# Gold Eagle 303 Aerospace Protectant

## Trico Products

Chemwatch Hazard Alert Code: **2**

Chemwatch: **64-8067**
Version No: **2.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **30/06/2023**
Print Date: **12/07/2023**
L.GHS.AUS.EN.E

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

### Product Identifier

| | |
|---|---|
| **Product name** | Gold Eagle 303 Aerospace Protectant |
| **Chemical Name** | Not Applicable |
| **Synonyms** | Pack Size:; 296ml Bottle (PN:30701, 30305M, 30307).; 473ml Bottle (PN: 30702, 30340M30308).; 473ml Bottle (PN: 30382); 946ml Bottle (PN: 30306M, 30313).; 3.78L (PN: 30320).; 59ml Bottle (PN:30302).; 10ml Pouch Wipe (PN: 30322).; 40pk Wipes (PN: 30381). |
| **Chemical formula** | Not Applicable |
| **Other means of identification** | Not Available |

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

| | |
|---|---|
| **Relevant identified uses** | Vinyl protectant.<br>Use according to manufacturer's directions. |

### Details of the manufacturer or supplier of the safety data sheet

| | |
|---|---|
| **Registered company name** | **Trico Products** |
| **Address** | Unit 1, 80 Fairbank Road Clayton VIC 3169 Australia |
| **Telephone** | +61 3 9271 3288 |
| **Fax** | +61 3 9271 3290 |
| **Website** | http://www.tricoproducts.com |
| **Email** | sales@tricoproducts.com.au |

### Emergency telephone number

| | |
|---|---|
| **Association / Organisation** | **Trico Products** |
| **Emergency telephone numbers** | **0 800 764 766 (NZ PIC number)** |
| **Other emergency telephone numbers** | **Not Available** |

## SECTION 2 Hazards identification

### Classification of the substance or mixture

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

**Chemwatch Hazard Ratings**

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

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

| | |
|---|---|
| **Poisons Schedule** | Not Applicable |
| **Classification [1]** | Serious Eye Damage/Eye Irritation Category 1, Reproductive Toxicity Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 3 |
| ***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** | **Danger** |

### Hazard statement(s)

**Page 1 continued...**

| | |
|---|---|
| **H318** | Causes serious eye damage. |
| **H361fd** | Suspected of damaging fertility. Suspected of damaging the unborn child. |
| **H412** | Harmful to aquatic life with long lasting effects. |

### Precautionary statement(s) Prevention

| | |
|---|---|
| **P201** | Obtain special instructions before use. |
| **P280** | Wear protective gloves, protective clothing, eye protection and face protection. |
| **P273** | Avoid release to the environment. |

### Precautionary statement(s) Response

| | |
|---|---|
| **P305+P351+P338** | IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing. |
| **P308+P313** | IF exposed or concerned: Get medical advice/ attention. |
| **P310** | Immediately call a POISON CENTER/doctor/physician/first aider. |

### Precautionary statement(s) Storage

| | |
|---|---|
| **P405** | Store locked up. |

### Precautionary statement(s) Disposal

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

## SECTION 3 Composition / information on ingredients

### Substances

See section below for composition of Mixtures

### Mixtures

| CAS No | %[weight] | Name |
|---|---|---|
| 63148-62-9 | 5-25 | polydimethylsiloxane |
| 160875-66-1 | 0.1-5 | 2-propylheptanol, ethoxylated |
| 50-70-4 | 0.001-5 | D-sorbitol |
| 127087-87-0 | 0.1-3 | 4-nonylphenol, branched, ethoxylated |
| 25322-68-3 | 0.01-0.1 | polyethylene glycol |
| 9014-93-1 | 0.01-0.1 | dinonylphenyl ethoxylate |
| 104810-48-2 | 0.01-0.05 | CG 20-568 ethoxylated |
| 104810-47-1 | 0.01-0.038 | di-CG 20-568 ethoxylated |
| 10377-60-3 | 0.001-0.005 | magnesium nitrate |
| 26172-55-4 | 0.001-0.005 | 5-chloro-2-methyl-4-isothiazolin-3-one |
| 2682-20-4 | 0.001-0.005 | 2-methyl-4-isothiazolin-3-one |
| 7732-18-5 | 80-99 | water |
| ***Legend:*** | *1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available* | |

## SECTION 4 First aid measures

### Description of first aid measures

| | |
|---|---|
| **Eye Contact** | If this product comes in contact with the eyes:<br>▸ Immediately hold eyelids apart and flush the eye continuously with running water.<br>▸ 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.<br>▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.<br>▸ Transport to hospital or doctor without delay.<br>▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. |
| **Skin Contact** | If skin contact occurs:<br>▸ Immediately remove all contaminated clothing, including footwear.<br>▸ Flush skin and hair with running water (and soap if available).<br>▸ Seek medical attention in event of irritation. |
| **Inhalation** | ▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area.<br>▸ Other measures are usually unnecessary. |
| **Ingestion** | ▸ Immediately give a glass of water.<br>▸ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor. |

### Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

## SECTION 5 Firefighting measures

**Continued...**

### Extinguishing media

The product contains a substantial proportion of water, therefore there are no restrictions on the type of extinguishing media which may be used. Choice of extinguishing media should take into account surrounding areas.
Though the material is non-combustible, evaporation of water from the mixture, caused by the heat of nearby fire, may produce floating layers of combustible substances.
In such an event consider:

- foam.
- dry chemical powder.
- carbon dioxide.

### Special hazards arising from the substrate or mixture

| | |
|---|---|
| **Fire Incompatibility** | None known. |

### Advice for firefighters

| | |
|---|---|
| **Fire Fighting** | ▸ Alert Fire Brigade and tell them location and nature of hazard.<br>▸ Wear breathing apparatus plus protective gloves in the event of a fire.<br>▸ Prevent, by any means available, spillage from entering drains or water courses.<br>▸ Use fire fighting procedures suitable for surrounding area.<br>▸ **DO NOT** approach containers suspected to be hot.<br>▸ Cool fire exposed containers with water spray from a protected location.<br>▸ If safe to do so, remove containers from path of fire.<br>▸ Equipment should be thoroughly decontaminated after use. |
| **Fire/Explosion Hazard** | ▸ The material is not readily combustible under normal conditions.<br>▸ However, it will break down under fire conditions and the organic component may burn.<br>▸ Not considered to be a significant fire risk.<br>▸ Heat may cause expansion or decomposition with violent rupture of containers.<br>▸ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO).<br>▸ May emit acrid smoke.<br><br>Decomposes on heating and produces toxic fumes of:<br>carbon dioxide ($CO_2$)<br>silicon dioxide ($SiO_2$)<br>other pyrolysis products typical of burning organic material.<br>May emit poisonous fumes.<br>May emit corrosive fumes.<br>**CARE**: Water in contact with hot liquid may cause foaming and a steam explosion with wide scattering of hot oil and possible severe burns.<br>Foaming may cause overflow of containers and may result in possible fire. |
| **HAZCHEM** | Not Applicable |

## SECTION 6 Accidental release measures

### Personal precautions, protective equipment and emergency procedures

See section 8

### Environmental precautions

See section 12

### Methods and material for containment and cleaning up

| | |
|---|---|
| **Minor Spills** | Environmental hazard - contain spillage.<br>▸ Clean up all spills immediately.<br>▸ Avoid breathing vapours and contact with skin and eyes.<br>▸ Control personal contact with the substance, by using protective equipment.<br>▸ Contain and absorb spill with sand, earth, inert material or vermiculite.<br>▸ Wipe up.<br>▸ Place in a suitable, labelled container for waste disposal. |
| **Major Spills** | Environmental hazard - contain spillage.<br>▸ Silicone fluids, even in small quantities, may present a slip hazard.<br>▸ It may be necessary to rope off area and place warning signs around perimeter.<br>▸ Clean up area from spill, with suitable absorbant, as soon as practically possible.<br>▸ Final cleaning may require use of steam, solvents or detergents.<br>Moderate hazard.<br>▸ Clear area of personnel and move upwind.<br>▸ Alert Fire Brigade and tell them location and nature of hazard.<br>▸ Wear breathing apparatus plus protective gloves.<br>▸ Prevent, by any means available, spillage from entering drains or water course.<br>▸ Stop leak if safe to do so.<br>▸ Contain spill with sand, earth or vermiculite.<br>▸ Collect recoverable product into labelled containers for recycling.<br>▸ Neutralise/decontaminate residue (see Section 13 for specific agent).<br>▸ Collect solid residues and seal in labelled drums for disposal.<br>▸ Wash area and prevent runoff into drains.<br>▸ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.<br>▸ If contamination of drains or waterways occurs, advise emergency services. |

Personal Protective Equipment advice is contained in Section 8 of the SDS.

## SECTION 7 Handling and storage

### Precautions for safe handling

| | |
|---|---|
| **Safe handling** | ▸ **DO NOT** allow clothing wet with material to stay in contact with skin |

**Continued...**

| | |
|---|---|
| | ▸ Avoid all personal contact, including inhalation.<br>▸ Wear protective clothing when risk of exposure occurs.<br>▸ Use in a well-ventilated area.<br>▸ Prevent concentration in hollows and sumps.<br>▸ **DO NOT** enter confined spaces until atmosphere has been checked.<br>▸ **DO NOT** allow material to contact humans, exposed food or food utensils.<br>▸ Avoid contact with incompatible materials.<br>▸ When handling, **DO NOT** eat, drink or smoke.<br>▸ Keep containers securely sealed when not in use.<br>▸ Avoid physical damage to containers.<br>▸ Always wash hands with soap and water after handling.<br>▸ Work clothes should be laundered separately. Launder contaminated clothing before re-use.<br>▸ Use good occupational work practice.<br>▸ Observe manufacturer's storage and handling recommendations contained within this SDS.<br>▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained. |
| **Other information** | ▸ Store in original containers.<br>▸ Keep containers securely sealed.<br>▸ Store in a cool, dry, well-ventilated area.<br>▸ Store away from incompatible materials and foodstuff containers.<br>▸ Protect containers against physical damage and check regularly for leaks.<br>▸ Observe manufacturer's storage and handling recommendations contained within this SDS. |

### Conditions for safe storage, including any incompatibilities

| | |
|---|---|
| **Suitable container** | ▸ Polyethylene or polypropylene container.<br>▸ Packing as recommended by manufacturer.<br>▸ Check all containers are clearly labelled and free from leaks. |
| **Storage incompatibility** | Traces of benzene, a carcinogen, may form when silicones are heated in air above 230 degrees C. Concentrated acids and bases cause degradation of polymer. Boiling water may soften and weaken material.<br>The substance may be or contains a "metalloid"<br>The following elements are considered to be metalloids; boron,silicon, germanium, arsenic, antimony, tellurium and (possibly) polonium<br>The electronegativities and ionisation energies of the metalloids are between those of the metals and nonmetals, so the metalloids exhibit characteristics of both classes. The reactivity of the metalloids depends on the element with which they are reacting. For example, boron acts as a nonmetal when reacting with sodium yet as a metal when reacting with fluorine.<br>Unlike most metals, most metalloids are amphoteric- that is they can act as both an acid and a base. For instance, arsenic forms not only salts such as arsenic halides, by the reaction with certain strong acid, but it also forms arsenites by reactions with strong bases.<br>Most metalloids have a multiplicity of oxidation states or valences. For instance, tellurium has the oxidation states +2, -2, +4, and +6. Metalloids react like non-metals when they react with metals and act like metals when they react with non-metals.<br>▸ Avoid strong acids, bases. |

| | | | | | | |
|---|---|---|---|---|---|---|
| + | x | o | x | + | + | + |

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

*Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.*

## SECTION 8 Exposure controls / personal protection

### Control parameters

**Occupational Exposure Limits (OEL)**

**INGREDIENT DATA**

Not Available

**Emergency Limits**

| Ingredient | TEEL-1 | TEEL-2 | TEEL-3 |
|---|---|---|---|
| polydimethylsiloxane | 65 mg/m3 | 720 mg/m3 | 4,300 mg/m3 |
| 4-nonylphenol, branched, ethoxylated | 30 mg/m3 | 330 mg/m3 | 2,000 mg/m3 |
| 4-nonylphenol, branched, ethoxylated | 30 mg/m3 | 330 mg/m3 | 2,000 mg/m3 |
| polyethylene glycol | 30 mg/m3 | 1,300 mg/m3 | 7,700 mg/m3 |
| magnesium nitrate | 30 mg/m3 | 330 mg/m3 | 2,000 mg/m3 |
| magnesium nitrate | 16 mg/m3 | 180 mg/m3 | 1,100 mg/m3 |
| 5-chloro-2-methyl-4-isothiazolin-3-one | 0.6 mg/m3 | 6.6 mg/m3 | 40 mg/m3 |

| Ingredient | Original IDLH | Revised IDLH |
|---|---|---|
| polydimethylsiloxane | Not Available | Not Available |
| 2-propylheptanol, ethoxylated | Not Available | Not Available |
| D-sorbitol | Not Available | Not Available |

**Continued...**

| Ingredient | Original IDLH | Revised IDLH |
|---|---|---|
| 4-nonylphenol, branched, ethoxylated | Not Available | Not Available |
| polyethylene glycol | Not Available | Not Available |
| dinonylphenyl ethoxylate | Not Available | Not Available |
| CG 20-568 ethoxylated | Not Available | Not Available |
| di-CG 20-568 ethoxylated | Not Available | Not Available |
| magnesium nitrate | Not Available | Not Available |
| 5-chloro-2-methyl-4-isothiazolin-3-one | Not Available | Not Available |
| 2-methyl-4-isothiazolin-3-one | Not Available | Not Available |
| water | Not Available | Not Available |

**Occupational Exposure Banding**

| Ingredient | Occupational Exposure Band Rating | Occupational Exposure Band Limit |
|---|---|---|
| 2-propylheptanol, ethoxylated | E | ≤ 0.1 ppm |
| 4-nonylphenol, branched, ethoxylated | E | ≤ 0.1 ppm |
| dinonylphenyl ethoxylate | E | ≤ 0.1 ppm |
| CG 20-568 ethoxylated | D | > 0.1 to ≤ 1 ppm |
| di-CG 20-568 ethoxylated | D | > 0.1 to ≤ 1 ppm |
| magnesium nitrate | E | ≤ 0.01 mg/m³ |
| 5-chloro-2-methyl-4-isothiazolin-3-one | E | ≤ 0.01 mg/m³ |
| 2-methyl-4-isothiazolin-3-one | D | > 0.01 to ≤ 0.1 mg/m³ |
| ***Notes:*** | *Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.* | |

**MATERIAL DATA**

## Exposure controls

**Appropriate engineering controls**

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.

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

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

Within each range the appropriate value depends on:

| Lower end of the range | Upper end of the range |
|---|---|
| 1: Room air currents minimal or favourable to capture | 1: Disturbing room air currents |
| 2: Contaminants of low toxicity or of nuisance value only | 2: Contaminants of high toxicity |
| 3: Intermittent, low production. | 3: High production, heavy use |
| 4: Large hood or large air mass in motion | 4: Small hood - local control only |

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

**Continued...**

| | |
|---|---|
| **Individual protection measures, such as personal protective equipment** |  |
| **Eye and face protection** | ‣ Safety glasses with side shields.<br>‣ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]<br>‣ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59]. |
| **Skin protection** | See Hand protection below |
| **Hands/feet protection** | ‣ Wear chemical protective gloves, e.g. PVC.<br>‣ Wear safety footwear or safety gumboots, e.g. Rubber<br>**NOTE:**<br>‣ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.<br>‣ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.<br>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.<br>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.<br>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.<br>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:<br>· frequency and duration of contact,<br>· chemical resistance of glove material,<br>· glove thickness and<br>· dexterity<br>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).<br>· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.<br>· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.<br>· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.<br>· Contaminated gloves should be replaced.<br>As defined in ASTM F-739-96 in any application, gloves are rated as:<br>· Excellent when breakthrough time > 480 min<br>· Good when breakthrough time > 20 min<br>· Fair when breakthrough time < 20 min<br>· Poor when glove material degrades<br>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.<br>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.<br>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.<br>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:<br>· 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.<br>· 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<br>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.<br>‣ P.V.C apron.<br>‣ Barrier cream.<br>‣ Skin cleansing cream.<br>‣ Eye wash unit. |

### Recommended material(s)

**GLOVE SELECTION INDEX**

Glove selection is based on a modified presentation of the:
***"Forsberg Clothing Performance Index".***
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
Gold Eagle 303 Aerospace Protectant

| Material | CPI |
|---|---|
| BUTYL | A |
| NEOPRENE | A |
| VITON | A |
| NATURAL RUBBER | C |
| PVA | C |

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion

### Respiratory protection

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

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

| Required minimum protection factor | Maximum gas/vapour concentration present in air p.p.m. (by volume) | Half-face Respirator | Full-Face Respirator |
|---|---|---|---|
| up to 10 | 1000 | A-AUS / Class1 P2 | - |
| up to 50 | 1000 | - | A-AUS / Class 1 P2 |
| up to 50 | 5000 | Airline * | - |
| up to 100 | 5000 | - | A-2 P2 |
| up to 100 | 10000 | - | A-3 P2 |

**Continued...**

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

| | | | |
|---|---|---|---|
| 100+ | | | Airline** |

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

## SECTION 9 Physical and chemical properties

### Information on basic physical and chemical properties

| | |
|---|---|
| **Appearance** | White opaque liquid; mixes with water. |

| | | | |
|---|---|---|---|
| **Physical state** | Liquid | **Relative density (Water = 1)** | 1.01 |
| **Odour** | Not Available | **Partition coefficient n-octanol / water** | Not Available |
| **Odour threshold** | Not Available | **Auto-ignition temperature (°C)** | Not Available |
| **pH (as supplied)** | 7-8 | **Decomposition temperature (°C)** | Not Available |
| **Melting point / freezing point (°C)** | Not Available | **Viscosity (cSt)** | 3@40C |
| **Initial boiling point and boiling range (°C)** | 100 | **Molecular weight (g/mol)** | Not Applicable |
| **Flash point (°C)** | Not Available | **Taste** | Not Available |
| **Evaporation rate** | <1 | **Explosive properties** | Not Available |
| **Flammability** | Not Available | **Oxidising properties** | Not Available |
| **Upper Explosive Limit (%)** | Not Available | **Surface Tension (dyn/cm or mN/m)** | Not Available |
| **Lower Explosive Limit (%)** | Not Available | **Volatile Component (%vol)** | Not Available |
| **Vapour pressure (kPa)** | 2.3 | **Gas group** | Not Available |
| **Solubility in water** | Miscible | **pH as a solution (1%)** | Not Available |
| **Vapour density (Air = 1)** | 0.62 | **VOC g/L** | .59 |

## SECTION 10 Stability and reactivity

| | |
|---|---|
| **Reactivity** | See section 7 |
| **Chemical stability** | ▸ Silicone fluids are stable under normal storage conditions.<br>▸ Hazardous polymerisation will not occur.<br>▸ At temperatures > 150 C, silicones can slowly react with the oxygen in air.<br>▸ When heated > 300 C, silicones can slowly depolymerise to volatile siloxanes whether or not air is present.<br>▸ Unstable in the presence of incompatible materials.<br>▸ Product is considered stable.<br>▸ Hazardous polymerisation will not occur. |
| **Possibility of hazardous reactions** | See section 7 |
| **Conditions to avoid** | See section 7 |
| **Incompatible materials** | See section 7 |
| **Hazardous decomposition products** | See section 5 |

## SECTION 11 Toxicological information

### Information on toxicological effects

| | |
|---|---|
| **Inhaled** | Not normally a hazard due to non-volatile nature of product<br><br>The low vapour pressure of silicone fluids make exposures to potentially harmful vapours unlikely. The vapours of a low molecular weight member of this family, hexamethyldisiloxane, were tolerated by guinea pigs at concentrations of 25000 ppm for 30 minutes without apparent ill-effect. Higher saturated vapour concentrations (39000-40000 ppm) produced death in 15-20 minutes; deaths appeared to occur as a result of respiratory failure as animals removed from exposure, prior to death, almost always survived. Although animal studies show that silicone fluids are removed very slowly from the lungs, their presence is not expected to produce adverse effects; exposure to aerosols is unlikely to result in damage to the health. When heated at high temperatures, the fumes and oxidation products of methyl silicones can be both irritating and produce toxic effects following inhalation. Massive exposures of heated silicone oil can produce central nervous system depression leading to death. |
| **Ingestion** | The material has **NOT** been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern. |
| **Skin Contact** | Limited 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 |

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| | redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.<br>Low molecular weight silicone fluids may exhibit solvent action and may produce skin irritation.<br>One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants<br>Open cuts, abraded or irritated skin should not be exposed to this material<br>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects.<br>Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.<br>Excessive use or prolonged contact may lead to defatting, drying and irritation of sensitive skin |
| **Eye** | When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.<br>When the eyes of human subjects where exposed to silicone fluids, there was evidence of transitory conjunctival irritation within a few hours; this resolved within 24 hours. When applied to the eyes of rabbits, silicone fluids produced transitory irritation which lasted no longer than 48 hours.<br>Injection into the various structures of the eye of animals produced corneal scarring, degenerative changes in the retina, foreign body reaction and cataracts.<br><br>Some nonionic surfactants may produce a localised anaesthetic effect on the cornea; this may effectively eliminate the warning discomfort produced by other substances and lead to corneal injury. Irritant effects range from minimal to severe dependent on the nature of the surfactant, its concentration and the duration of contact. Pain and corneal damage represent the most severe manifestation of irritation. |
| **Chronic** | Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.<br><br>Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.<br>There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals. |

| **Gold Eagle 303 Aerospace Protectant** | **TOXICITY** | **IRRITATION** |
|---|---|---|
| | Not Available | Not Available |

| **polydimethylsiloxane** | **TOXICITY** | **IRRITATION** |
|---|---|---|
| | Dermal (rabbit) LD50: >3000 mg/kg[2] | Eye (rabbit): 100 mg/1h - mild |
| | Oral (Rat) LD50: >35000 mg/kg[2] | |

| **2-propylheptanol, ethoxylated** | **TOXICITY** | **IRRITATION** |
|---|---|---|
| | Not Available | Not Available |

| **D-sorbitol** | **TOXICITY** | **IRRITATION** |
|---|---|---|
| | Oral (Rat) LD50: 15900 mg/kg[2] | Not Available |

| **4-nonylphenol, branched, ethoxylated** | **TOXICITY** | **IRRITATION** |
|---|---|---|
| | Oral (Rat) LD50: 1310 mg/kg[2] | Eye (rabbit): SEVERE |
| | | Eye: adverse effect observed (irritating)[1] |
| | | Eye: no adverse effect observed (not irritating)[1] |
| | | Skin (rabbit): Mild |
| | | Skin: no adverse effect observed (not irritating)[1] |

| **polyethylene glycol** | **TOXICITY** | **IRRITATION** |
|---|---|---|
| | dermal (rat) LD50: >2000 mg/kg[1] | Eye (rabbit): 500mg/24h - mild. |
| | Oral (Rat) LD50: 600 mg/kg[2] | Eye: no adverse effect observed (not irritating)[1] |
| | | Skin (rabbit): 500mg/24h - mild. |
| | | Skin: no adverse effect observed (not irritating)[1] |

| **dinonylphenyl ethoxylate** | **TOXICITY** | **IRRITATION** |
|---|---|---|
| | Not Available | Not Available |

| **CG 20-568 ethoxylated** | **TOXICITY** | **IRRITATION** |
|---|---|---|
| | dermal (rat) LD50: >2000 mg/kg[1] | Skin (guinea pig): Strong sensit. [Ciba-Geigy] |
| | Inhalation(Rat) LC50: >5.8 mg/l4h[1] | Skin (rabbit): non-irritant |
| | Oral (Rat) LD50: >5000 mg/kg[1] | |

| **di-CG 20-568 ethoxylated** | **TOXICITY** | **IRRITATION** |
|---|---|---|
| | Not Available | Not Available |

| **magnesium nitrate** | **TOXICITY** | **IRRITATION** |
|---|---|---|
| | Oral (Rat) LD50: 5440 mg/kg[2] | Eye (rabbit): 500 mg/24h - mild |

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|---|---|
| | Skin (rabbit): 500 mg/24h - mild |

| **5-chloro-2-methyl-4-isothiazolin-3-one** | |
|---|---|
| **TOXICITY** | **IRRITATION** |
| dermal (rat) LD50: >1008 mg/kg[2] | Eye: adverse effect observed (irreversible damage)[1] |
| Inhalation(Rat) LC50: 1.23 mg/l4h[2] | Skin: adverse effect observed (corrosive)[1] |
| Oral (Rat) LD50: 53 mg/kg[2] | Skin: adverse effect observed (irritating)[1] |

| **2-methyl-4-isothiazolin-3-one** | |
|---|---|
| **TOXICITY** | **IRRITATION** |
| dermal (rat) LD50: 242 mg/kg[1] | Eye: adverse effect observed (irreversible damage)[1] |
| Inhalation(Rat) LC50: 0.1 mg/l4h[1] | Skin: adverse effect observed (corrosive)[1] |
| Oral (Rat) LD50: 120 mg/kg[1] | |

| **water** | |
|---|---|
| **TOXICITY** | **IRRITATION** |
| Oral (Rat) LD50: >90000 mg/kg[2] | Not Available |

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

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|---|---|
| **POLYDIMETHYLSILOXANE** | No toxic response noted during 90 day subchronic inhalation toxicity studies The no observable effect level is 450 mg/m3. Non-irritating and non-sensitising in human patch test. [Xerox]*<br>For siloxanes:<br>Effects which based on the reviewed literature do not seem to be problematic are acute toxicity, irritant effects, sensitization and genotoxicity.<br>Some studies indicate that some of the siloxanes may have endocrine disrupting properties, and reproductive effects have caused concern about the possible effects of the siloxanes on humans and the environment.<br>Only few siloxanes are described in the literature with regard to health effects, and it is therefore not possible to make broad conclusions and comparisons of the toxicity related to short-chained linear and cyclic siloxanes based on the present evaluation. Data are primarily found on the cyclic siloxanes D4 (octamethylcyclotetrasiloxane)<br>and D5 (decamethylcyclopentasiloxane) and the short-linear HMDS (hexamethyldisiloxane).<br>These three siloxanes have a relatively low order of acute toxicity by oral, dermal and inhalatory routes and do not require classification for this effect.<br>They are not found to be irritating to skin or eyes and are also not found sensitizing by skin contact. Data on respiratory sensitization have not been identified.<br>Subacute and subchronic toxicity studies show that the liver is the main target organ for D4 which also induces liver cell enzymes. This enzyme induction contributes to the elimination of the substance from the tissues. Primary target organ for D5 exposure by inhalation is the lung. D5 has an enzyme induction profile similar to that of D4. Subacute and subchronic inhalation of HMDS affect in particular the lungs and kidneys in rats.<br>None of the investigated siloxanes show any signs of genotoxic effects *in vitro* or *in vivo*. Preliminary results indicate that D5 has a potential carcinogenic effect.<br>D4 is considered to impair fertility in rats by inhalation and is classified as a substance toxic to reproduction in category 3 with the risk phrase R62 ('Possible risk of impaired fertility').<br>The results of a study to screen for oestrogen activity indicate that D4 has very weak oestrogenic and antioestrogenic activity and is a partial agonist (enhances the effect of the estrogen). It is not uncommon for compounds that are weakly<br>oestrogenic to also have antioestrogenic properties. Comparison of the oestrogenic potency of D4 relative to ethinyloestradiol (steroid hormone) indicates that D4 is 585,000 times less potent than ethinyloestradiol in the rat stain Sprague- Dawley and 3.7 million times less potent than ethinyloestradiol in the Fisher-344 rat strain. Because of the lack of effects on other endpoints designated to assess oestrogenicity, the oestrogenicity as mode of action for the D4 reproductive effects has been questioned. An indirect mode of action causing a delay of the LH (luteinising hormone) surge necessary for optimal timing of ovulation has been suggested as the mechanism.<br>Based on the reviewed information, the critical effects of the siloxanes are impaired fertility (D4) and potential carcinogenic effects (uterine tumours in females). Furthermore there seem to be some effects on various organs following<br>repeated exposures, the liver (D4), kidney (HMDS) and lung (D5 and HMDS) being the target organs.<br>A possible oestrogenic effect contributing to the reproductive toxicity of D4 is debated. There seems however to be some indication that this toxicity may be caused by another mechanism than oestrogen activity.<br>Studies are available for linear siloxanes from an analogue group comprising di- to hexa- siloxanes, as well as key physicochemical properties, The results of the acute toxicity studies for this analogue group are in agreement: there is no evidence from any of the available studies that the substances in this group have any potential for acute toxicity (in terms of either lethality or adverse clinical effects) by any route up to and exceeding the maximum dose levels tested according to current OECD guidelines. It is therefore valid to read-across the lack of acute toxicity between the members of the group where there are data gaps<br>The metabolism of silanes and siloxanes is influenced by the chemistry of silicon, and it is fundamentally different from that of carbon compounds. These differences are due to the fact that silicon is more electropositive than carbon; Si-Si bonds are less stable than C-C bonds and Si-O bonds form very readily, the latter due to their high bond energy. Functional groups such as -OH, -CO2H, and -CH2OH are commonly seen in organic drug metabolites. If such functionalities are formed from siloxane metabolism, they will undergo rearrangement with migration of the Si atom from carbon to oxygen. Consequently, alpha hydroxysilanes may isomerise to silanols and this provides a mechanism by which very polar metabolites may be formed from highly hydrophobic alkylsiloxanes in relatively few metabolic steps |
| **4-NONYLPHENOL, BRANCHED, ETHOXYLATED** | For nonylphenol and its compounds:<br>Alkylphenols like nonylphenol and bisphenol A have estrogenic effects in the body. They are known as xenoestrogens. Estrogenic substances and other endocrine disruptors are compounds that have hormone-like effects in both wildlife and humans. Xenoestrogens usually function by binding to estrogen receptors and acting competitively against natural estrogens. Nonylphenol has been found to act as an agonist of GPER (G protein-coupled estrogen receptor),. Nonylphenol has been shown to mimic the natural hormone 17beta-estradiol, and it competes with the endogeous hormone for binding with the estrogen receptors ERalpha and ERbeta.<br>Effects in pregnant women.<br>Subcutaneous injections of nonylphenol in late pregnancy causes the expression of certain placental and uterine proteins, namely CaBP-9k, which suggest it can be transferred through the placenta to the fetus. It has also been shown to have a higher potency on the first trimester placenta than the endogenous estrogen 17beta-estradiol. In addition, early prenatal exposure to low doses of nonylphenol cause an increase in apoptosis (programmed cell death) in placental cells. These “low doses” ranged from 10-13-10-9 M, which is lower than what is generally found in the environment.<br>Nonylphenol has also been shown to affect cytokine signaling molecule secretions in the human placenta. In vitro cell cultures of human placenta during the first trimester were treated with nonylphenol, which increase the secretion of cytokines including interferon gamma, interleukin 4, and interleukin 10, and reduced the secretion of tumor necrosis factor alpha. This unbalanced cytokine profile at this part of pregnancy has been documented to result in implantation failure, pregnancy loss, and other complications.<br>Effects on metabolism<br>Nonylphenol has been shown to act as an obesity enhancing chemical or obesogen, though it has paradoxically been shown to have anti-obesity |

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properties. Growing embryos and newborns are particularly vulnerable when exposed to nonylphenol because low-doses can disrupt sensitive processes that occur during these important developmental periods. Prenatal and perinatal exposure to nonylphenol has been linked with developmental abnormalities in adipose tissue and therefore in metabolic hormone synthesis and release. Specifically, by acting as an estrogen mimic, nonylphenol has generally been shown to interfere with hypothalamic appetite control. The hypothalamus responds to the hormone leptin, which signals the feeling of fullness after eating, and nonylphenol has been shown to both increase and decrease eating behavior by interfering with leptin signaling in the midbrain. Nonylphenol has been shown mimic the action of leptin on neuropeptide Y and anorectic POMC neurons, which has an anti-obesity effect by decreasing eating behavior. This was seen when estrogen or estrogen mimics were injected into the ventromedial hypothalamus. On the other hand, nonylphenol has been shown to increase food intake and have obesity enhancing properties by lowering the expression of these anorexigenic neurons in the brain. Additionally, nonylphenol affects the expression of ghrelin: an enzyme produced by the stomach that stimulates appetite. Ghrelin expression is positively regulated by estrogen signaling in the stomach, and it is also important in guiding the differentiation of stem cells into adipocytes (fat cells). Thus, acting as an estrogen mimic, prenatal and perinatal exposure to nonylphenol has been shown to increase appetite and encourage the body to store fat later in life. Finally, long-term exposure to nonylphenol has been shown to affect insulin signaling in the liver of adult male rats.

Cancer

Nonylphenol exposure has also been associated with breast cancer. It has been shown to promote the proliferation of breast cancer cells, due to its agonistic activity on ERalpha (estrogen receptor alpha) in estrogen-dependent and estrogen-independent breast cancer cells. Some argue that nonylphenol's suggested estrogenic effect coupled with its widespread human exposure could potentially influence hormone-dependent breast cancer disease

For high boiling ethylene glycol ethers (typically triethylene- and tetraethylene glycol ethers):

**Skin absorption:** Available skin absorption data for triethylene glycol ether (TGBE), triethylene glycol methyl ether (TGME), and triethylene glycol ethylene ether (TGEE) suggest that the rate of absorption in skin of these three glycol ethers is 22 to 34 micrograms/cm2/hr, with the methyl ether having the highest permeation constant and the butyl ether having the lowest. The rates of absorption of TGBE, TGEE and TGME are at least 100-fold less than EGME, EGEE, and EGBE, their ethylene glycol monoalkyl ether counterparts, which have absorption rates that range from 214 to 2890 micrograms/ cm2/hr . Therefore, an increase in either the chain length of the alkyl substituent or the number of ethylene glycol moieties appears to lead to a decreased rate of percutaneous absorption. However, since the ratio of the change in values of the ethylene glycol to the diethylene glycol series is larger than that
of the diethylene glycol to triethylene glycol series , the effect of the length of the chain and number of ethylene glycol moieties on absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetraethylene glycol methyl; ether (TetraME) and tetraethylene glycol butyl ether (TetraBE) are expected to be less permeable to skin than TGME and TGBE, the differences in permeation between these molecules may only be slight.

**Metabolism:** The main metabolic pathway for metabolism of ethylene glycol monoalkyl ethers (EGME, EGEE, and EGBE) is oxidation via alcohol and aldehyde dehydrogenases (ALD/ADH) that leads to the formation of an alkoxy acids. Alkoxy acids are the only toxicologically significant metabolites of glycol ethers that have been detected *in vivo.* The principal metabolite of TGME is believed to be 2-[2-(2-methoxyethoxy)ethoxy] acetic acid . Although ethylene glycol, a known kidney toxicant, has been identified as an impurity or a minor metabolite of glycol ethers in animal studies it does not appear to contribute to the toxicity of glycol ethers.

The metabolites of category members are not likely to be metabolized to any large extent to toxic molecules such as ethylene glycol or the mono alkoxy acids because metabolic breakdown of the ether linkages also has to occur

**Acute toxicity:** Category members generally display low acute toxicity by the oral, inhalation and dermal routes of exposure. Signs of toxicity in animals receiving lethal oral doses of TGBE included loss of righting reflex and flaccid muscle tone, coma, and heavy breathing. Animals administered lethal oral doses of TGEE exhibited lethargy, ataxia, blood in the urogenital area and piloerection before death.

**Irritation:** The data indicate that the glycol ethers may cause mild to moderate skin irritation. TGEE and TGBE are highly irritating to the eyes. Other category members show low eye irritation.

**Repeat dose toxicity**: Results of these studies suggest that repeated exposure to moderate to high doses of the glycol
ethers in this category is required to produce systemic toxicity

In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME. Testicular effects included spermatid giant cells, focal tubular hypospermatogenesis, and increased cytoplasmic vacuolisation . Due to a high incidence of similar spontaneous changes
in normal New Zealand White rabbits , the testicular effects were considered not to be related to treatment . Thus, the NOAELs for TGME, TGEE and TGBE were established at 1000 mg/kg/day. Findings from this report were considered
unremarkable.

A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day . In this study, significantly-increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500 mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or
haemolysed blood in the stomach These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats

In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity

**Mutagenicity:** Mutagenicity studies have been conducted for several category members. All in vitro and in vivo studies were negative at concentrations up to 5,000 micrograms/plate and 5,000 mg/kg, respectively, indicating that the category members are not genotoxic at the concentrations used in these studies. The uniformly negative outcomes of various mutagenicity studies performed on category members lessen the concern for carcinogenicity.

**Reproductive toxicity:** Although mating studies with either the category members or surrogates have not been performed, several of the repeated dose toxicity tests with the surrogates have included examination of reproductive organs. A lower molecular weight glycol ether, ethylene glycol methyl ether (EGME), has been shown to be a testicular toxicant. In addition, results of repeated dose toxicity tests with TGME clearly show testicular toxicity at an oral dose of 4,000 mg/kg/day four times greater that the limit dose of 1,000 mg/kg/day recommended for repeat dose studies. It should be noted that TGME is 350 times less potent for testicular effects than EGME. TGBE is not associated with testicular toxicity, TetraME is not likely to be metabolised by any large extent to 2-MAA (the toxic metabolite of EGME), and a mixture containing predominantly methylated glycol ethers in the C5-C11 range does not produce testicular toxicity (even when administered intravenously at 1,000 mg/kg/day).

**Developmental toxicity**: The bulk of the evidence shows that effects on the foetus are not noted in treatments with . 1,000 mg/kg/day during gestation. At 1,250 to 1,650 mg/kg/day TGME (in the rat) and 1,500 mg/kg/day (in the rabbit), the developmental effects observed included skeletal variants and decreased body weight gain.

for nonylphenol:

Nonylphenol was studied for oral toxicity in rats in a 28-day repeat dose toxicity test at doses of 0, 4, 15, 60 and 250 mg/kg/day. Changes suggesting renal dysfunction were mainly noted in both sexes given 250 mg/kg. Liver weights were increased in males given 60 mg/kg and in both sexes given 250 mg/kg group. Histopathologically, hypertrophy of the centrilobular hepatocytes was noted in both sexes given 250 mg/kg. Kidney weights were increased in males given 250 mg/kg and macroscopically, disseminated white spots, enlargement and pelvic dilatation were noted in females given 250 mg/kg. Histopathologically, the following lesions were noted in the 250 mg/kg group: basophilic change of the proximal tubules in both sexes, single cell necrosis of the proximal tubules, inflammatory cell infiltration in the interstitium and casts in females, basophilic change and dilatation of the collecting tubules in both sexes, simple hyperplasia of the pelvic mucosa and pelvic dilatation in females. In the urinary bladder, simple hyperplasia was noted in both sexes given 250 mg/kg. In the caecum, macroscopic dilatation was noted in both

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| | sexes given 250 mg/kg. Almost all changes except those in the kidney disappeared after a 14-day recovery period. The NOELs for males and females are considered to be 15 mg/kg/day and 60 mg/kg/day, respectively, under the conditions of the present study.<br>Nonylphenol was not mutagenic to Salmonella typhimurium, TA100, TA1535, TA98, TA1537 and Escherichia coli WP2 uvrA, with or without an exogeneous metabolic activation system.<br>Nonylphenol induced neither structural chromosomal aberrations nor polyploidy in CHL/IU cells, in the absence or presence of an exogenous metabolic activation system.<br><br>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.<br>for linear material: Maternal effects, effects on fertility recorded. |
| **POLYETHYLENE GLYCOL** | for molecular weights (200-8000) * Oral (rat) LD50: 31000->50000 mg/kg Oral (mice) LD50: 38000->50000 mg/kg Oral (g.pig) LD50: 17000->50000 mg/kg Oral (rabbit) LD50: 14000->50000 mg/kg * AIHA WEEL Guides Intraperitoneal (mice) LD50: 3100-12900 mg/kg<br>for polyethylene glycols<br>Pure polyethylene glycols have essentially similar toxicity, with toxicity being inverse to molecular weights. Absorption from the gastrointestinal tract decreases with increasing molecular weight<br>The G.I. absorption of a series of polyethylene glycols has been studied. Polyethylene glycols having average molecular weights of 4000 and 6000 showed no absorption from the rat intestine over a five-hour period, while polyethylene glycols of 1000 and 1540 molecular weights showed a slight absorption amounting to less than 2% of the total dose during the same period. When 1 g doses of polyethylene glycols of molecular weight 1000 (PEG 1000) and 6000 (PEG 6000) were given intravenously to six human subjects, 85% of PEG 1000 and 96% of PEG 6000 were excreted in the urine in 12 hours. When these two same materials in 10 g doses were given orally to five human subjects, none of the PEG 6000 was found in the urine in the following 24 hours, whereas about 8% of PEG 1000 administered was found to excrete in urine within 24 hours<br>When PEG 400 was given intravenously to three human subjects, an average of 77% recovery of this material was found in the urine in 12 hours. However, when the same substance was given orally to the same three human subjects, a recovery of between 40 and 50% of the dose was determined in the urine in the course of the following 24 hours. Single oral doses of PEG 400 were incompletely recovered from urine and faeces of rabbits even when collection of excreta was continued as long as four days following the dose. Evidence from all these and other studies indicate that ethylene glycol is not formed as a metabolite of PEG 400<br>Prolonged skin contact of PEG 1500 and 4000 upon the skin of rabbits in dosages of 10 g/kg bw showed no deleterious effects on internal organs and little, if any, of the materials was absorbed through the skin.<br>Although early reports indicated that skin sensitization was observed among a few human subjects and in guinea pigs tested with certain polyethylene glycols, later studies showed that currently produced materials were without irritating or sensitizing properties. However, recent report (Fischer, 1978) demonstrated that four patients showed allergic reactions to lower molecular weight liquid polyethylene glycols in topical medications. Two had immediate urticarial reactions to PEG 400. Two other patients had delayed allergic eczematous reactions, one to PEG 200, and one to PEG 300. |
| **CG 20-568 ETHOXYLATED** | Inhalation (rat) LC50: > 5.8 mg/l Aerosol Eye (rabbit): non-irritant Ames Test: Non Mutagenic Increase in absolute liver weight observed. No effect on microsomal protein content was noted, while a dose-dependent decrease in cytosolic protein content was observed. Decreased microsomal hydrolase activity and glutathione S-transferase activity were observed at 50 and 100 mg/kg. Comparatively, increased peroxyiomal fatty acid ß-oxidation activity and bilirubin UDP-glucuronosyltransferase activity were observed at all tested doses. Dose-dependent increases in lauric acid 11- and 12-hydroxylase activity and decreases in morphine UDP-glucuronosyltransferase activity were noted. Ethoxyresorufin O-de-ethylase activity was significantly decreased at 100 mg/kg and pentoxyresorufin O-dependentylase was increased at 50 mg/kg. Immunohistochemical studies indicated conflicting effects on various microsomal P450 isoform levels. Total number and structural changes were increased in hepatocyte organelles. Enlarged hepatic peroxisomes containing matrical plates were observed at 50 and 100 mg/kg Dam livers showed "moderate to striking peroxisome proliferation at all investigated periods of gestation." Peroxisomes were identified as "slightly increased" or "increased." No mitochondrial changes and a slight decrease in glycogen content on GD 21 were noted. Absolute liver weight was increased. Additionally, peroxisomal fatty acid ß-oxidation, lauric acid 11- and 12-hydroxylase, and catalase activities were increased at all time points. Liver malondialdehyde content was increased at GD 15. Selenium-dependent and -independent glutathione peroxidase activities were decreased at GD 15, 18, and 21, and 21, respectively. Subcutaneous and skeletal muscular hemorrhages within the connective tissues noted. Peroxisome proliferation was moderately to strikingly increased at all time points in fetuses. Peroxisome size was also increased. Increased mitochondrial volume and enlarged mitochondria were noted on GD 18 and 21. Glycogen content was "marginal" on GD 18 and 21. Absolute liver weight was not affected. Peroxisomal fatty acid ß-oxidation activity was increased at all time points while lauric acid 11- and 12-hydroxylase, and catalase activities were increased at GD 18 and 21. Liver malondialdehyde content was increased at GD 21. Liver total glutathione content and liver content of reduced glutathione were decreased on GD 21 while selenium-dependent glutathione peroxidase activity was increased on G |
| **DI-CG 20-568 ETHOXYLATED** | Increase in absolute liver weight observed. No effect on microsomal protein content was noted, while a dose-dependent decrease in cytosolic protein content was observed. Decreased microsomal hydrolase activity and glutathione S-transferase activity were observed at 50 and 100 mg/kg. Comparatively, increased peroxyiomal fatty acid ß-oxidation activity and bilirubin UDP-glucuronosyltransferase activity were observed at all tested doses. Dose-dependent increases in lauric acid 11- and 12-hydroxylase activity and decreases in morphine UDP-glucuronosyltransferase activity were noted. Ethoxyresorufin O-de-ethylase activity was significantly decreased at 100 mg/kg and pentoxyresorufin O-dependentylase was increased at 50 mg/kg. Immunohistochemical studies indicated conflicting effects on various microsomal P450 isoform levels. Total number and structural changes were increased in hepatocyte organelles. Enlarged hepatic peroxisomes containing matrical plates were observed at 50 and 100 mg/kg. No clinical signs were observed at 10 mg/kg/day for F and at 10 and 50 mg/kg/day for M. Drooling was observed in M and F at 200 and 1000 mg/kg. Alopecia was observed in F at 50 and 1000 mg/kg. 4/5 M died at the highest dose. At 10, 200, and 100 mg/kg, one F died in each group. Decreased body weights were seen at 200 and 1000 mg/kg and 1000 mg/kg in M and F, respectively. Increased albumin levels were noted in all dosed F and 50 and 200 mg/kg M. ß-Globulin levels were decreased in 200 mg/kg M and 200 and 1000 mg/kg F. Increased patelet counts in M and F at =200 mg/kg and decreasedin lymphocytes in three animals at 1000 mg/kg were noted. Increased alkaline phosphatase activity was observed in M and F at 200 mg/kg and F at 1000 mg/kg. Reduced organ size and weight changes were observed in M and F at 200 and/or 1000 mg/kg. A dose-dependent increase in the development of liver necrosis foci was observed starting at 50 mg/kg. Renal tubular degeneration was noted in all M at 1000 mg/kg. Dam livers showed "moderate to striking peroxisome proliferation at all investigated periods of gestation." Peroxisomes were identified as "slightly increased" or "increased." No mitochondrial changes and a slight decrease in glycogen content on GD 21 were noted. Absolute liver weight was increased. Additionally, peroxisomal fatty acid ß-oxidation, lauric acid 11- and 12-hydroxylase, and catalase activities were increased at all time points. Liver malondialdehyde content was increased at GD 15. Selenium-dependent and -independent glutathione peroxidase activities were decreased at GD 15, 18, and 21, and 21, respectively. Subcutaneous and skeletal muscular hemorrhages within the connective tissues noted. Peroxisome proliferation was moderately to strikingly increased at all time points in fetuses. Peroxisome size was also increased. Increased mitochondrial volume and enlarged mitochondria were noted on GD 18 and 21. Glycogen content was "marginal" on GD 18 and 21. Absolute liver weight was not affected. Peroxisomal fatty acid ß-oxidation activity was increased at all time points while lauric acid 11- and 12-hydroxylase, and catalase activities were increased at GD 18 and 21. Liver malondialdehyde content was increased at GD 21. Liver total glutathione content and liver content of reduced glutathione were decreased on GD 21 while selenium-dependent glutathione peroxidase activity was increased on GD 21. |
| **MAGNESIUM NITRATE** | Magnesium nitrate heaxahydrate is a methaemoglobin-forming agent which if inhaled or ingested in high enough concentrations may cause fatigue, headache, dizziness. (Source: I.L.O. Encyclopaedia) |
| **5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE** | Considered to be the major sensitiser in Kathon CG (1) |
| **2-METHYL-4-ISOTHIAZOLIN-3-ONE** | **NOTE:** Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.<br>Considered to be a minor sensitiser in Kathon CG (1) |

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| **POLYDIMETHYLSILOXANE & POLYETHYLENE GLYCOL & MAGNESIUM NITRATE & 5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE & 2-METHYL-4-ISOTHIAZOLIN-3-ONE** | The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. |
| **2-PROPYLHEPTANOL, ETHOXYLATED & 4-NONYLPHENOL, BRANCHED, ETHOXYLATED** | Human beings have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents, and other cleaning products . Exposure to these chemicals can occur through ingestion, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that volumes well above a reasonable intake level would have to occur to produce any toxic response. Moreover, no fatal case of poisoning with alcohol ethoxylates has ever been reported. Multiple studies investigating the acute toxicity of alcohol ethoxylates have shown that the use of these compounds is of low concern in terms of oral and dermal toxicity .<br>Clinical animal studies indicate these chemicals may produce gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhea, and lethargy. Similarly, slight to severe irritation of the skin or eye was generated when undiluted alcohol ethoxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotoxin, carcinogen, or mutagen (HERA 2007). No information was available on levels at which these effects might occur, though toxicity is thought to be substantially lower than that of nonylphenol ethoxylates. Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.<br>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 .<br>On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However,their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose allergic contact dermatitis (ACD) to these compounds by patch testing<br>Overall, alcohol alkoxylates (AAs) are not expected to be systemically toxic, although some short chain ethylene glycol ethers, e.g. methyl and ethyl homologues are of concern for a range of adverse health effects. They include skin and eye irritation, liver and kidney damage, bone marrow and central nervous system (CNS) depression, testicular atrophy, developmental toxicity, and immunotoxicity. For higher propyl and butyl homologues, the toxicity involves haemolysis (anaemia) with secondary effects relating to haemosiderin accumulation in the spleen, liver and kidney, and compensatory haematopoiesis in the bone marrow. Systemic toxicity was shown to decrease with increasing alkyl chain lengths and/or alkoxylation degrees (ECETOC, 2005; US EPA, 2010). The chemicals ethylene glycol hexyl ether (with a longer alkyl chain length, CAS No. 112-25-4) and diethylene glycol butyl ether (with a higher ethoxylation degree, CAS No. 112-34-5) have no evidence of systemic effects including haemolysis.<br>Commercially available AAs are mixtures of homologues of varying carbon chain lengths and it is possible that some of the chemicals with an average alkyl chain length C >=6 may also contain shorter alkyl chains C <6. It is not practical to quantify the proportion of shorter C <6 chain lengths present in such chemicals, or these shorter chain lengths may not be present at all. The available data suggest a lack of systemic toxicity for the AE chemicals with potential short alkyl chain presence (NICNASa); therefore, the toxicity of the chemicals in this assessment is unlikely to be significantly affected by the presence of shorter chain alkyl groups. |
| **2-PROPYLHEPTANOL, ETHOXYLATED & 4-NONYLPHENOL, BRANCHED, ETHOXYLATED & DINONYLPHENYL ETHOXYLATE** | Alcohol ethoxylates are according to CESIO (2000) classified as Irritant or Harmful depending on the number of EO-units:<br>EO < 5 gives Irritant (Xi) with R38 (Irritating to skin) and R41 (Risk of serious damage to eyes)<br>EO > 5-15 gives Harmful (Xn) with R22 (Harmful if swallowed) - R38/41<br>EO > 15-20 gives Harmful (Xn) with R22-41<br>>20 EO is not classified (CESIO 2000)<br>Oxo-AE, C13 EO10 and C13 EO15, are Irritating (Xi) with R36/38 (Irritating to eyes and skin) .<br>AE are not included in Annex 1 of the list of dangerous substances of the Council Directive 67/548/EEC<br><br>In general, alcohol ethoxylates (AE) are readily absorbed through the skin of guinea pigs and rats and through the gastrointestinal mucosa of rats. AE are quickly eliminated from the body through the urine, faeces, and expired air ($CO_2$).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 ($CO_2$) ). The metabolism of C12 AE yields PEG, carboxylic acids, and $CO_2$ as metabolites. The LD50 values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.<br><br>The ability of nonionic surfactants to cause a swelling of the stratum corneum of guinea pig skin has been studied. The swelling mechanism of the skin involves a combination of ionic binding of the hydrophilic group as well as hydrophobic interactions of the alkyl chain with the substrate. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants. A substantial amount of toxicological data and information in vivo and in vitro demonstrates that there is no evidence for alcohol ethoxylates (AEs) being genotoxic, mutagenic or carcinogenic. No adverse reproductive or developmental effects were observed. The majority of available toxicity studies revealed NOAELs in excess of 100 mg/kg bw/d but the lowest NOAEL for an individual AE was established to be 50 mg/kg bw/day. This value was subsequently considered as a conservative, representative value in the risk assessment of AE. The effects were restricted to changes in organ weights with no histopathological organ changes with the exception of liver hypertrophy (indicative of an adaptive response to metabolism rather than a toxic effect). It is noteworthy that there was practically no difference in the NOAEL in oral studies of 90-day or 2 years of duration in rats. A comparison of the aggregate consumer exposure and the systemic NOAEL (taking into account an oral absorption value of 75%) results in a Margin of Exposure of 5,800. Taking into account the conservatism in the exposure assessment and the assigned systemic NOAEL, this margin of exposure is considered more than adequate to account for the inherent uncertainty and variability of the hazard database and inter and intra-species extrapolations.<br><br>AEs are not contact sensitisers. Neat AE are irritating to eyes and skin. The irritation potential of aqueous solutions of AEs depends on concentrations. Local dermal effects due to direct or indirect skin contact in certain use scenarios where the products are diluted are not of concern as AEs are not expected to be irritating to the skin at in-use concentrations. Potential irritation of the respiratory tract is not a concern given the very low levels of airborne AE generated as a consequence of spray cleaner aerosols or laundry powder detergent dust.<br><br>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-PROPYLHEPTANOL, ETHOXYLATED & DINONYLPHENYL ETHOXYLATE & CG 20-568 ETHOXYLATED & DI-CG 20-568 ETHOXYLATED & 5-CHLORO-2-METHYL-** | No significant acute toxicological data identified in literature search. |

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| **4-ISOTHIAZOLIN-3-ONE & 2-METHYL-4-ISOTHIAZOLIN-3-ONE & WATER** | |
| **4-NONYLPHENOL, BRANCHED, ETHOXYLATED & POLYETHYLENE GLYCOL & CG 20-568 ETHOXYLATED & DI-CG 20-568 ETHOXYLATED** | Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.<br>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 .<br>On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.<br>Allergic Contact Dermatitis—Formation, Structural Requirements,and Reactivity of Skin Sensitizers.<br>Ann-Therese Karlberg et al; Chem. Res. Toxicol.2008,21,53-69<br>Polyethylene glycols (PEGs) have a wide variety of PEG-derived mixtures due to their readily linkable terminal primary hydroxyl groups in combination with many possible compounds and complexes such as ethers, fatty acids, castor oils, amines, propylene glycols, among other derivatives. PEGs and their derivatives are broadly utilized in cosmetic products as surfactants, emulsifiers, cleansing agents, humectants, and skin conditioners.<br>PEGs and PEG derivatives were generally regulated as safe for use in cosmetics, with the conditions that impurities and by-products, such as ethylene oxides and 1,4-dioxane, which are known carcinogenic materials, should be removed before they are mixed in cosmetic formulations. Most PEGs are commonly available commercially as mixtures of different oligomer sizes in broadly- or narrowly-defined molecular weight (MW) ranges. For instance, PEG-10,000 typically designates a mixture of PEG molecules (n = 195 to 265) having an average MW of 10,000. PEG is also known as polyethylene oxide (PEO) or polyoxyethylene (POE), with the three names being chemical synonyms. However, PEGs mainly refer to oligomers and polymers with molecular masses below 20,000 g/mol, while PEOs are polymers with molecular masses above 20,000 g/mol, and POEs are polymers of any molecular mass. Relatively small molecular weight PEGs are produced by the chemical reaction between ethylene oxide and water or ethylene glycol (or other ethylene glycol oligomers), as catalyzed by acidic or basic catalysts. To produce PEO or high-molecular weight PEGs, synthesis is performed by suspension polymerization. It is necessary to hold the growing polymer chain in solution during the course of the poly-condensation process. The reaction is catalyzed by magnesium-, aluminum-, or calcium-organoelement compounds. To prevent coagulation of polymer chains in the solution, chelating additives such as dimethylglyoxime are used<br>Safety Evaluation of Polyethyene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of Toxicology http://doi.org/10.5487/TR.2015.31.2.105 |
| **4-NONYLPHENOL, BRANCHED, ETHOXYLATED & POLYETHYLENE GLYCOL & MAGNESIUM NITRATE & 5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE & 2-METHYL-4-ISOTHIAZOLIN-3-ONE** | 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. |
| **CG 20-568 ETHOXYLATED & DI-CG 20-568 ETHOXYLATED & 5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE & 2-METHYL-4-ISOTHIAZOLIN-3-ONE** | The following information refers to contact allergens as a group and may not be specific to this product.<br>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. |
| **CG 20-568 ETHOXYLATED & DI-CG 20-568 ETHOXYLATED** | For benzotriazoles<br>There are several indications that the effects of phenolic benzotriazoles described in the literature might be caused by endocrine disruption, e.g. reduced concentrations of testosterone, higher concentrations of CYP 450, or higher activity of ethoxyresorufin-O-deethylase (EROD-activity). As in these cases there are also indications for toxic effects on the liver reported, the effects might actually be only secondary effects. With the present knowledge it is not possible to attribute them unambiguously as endocrine adverse effects of an equivalent level of concern.<br>Several benzotriazole UV stabilisers showed significant human aryl hydrocarbon receptor (AhR) ligand activity.The AhR has roles in regulating immunity, stem cell maintenance, and cellular differentiation A study indicated that certain benzotriazole UV stabilisers have the potential to accumulate and exert potent physiological effects in humans, analogous to polycyclic aromatic hydrocarbons and dioxins, which are known stable and toxic ligands. The polycyclic aromatic hydrocarbon the polycyclic aromatic hydrocarbon, benzo[a]pyrene (BaP), a ligand for AhR, induces its own metabolism and bioactivation to a toxic metabolites.<br>Benzotriazole is the core structure present within the phenolic benzotriazole class. In vitro metabolism with rat liver microsomes yielded formation of 5- and 4-hydroxybenzotriazole (1.6 and 0.32% of the amount added, respectively).Overall metabolism was low (<5% of the total amount added) Oral acute studies in rats and mice yielded LD50 values that ranged from 560 to 909 mg/kg. Intraperitoneal LD50 values in mice and rats ranged from 400-1000 and 500-900 mg/kg, respectively. A mouse intravenous LD50 of 238 mg/kg was identified. Dermal LD50 values were =1000 mg/kg in rats and rabbits, and inhalation LC50 values in rats were 1.5 mg/L and 1.91 mg/L/3 hours). Subchronic and short-term studies showed that oral administration to mice produced minimal effects on body weight while dose-dependent decreases in body weight were observed in rats. Endocrine effects, normocytic anemia, and leukopenia were noted in rats dosed for 26 weeks.The TDLo was 109 mg/kg. No effects on deaths and no clinical symptoms were noted in mice or rats orally administered (in food) benzotriazole =78 weeks. Additionally, no dose-related effects on reproductive organs were noted in either sex. Neoplastic liver nodules were observed in male Fischer rats fed 12,100 ppm benzotriazole for 78 weeks.However,historic laboratory controls incidences varied from 0 to 11% so the treatment-related effects could not be determined. Brain tumors occurred in three males and one female rat. Incidence of endometrial stromal polyps was increased significantly in female rats fed 6700 ppm for 78 weeks (22%), but not in female rats fed 12,100 ppm (16%). Significant increase in alveolar/bronchiolar carcinomas (18%) was observed female B6C3F1 fed 11,700 ppm benzotriazole for 104 weeks. Comparatively, a.similar increase was not observed in female mice fed 23,500 ppm benzotriazole for the same period of time (6% increase). Historical laboratory control incidences varied from 0 to 7%. Genotoxicity studies indicate that the compound was not mutagenic to S. typhimurium strains TA97, TA98, or TA100 in the presence or absence of S9, or Chinese hamster ovary cells. Benzotriazole was also not mutagenic to S. typhimurium strain TA1535 in the absence of S9, but was mutagenic in the presence of S9.Conflicting results were obtained for effects in S. typhimurium strains TA1537 and TA1538 and E. coli WP2 uvrA. It did not produce DNA damage in E. coli PQ37. In Chinese hamster ovary cells, benzotriazole induced chromosomal aberrations in the presence of S9 and sister chromatid exchange in the absence of S9. Benzotriazole was not genotoxic in the mouse micronucleus assay at 800 mg/kg. Benzotriazole was identified as a non-sensitizer in the guinea pig maximization test. Benzotriazole was identified as irritating to rabbit eyes and minimally irritating to rabbit and guinea pig skin<br>For phenolic benzotriazoles<br>Overall, oral exposure (either through gavage or in feed) of the tested chemicals to rats led to liver effects. Increased absolute and/or relative liver weights were observed in several studies. Body weight and body weight gain changes were observed after administration of several test substances. Histopathological changes (e.g.,foci, hypertrophy, and cytoplasmic vacuolization) and altered liver enzyme content and activities were also noted after treatment with different phenolic benzotriazoles. Haematological effects (e.g., altered white and red blood cell counts, altered albumin levels, and packed cell volume) were observed. For those studies that calculated no observed adverse effect levels (NOAELs), |

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| | the values ranged from <0.5 to ~5685 mg/kg/day<br>**Reproductive and teratology effects**: The chemicals tested produced a variety of effects. Some chemicals were shown to affect reproductive organ weights, but no direct studies in reproduction and development were located.<br>**Genotoxicity** None of the tested compounds were identified as mutagenic in vitro in the absence or presence of a metabolic system (S9) or in vivo<br>Chemical Information Review Document for Phenolic Benzotriazoles: Supporting Nomination for Toxicological Evaluation by the National Toxicology Program October 2011<br>http://ntp.niehs.nih.gov/ntp/noms/support_docs/phenolicbenzotriazoles_cird_oct2011_508.pdf |
| **5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE & 2-METHYL-4-ISOTHIAZOLIN-3-ONE** | (1). Bruze etal - Contact Dermatitis 20: 219-39, 1989<br>Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.<br>Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of<br>appropriate studies with similar materials using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.<br>In light of potential adverse effects, and to ensure a harmonised risk assessment and management, the EU regulatory framework for biocides has been established with the objective of ensuring a high level of protection of human and animal health and the environment. To this aim, it is required that risk assessment of biocidal products is carried out before they can be placed on the market. A central element in the risk assessment of the biocidal products are the utilization instructions that defines the dosage, application method and amount of applications and thus the exposure of humans and the environment to the biocidal substance.<br>Humans may be exposed to biocidal products in different ways in both occupational and domestic settings. Many biocidal products are intended for industrial sectors or professional uses only, whereas other biocidal products are commonly available for private use by non-professional users. In addition, potential exposure of non-users of biocidal products (i.e. the general public) may occur indirectly via the environment, for example through drinking water, the food chain, as well as through atmospheric and residential exposure. Particular attention should be paid to the exposure of vulnerable sub-populations, such as the elderly, pregnant women, and children. Also pets and other domestic animals can be exposed indirectly following the application of biocidal products. Furthermore, exposure to biocides may vary in terms of route (inhalation, dermal contact, and ingestion) and pathway (food, drinking water, residential, occupational) of exposure, level, frequency and duration.<br>The European Union has reclassified several formaldehyde-releasing agents (FRAs) such as methylenedimorpholine (MBM), oxazolidine (MBO) and hydroxypropylamine (HPT) as category 1B carcinogens. Previously, formaldehyde itself was classed as a carcinogen – but formaldehyde-releasing agents were not. This is no longer the case. Based on this regulation, formulations for which the maximum theoretical concentration of releasable formaldehyde is more than > 1000 ppm (>0.1%), have to be labelled as carcinogenic.<br>Water mix metalworking fluids are subject to contamination by bacteria and fungi, and the control of this is an essential part of good fluid maintenance. The use of preservatives both within the formulation and tank-side treatment plays a significant contribution in the protection of potentially harmful microbes that could cause health problems for workers.<br>A large proportion of bactericides on the market today are classed as formaldehyde releasing biocides which means that under specific conditions they release small amounts of formaldehyde – this is their mode of action in the presence of bacteria. Although they are effective as a biocide their use may become restricted or unfavourable due to potential changes in legislation.<br>A decision by the ECHA (European Chemicals Agency) was made to re-classify formaldehyde as a category 1b H350 carcinogen and category 2 mutagen in June 2015.<br>It has also been proposed by the ECHA Risk Assessment Committee (RAC) that formaldehyde release biocides should be classified the same as formaldehyde because formaldehyde is released when these substances come into contact under favorable conditions (i.e. interaction with microorganisms).<br>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.<br>Many countries are placing regulatory pressure on suppliers and users to replace formaldehyde generators.<br>Formaldehyde generators are a diverse group of chemicals that can be recognised by a small, easily detachable formaldehyde moiety, prepared by reacting an amino alcohol with formaldehyde ("formaldehyde-condensates"),<br>There is concern that when formaldehyde-releasing preservatives are present in a formulation that also includes amines, such as triethanolamine (TEA), diethanolamine (DEA), or monoethanolamine (MEA), nitrosamines can be formed,; nitrosamines are carcinogenic substances that can potentially penetrate skin.<br>One widely-discussed hypothesis states that formaldehyde-condensate biocides, such as triazines and oxazolidines, may cause an imbalance in the microbial flora of in-use metalworking fluids (MWFs).The hypothesis further asserts that this putative microbial imbalance favours the proliferation of certain nontuberculosis mycobacteria (NTM) in MWFs and that the subsequent inhalation of NTM-containing aerosols can cause hypersensitivity pneumonitis (HP), also known as extrinsic allergic alveolitis, in a small percentage of susceptible workers. Symptoms of HP include flu-like illness accompanied by chronic dyspnea, i.e., difficult or laboured respiration<br>According to Annex VI of the Cosmetic Directive 76/768/EC, the maximum authorised concentration of free formaldehyde is 0.2% (2000 ppm). In addition, the provisions of Annex VI state that,<br>*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%.*<br>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. |

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

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

## SECTION 12 Ecological information

**Toxicity**

**Continued...**

**Gold Eagle 303 Aerospace Protectant**

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

**polydimethylsiloxane**

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

**2-propylheptanol, ethoxylated**

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

**D-sorbitol**

| Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|
| EC10(ECx) | 168h | Algae or other aquatic plants | 8408.53mg/L | 4 |

**4-nonylphenol, branched, ethoxylated**

| Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|
| EC50 | 72h | Algae or other aquatic plants | 19.485mg/l | 2 |
| EC50 | 48h | Crustacea | 14mg/l | 2 |
| EC50 | 96h | Algae or other aquatic plants | 12mg/l | 2 |
| LC50 | 96h | Fish | >10mg/l | 2 |
| NOEC(ECx) | 96h | Algae or other aquatic plants | 8mg/l | 2 |

**polyethylene glycol**

| Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|
| EC50 | 48h | Crustacea | >100mg/l | 2 |
| EC50 | 96h | Algae or other aquatic plants | >100mg/l | 2 |
| EC50(ECx) | 48h | Crustacea | >100mg/l | 2 |
| LC50 | 96h | Fish | >100mg/l | 2 |

**dinonylphenyl ethoxylate**

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

**CG 20-568 ethoxylated**

| Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|
| EC50 | 48h | Crustacea | 4mg/l | 2 |
| EC50 | 72h | Algae or other aquatic plants | ~9mg/l | 2 |
| LC50 | 96h | Fish | 2.8mg/l | 2 |
| NOEC(ECx) | 72h | Algae or other aquatic plants | 0.11mg/l | 2 |

**di-CG 20-568 ethoxylated**

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

**magnesium nitrate**

| Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|
| EC50(ECx) | 24h | Crustacea | 6075mg/L | 5 |

**5-chloro-2-methyl-4-isothiazolin-3-one**

| Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|
| EC50 | 72h | Algae or other aquatic plants | 0.018-0.026mg/L | 4 |
| EC50 | 48h | Crustacea | 4.71mg/l | 1 |
| EC50 | 96h | Algae or other aquatic plants | 0.03-0.13mg/L | 4 |
| LC50 | 96h | Fish | 0.13-0.31mg/L | 4 |
| NOEC(ECx) | 504h | Crustacea | 0.172mg/l | 1 |

**2-methyl-4-isothiazolin-3-one**

| Endpoint | Test Duration (hr) | Species | Value | Source |
|---|---|---|---|---|
| EC50 | 72h | Algae or other aquatic plants | 0.057mg/L | 2 |
| EC50 | 48h | Crustacea | 0.189-0.257mg/L | 4 |
| EC50 | 96h | Algae or other aquatic plants | 0.061mg/L | 2 |
| LC50 | 96h | Fish | 0.081-0.122mg/L | 4 |
| NOEC(ECx) | 96h | Algae or other aquatic plants | 0.01mg/l | 2 |

**water**

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

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

**Continued...**

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
**DO NOT** discharge into sewer or waterways.

### Persistence and degradability

| Ingredient | Persistence: Water/Soil | Persistence: Air |
|---|---|---|
| D-sorbitol | LOW | LOW |
| polyethylene glycol | LOW | LOW |
| 5-chloro-2-methyl-4-isothiazolin-3-one | HIGH | HIGH |
| 2-methyl-4-isothiazolin-3-one | HIGH | HIGH |
| water | LOW | LOW |

### Bioaccumulative potential

| Ingredient | Bioaccumulation |
|---|---|
| D-sorbitol | LOW (LogKOW = -3.0108) |
| polyethylene glycol | LOW (LogKOW = -1.1996) |
| 5-chloro-2-methyl-4-isothiazolin-3-one | LOW (LogKOW = 0.0444) |
| 2-methyl-4-isothiazolin-3-one | LOW (LogKOW = -0.8767) |

### Mobility in soil

| Ingredient | Mobility |
|---|---|
| D-sorbitol | LOW (KOC = 10) |
| polyethylene glycol | HIGH (KOC = 1) |
| 5-chloro-2-methyl-4-isothiazolin-3-one | LOW (KOC = 45.15) |
| 2-methyl-4-isothiazolin-3-one | LOW (KOC = 27.88) |

## SECTION 13 Disposal considerations

### Waste treatment methods

| | |
|---|---|
| **Product / Packaging disposal** | Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.<br>A Hierarchy of Controls seems to be common - the user should investigate:<br>▸ Reduction<br>▸ Reuse<br>▸ Recycling<br>▸ Disposal (if all else fails)<br>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.<br>▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains.<br>▸ It may be necessary to collect all wash water for treatment before disposal.<br>▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.<br>▸ Where in doubt contact the responsible authority.<br>▸ Recycle wherever possible.<br>▸ 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.<br>▸ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible material).<br>▸ 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

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

| Product name | Group |
|---|---|
| polydimethylsiloxane | Not Available |

**Continued...**

| Product name | Group |
|---|---|
| 2-propylheptanol, ethoxylated | Not Available |
| D-sorbitol | Not Available |
| 4-nonylphenol, branched, ethoxylated | Not Available |
| polyethylene glycol | Not Available |
| dinonylphenyl ethoxylate | Not Available |
| CG 20-568 ethoxylated | Not Available |
| di-CG 20-568 ethoxylated | Not Available |
| magnesium nitrate | Not Available |
| 5-chloro-2-methyl-4-isothiazolin-3-one | Not Available |
| 2-methyl-4-isothiazolin-3-one | Not Available |
| water | Not Available |

**Transport in bulk in accordance with the IGC Code**

| Product name | Ship Type |
|---|---|
| polydimethylsiloxane | Not Available |
| 2-propylheptanol, ethoxylated | Not Available |
| D-sorbitol | Not Available |
| 4-nonylphenol, branched, ethoxylated | Not Available |
| polyethylene glycol | Not Available |
| dinonylphenyl ethoxylate | Not Available |
| CG 20-568 ethoxylated | Not Available |
| di-CG 20-568 ethoxylated | Not Available |
| magnesium nitrate | Not Available |
| 5-chloro-2-methyl-4-isothiazolin-3-one | Not Available |
| 2-methyl-4-isothiazolin-3-one | Not Available |
| water | Not Available |

## SECTION 15 Regulatory information

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

**polydimethylsiloxane is found on the following regulatory lists**

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C
Australian Inventory of Industrial Chemicals (AIIC)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

**2-propylheptanol, ethoxylated is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

**D-sorbitol is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

**4-nonylphenol, branched, ethoxylated is found on the following regulatory lists**

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

**polyethylene glycol is found on the following regulatory lists**

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2
Australian Inventory of Industrial Chemicals (AIIC)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 3

**dinonylphenyl ethoxylate is found on the following regulatory lists**

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

**CG 20-568 ethoxylated is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

**di-CG 20-568 ethoxylated is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

**magnesium nitrate is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2A: Probably carcinogenic to humans
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

**Continued...**

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

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

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

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

**water is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

### National Inventory Status

| National Inventory | Status |
|---|---|
| Australia - AIIC / Australia Non-Industrial Use | Yes |
| Canada - DSL | No (2-propylheptanol, ethoxylated) |
| Canada - NDSL | No (polydimethylsiloxane; 2-propylheptanol, ethoxylated; D-sorbitol; 4-nonylphenol, branched, ethoxylated; polyethylene glycol; dinonylphenyl ethoxylate; CG 20-568 ethoxylated; di-CG 20-568 ethoxylated; magnesium nitrate; 5-chloro-2-methyl-4-isothiazolin-3-one; 2-methyl-4-isothiazolin-3-one; water) |
| China - IECSC | Yes |
| Europe - EINEC / ELINCS / NLP | No (polydimethylsiloxane; 2-propylheptanol, ethoxylated; dinonylphenyl ethoxylate; CG 20-568 ethoxylated; di-CG 20-568 ethoxylated) |
| Japan - ENCS | No (dinonylphenyl ethoxylate; CG 20-568 ethoxylated; di-CG 20-568 ethoxylated) |
| Korea - KECI | Yes |
| New Zealand - NZIoC | Yes |
| Philippines - PICCS | Yes |
| USA - TSCA | Yes |
| Taiwan - TCSI | Yes |
| Mexico - INSQ | No (2-propylheptanol, ethoxylated; polyethylene glycol; dinonylphenyl ethoxylate; CG 20-568 ethoxylated; di-CG 20-568 ethoxylated) |
| Vietnam - NCI | Yes |
| Russia - FBEPH | No (2-propylheptanol, ethoxylated; dinonylphenyl ethoxylate; CG 20-568 ethoxylated; di-CG 20-568 ethoxylated) |
| ***Legend:*** | *Yes = All CAS declared ingredients are on the inventory*<br>*No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.* |

## SECTION 16 Other information

| | |
|---|---|
| **Revision Date** | 30/06/2023 |
| **Initial Date** | 28/07/2016 |

### SDS Version Summary

| Version | Date of Update | Sections Updated |
|---|---|---|
| 2.1 | 28/07/2016 | Toxicological information - Acute Health (eye), Toxicological information - Acute Health (swallowed), Toxicological information - Chronic Health, Composition / information on ingredients - Ingredients, Handling and storage - Storage (storage incompatibility), Identification of the substance / mixture and of the company / undertaking - Synonyms |

### Other information

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

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

### Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
ES: Exposure Standard
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index
AIIC: Australian Inventory of Industrial Chemicals
DSL: Domestic Substances List
NDSL: Non-Domestic Substances List
IECSC: Inventory of Existing Chemical Substance in China

**Continued...**

EINECS: European INventory of Existing Commercial chemical Substances
ELINCS: European List of Notified Chemical Substances
NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory
KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals
PICCS: Philippine Inventory of Chemicals and Chemical Substances
TSCA: Toxic Substances Control Act
TCSI: Taiwan Chemical Substance Inventory
INSQ: Inventario Nacional de Sustancias Químicas
NCI: National Chemical Inventory
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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**end of SDS**