcohol propoxylated & alcohols C11-14-ISO-, C13-rich, ethoxylated & tridecyl alcohol, 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 the 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 ethyxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotixin, 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. Ployethers, for example, ethoxylated surfacants 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 nonsenitising 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 detection of their corresponding aldehydes in the oxidation mixture.
On basis of the lower irritancy, non-ionic 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.

Butyl alcohol propoxylated & Polyethylene glycol (10) stearyl ether & alcohols C11-14-ISO, C13-rich, ethoxylated & tridecyl alcohol, 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 of 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 he 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 administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.

The ability to non-ionic 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 or 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 non-ionic surfactants, and proteins with poor solubility are not solubilized by non-ionic 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 OAELs 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 weight 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 5800.

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 sensitizers. 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 concern as AEs are not expected to be irritating to the skin at the 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 urticarial 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 urticarial, 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.

Diethylene glycol monobutyl ether & alcohols C11-14-ISO, C13-rich, ethoxylated & tridecyl alcohol, ethoxylated

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

Acute Toxicity: X
Skin Irritation/ Corrosion: √
Serious Eye Damage/Irritation: √
Respirator or Skin Sensitisation: X
Mutagenicity: X
Carcinogenicity: X
Reproductivity: X
STOT – Single Exposure: X
STOT – Repeated Exposure: X
Aspiration Hazard: X

Legend: X – Data either not available or does not fill the criteria for classification
√ - Data available to make classification

SECTION 12: ECOLOGICAL INFORMATION

Toxicity:

Ingredient	Endpoint	Test Duration	Species	Value	Source
Acrylic Polymer	Not available	Not available	Not available	Not available	Not available
Dipropylene glycol monomethyl ether	LC50	96	Fish	1-mg/L	2
	EC50	48	Crustacea	1-930mg/L	2
	EC50	72	Algae or other aquatic plants	6-999mg/L	2
	NOEC	528	Crustacea	>=0.5mg/L	2
2,2,4-trimethyl-1,3-petanediol monoisobutyrate	LC50	96	Fish	>19mg/L	2
	EC50	48	Crustacea	>19mg/L	2
	EC50	72	Algae or other aquatic plants	8.1mg/L	2
	NOEC	72	Algae or other aquatic plants	2mg/L	2
Propylene Glycol	LC50	96	Fish	>10-mh/L	2
	EC50	48	Crustacea	43-500mg/L	2
	EC50	96	Algae or other aquatic plants	19-100mg/L	2
	NOEC	168	Fish	11-530mg/L	2
Butyl alcohol propoxylated	LC50	96	Fish	2-181.5mg/L	2
	EC50	48	Crustacea	2-705mg/L	2
	EC50	72	Algae or other aquatic plants	1-589mg/L	2
	EC0	24	Crustacea	1-989.5mg/L	2
	NOEC	96	Fish	1-mg/L	2
	LC50	96	Fish	564mg/L	2
	EC50	48	Crustacea	>100mg/L	2
	EC50	96	Algae or other aquatic plants	315mg/L	2
	EC0	48	Crustacea	>=100mg/L	2
	NOEC	48	Crustacea	1-mg/L	2
	LC50	96	Fish	104mg/L	2
	EC50	48	Crustacea	>100mg/L	2
	EC50	72	Algae or other aquatic plants	ca.112mg/L	2
	EL10	72	Algae or other aquatic plants	ca.72.3mg/L	2
	NOEC	48	Crustacea	1-mg/L	2
2-methyl-4-isothiazolin-3-one	LC50	96	Fish	4.77mg/L	2
	EC50	48	Crustacea	1.6mg/L	2
	EC50	72	Algae or other aquatic plants	0.0569mg/L	2
	EC10	72	Algae or other aquatic plants	0.0346mg/L	2
	NOEC	48	Algae or other aquatic plants	0.01mg/L	2

1,2-benzisothiazoline-3-one	LC50	96	Fish	1.6mg/L	2
	EC50	48	Crustacea	2.9mg/L	2
	EC50	72	Algae or other aquatic plants	0.0403mg/L	2
	NOEC	72	Algae or other aquatic plants	0.055mg/L	2
Diethylene glycol monobutyl ether	LC50	96	Fish	1-300mg/L	2
	EC50	48	Crustacea	4-950mg/L	2
	EC50	72	Algae or other aquatic plants	1-101mg/L	2
	NOEC	96	Algae or other aquatic plants	>=100mg/L	1
Terbutryn	EC50	96	Algae or other aquatic plants	0.0027mg/L	5
2-octyl-4-isothiazolin-3-one	LC50	96	Fish	0.122mg/L	2
	EC50	96	Algae or other aquatic plants	0.15mg/L	2
	NOEC	504	Crustacea	0.035mg/L	2
Polyethylene glycol (10) stearyl ether	LC50	96	Fish	>5.6mg/L	2
	EC50	48	Crustacea	51mg/L	2
	EC50	72	Algae or other aquatic plants	>10mg/L	2
	EC20	72	Algae or other aquatic plants	0.06mg/L	2
	NOEC	240	Fish	0.16mg/L	2
Octamethylcyclotetrasiloxane	LC50	96	Fish	>0.0063mg/L	2
	EC50	48	Crustacea	>0.015mg/L	2
	EC50	96	Algae or other aquatic plants	>0.022mg/L	2
	NOEC	336	Fish	<=0.0044mg/L	1
Alcohols C11-14-iso-, C13-rich, ethoxylated	Not available	Not available	Not available	Not available	Not available
Tridecyl alohol, ethoxylated, phosphate, potassium salt	Not available	Not available	Not available	Not available	Not available
Tridecyl alohol, ethoxylated	Not available	Not available	Not available	Not available	Not available
2,5-BIS(5-Tert-Butyl-2-Benzoxazolyl) Thiophene	LC50	96	Fish	>100mg/L	2
	EC50	48	Algae or other aquatic plants	>100mg/L	2
	EC50	72	Crustacea	>100mg/L	2
	EC0	24	Crustacea	>=100mg/L	2
	NOEC	528	Crustacea	>=10mg/L	2

Gamma-glycidoxypropythrimethoxysilane	LC50	96	Fish	4.9mg/L	2
	EC50	48	Crustacea	473mg/L	2
	EC50	96	Algae or other aquatic plants	250mg/L	2
	EC0	48	Crustacea	1-mg/L	2
	NOEC	96	Fish	1.5mg/L	2

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

DO NOT discharge into waterways

Persistence and Degradability:

Ingredient	**Persistence: Water/Soil**	**Persistence: Air**
Dipropylene glycol monomethyl ether	HIGH	HIGH
2,2,4-trimethyl-1,3-petanediol monoisobutyrate	LOW	LOW
Propylene Glycol	LOW	LOW
Butyl alcohol propoxylated	LOW	LOW
2-methyl-4-isothiazolin-3-one		
Diethylene glycol monobutyl ether	LOW	LOW
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-glycidoxypropythrimethoxysilane	HIGH	HIGH

Bioaccumulative Potential:

Ingredient	Bioaccumulation
Dipropylene glycol monomethyl ether	LOW (BCF = 100)
2,2,4-trimethyl-1,3-petanediol monoisobutyrate	LOW (LogKOW = 2.9966)
Propylene Glycol	LOW (BCF = 1)
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-glycidoxypropythrimethoxysilane	LOW(LogKOW = 0.9152)

Mobility in Soil:

Ingredient	Mobility
Dipropylene glycol monomethyl ether	LOW (KOC = 10)
2,2,4-trimethyl-1,3-petanediol monoisobutyrate	LOW (KOC = 22.28)
Propylene Glycol	HIGH (KOC = 1)
Butyl alcohol propoxylated	LOW (KOC = 10)
2-methyl-4-isothiazolin-3-one	LOW (KOC = 27.8)
Diethylene glycol monobutyl ether	LOW (KOC = 10)
Terbutryn	LOW (KOC = 3590)

2-octyl-4-isothiazolin-3-one	OW (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-glycidoxypropythrimethoxysilane	LOW (KOC = 90.22)

SECTION 13: DISPOSAL CONSIDERATIONS

Waste Treatment Methods

Product/Packaging Disposal: Legislation addressing waste requirements may differ by country, state and/or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common – the user should investigate:
- Reduction
- Reuse
- Recycling
- Disposal (if all else fails)

This material may be recycled if unused or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use and recycling and or reuse may not always be appropriate.
- DO NOT allow wash water from cleaning or process equipment to enter drains.
- It may be necessary to collect all wash water for treatment before disposal.
- In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
- Where in doubt contact the responsible authority.
- Recycle wherever possible.
- Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified.
- Dispose of by: burial in a land-fill specifically licensed to accept chemical and/or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible materials).
- Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14: TRANSPORT INFORMATION

Labels Required:	
Marine Pollutant	No
HAZCHEM	Not applicable

Land Transport (ADG): Not regulated for transport of dangerous goods

Air transport (ICAO-IATA/DGR): Not regulated for transport of dangerous goods.

Sea Transport (IMDG-Code/GGVsee): Not regulated for transport of dangerous goods.

Transport in bulk according to Annex II of MARPOL and the IBC code: Not applicable

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
Dipropylene glycol monomethyl ether is found on the following regulatory lists:	Australian Inventory of Industrial Chemicals (AIIC).
2,2,4-trimethyl-1,3-petanediol monoisobutyrate is found on the following regulatory lists:	Australian Inventory of Industrial Chemicals (AIIC).
Propylene Glycol 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).
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. Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) – Schedule 6. Australian Inventory of Industrial Chemicals (AIIC).
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. Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) – Schedule5. Australian Inventory of Industrial Chemicals (AIIC).

Terbutryn is found on the following regulatory lists:	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) – Schedule5. 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. Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) – Schedule 6. Australian Inventory of Industrial Chemicals (AIIC).
Polyethylene glycol (10) stearyl ether is found on the following regulatory lists:	Australian Inventory of Industrial Chemicals (AIIC).
Octamethylcyclotetrasiloxane is found on the following regulatory lists:	Australia Hazardous Chemical Information System (HCIS) – Hazardous Chemicals. Australian Inventory of Industrial Chemicals (AIIC). Chemical Footprint Project – Chemicals of High Concern List.
Alcohols C11-14-iso-, C13-rich, ethoxylated is found on the following regulatory lists:	Australian Inventory of Industrial Chemicals (AIIC).
Tridecyl alohol, ethoxylated, phosphate, potassium salt is found on the following regulatory lists:	Australian Inventory of Industrial Chemicals (AIIC).
Tridecyl alohol, ethoxylated is found on the following regulatory lists:	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).
Gamma-glycidoxypropythrimethoxysilane is found on the following regulatory lists:	Australian Inventory of Industrial Chemicals (AIIC).

National Inventory	**Status**
Australia – AIIC:	Yes
Australia Non-Industrial Use:	No (Dipropylene glycol monomethyl ether, 2,2,4-trimethyl-1,3-petanediol monoisobutyrate, Propylene Glycol, 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, Alcohols C11-14-iso-, C13-rich, ethoxylated, Tridecyl alohol, ethoxylated, phosphate, potassium salt, Tridecyl alohol, ethoxylated, 2,5-BIS(5-Tert-Butyl-2-Benzoxazolyl)Thiophene, Gamma-glycidoxypropythrimethoxysilane).
Canada – DSL:	No (terbutryn)

Canada – NDSL:	No (Dipropylene glycol monomethyl ether, 2,2,4-trimethyl-1,3-petanediol monoisobutyrate, Propylene Glycol, 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, Alcohols C11-14-iso-, C13-rich, ethoxylated, Tridecyl alohol, ethoxylated, phosphate, potassium salt, Tridecyl alohol, ethoxylated, 2,5-BIS(5-Tert-Butyl-2-Benzoxazolyl)Thiophene, Gamma-glycidoxypropythrimethoxysilane).
China – IECSC:	Yes
Europe – EINEC/ELINCS/NLP:	No (Alcohols C11-14-iso-, C13-rich, ethoxylated, Tridecyl alohol, ethoxylated, phosphate, potassium salt, Tridecyl alohol, ethoxylated).
Japan – ENCS:	No (Terbutryn, Polyethylene glycol (10) stearyl ether, Alcohols C11-14-iso-, C13-rich, ethoxylated, Tridecyl alohol, ethoxylated, phosphate, potassium salt, Tridecyl alohol, ethoxylated).
Korea – KECI:	Yes
New Zealand - NZloC:	Yes
Philippines – PICCS:	No (terbutryn)
USA – TSCA:	No (terbutryn)
Taiwan – TCSI:	Yes
Mexico – INSQ:	No (Tridecyl alohol, ethoxylated, phosphate, potassium salt, Gamma-glycidoxypropythrimethoxysilane).
Vietnam – NCI:	Yes
Russia – ARIPS:	No (terbutryn, Tridecyl alohol, ethoxylated, phosphate, potassium salt).

Legend: Y = all ingredients re on the inventory, N = Not determined or one or more ingredients are not on the inventory and are exempt from listing (see specific ingredients in brackets).

SECTION 16: PREPARATION INFORMATION OTHER INFORMATION

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
IDHL: Immediately Dangerous to Life or Health Concentrations
OSF: Odour Safety Factor

NOAEL: No Observed Adverse Side Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LODL Limit of Detection
OTV: Odour Threshold Value
BCF: Bio Concentration Factors
BEI: Biological Exposure Index

PREPARED BY:	WHMIS Committee
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EMERGENCY NUMBER:	For 24hr Information call 613-996-6666
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