

# Hoover 1.47L Oxy Formula Techtronic Industries Australia

Chemwatch Hazard Alert Code: 2

Chemwatch: 5458-60

Issue Date: 12/03/2021

Version No: 2.1.1.1

Print Date: 30/03/2021

Safety Data Sheet according to WHS and ADG requirements

L.GHS.AUS.EN

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

### Product Identifier

Product name	Hoover 1.47L Oxy Formula
Chemical Name	Not Applicable
Synonyms	Not Available
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Carpet cleaner concentrate. Use according to manufacturer's directions.
--------------------------	--

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

Registered company name	Techtronic Industries Australia
Address	31 Gilby Road Mount Waverley VIC 3149 Australia
Telephone	1300 829 287
Fax	Not Available
Website	<a href="http://www.hoover.com.au/">http://www.hoover.com.au/</a>
Email	customerservice@ttibrands.com.au

### Emergency telephone number


Association / Organisation	Poison Information Centre
Emergency telephone numbers	13 11 26 (24 hours a day, seven days a week, Australia)
Other emergency telephone numbers	0800 764 766 (New Zealand)

## SECTION 2 Hazards identification

### Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification <sup>[1]</sup>	Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

### Label elements

Hazard pictogram(s)	
Signal word	Warning

Hoover 1.47L Oxy Formula

**Hazard statement(s)**

H315	Causes skin irritation.
H319	Causes serious eye irritation.

**Precautionary statement(s) Prevention**

P280	Wear protective gloves/protective clothing/eye protection/face protection/hearing protection/...
------	--

**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.
P337+P313	If eye irritation persists: Get medical advice/attention.
P302+P352	IF ON SKIN: Wash with plenty of water.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

**Precautionary statement(s) Storage**

Not Applicable

**Precautionary statement(s) Disposal**

Not Applicable

**SECTION 3 Composition / information on ingredients**

**Substances**

See section below for composition of Mixtures

**Mixtures**

CAS No	%[weight]	Name
68439-46-3	1-<10	<u>alcohols C9-11 ethoxylated</u>
7722-84-1	<5	<u>hydrogen peroxide</u>
2605-78-9	<3	<u>N,N-dimethyloctylamine N-oxide</u>
Not Available	balance	Ingredients determined not to be hazardous

**SECTION 4 First aid measures**

**Description of first aid measures**

<b>Eye Contact</b>	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> <li>▶ Wash out immediately with fresh running water.</li> <li>▶ 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.</li> <li>▶ Seek medical attention without delay; if pain persists or recurs seek medical attention.</li> <li>▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> </ul>
<b>Skin Contact</b>	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> <li>▶ Immediately remove all contaminated clothing, including footwear.</li> <li>▶ Flush skin and hair with running water (and soap if available).</li> <li>▶ Seek medical attention in event of irritation.</li> </ul>
<b>Inhalation</b>	<ul style="list-style-type: none"> <li>▶ If fumes or combustion products are inhaled remove from contaminated area.</li> <li>▶ Lay patient down. Keep warm and rested.</li> <li>▶ Prosthesis such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.</li> <li>▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.</li> <li>▶ Transport to hospital, or doctor.</li> </ul>
<b>Ingestion</b>	<ul style="list-style-type: none"> <li>▶ <b>If swallowed do NOT induce vomiting.</b></li> <li>▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.</li> <li>▶ Observe the patient carefully.</li> <li>▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.</li> <li>▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.</li> <li>▶ Seek medical advice.</li> </ul>

Hoover 1.47L Oxy Formula

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

Hydrogen peroxide at moderate concentrations (5% or more) is a strong oxidant.

- Direct contact with the eye is likely to cause corneal damage especially if not washed immediately. Careful ophthalmologic evaluation is recommended and the possibility of local corticosteroid therapy should be considered.
- Because of the likelihood of systemic effects attempts at evacuating the stomach via emesis induction or gastric lavage should be avoided.
- There is remote possibility, however, that a nasogastric or orogastric tube may be required for the reduction of severe distension due to gas formation"

Fisher Scientific SDS

**SECTION 5 Firefighting measures**

**Extinguishing media**

For hydrogen peroxide

NOTE: Chemical extinguishing agents may accelerate decomposition. [CCINFO]

- Water spray or fog.
- Foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.

**Special hazards arising from the substrate or mixture**

<b>Fire Incompatibility</b>	<ul style="list-style-type: none"> <li>▸ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result</li> </ul>
-----------------------------	--

**Advice for firefighters**

<b>Fire Fighting</b>	<ul style="list-style-type: none"> <li>▸ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▸ Wear full body protective clothing with breathing apparatus.</li> <li>▸ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▸ Use water delivered as a fine spray to control fire and cool adjacent area.</li> <li>▸ Avoid spraying water onto liquid pools.</li> <li>▸ <b>DO NOT</b> approach containers suspected to be hot.</li> <li>▸ Cool fire exposed containers with water spray from a protected location.</li> <li>▸ If safe to do so, remove containers from path of fire.</li> </ul>
<b>Fire/Explosion Hazard</b>	<ul style="list-style-type: none"> <li>▸ Combustible.</li> <li>▸ Slight fire hazard when exposed to heat or flame.</li> <li>▸ Heating may cause expansion or decomposition leading to violent rupture of containers.</li> <li>▸ On combustion, may emit toxic fumes of carbon monoxide (CO).</li> <li>▸ May emit acrid smoke.</li> <li>▸ Mists containing combustible materials may be explosive.</li> </ul> <p>Combustion products include: carbon dioxide (CO<sub>2</sub>) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.</p>
<b>HAZCHEM</b>	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**

<b>Minor Spills</b>	<ul style="list-style-type: none"> <li>▸ Remove all ignition sources.</li> <li>▸ Clean up all spills immediately.</li> <li>▸ Avoid breathing vapours and contact with skin and eyes.</li> <li>▸ Control personal contact with the substance, by using protective equipment.</li> <li>▸ Contain and absorb spill with sand, earth, inert material or vermiculite.</li> <li>▸ Wipe up.</li> <li>▸ Place in a suitable, labelled container for waste disposal.</li> </ul>
---------------------	--

Hoover 1.47L Oxy Formula

<b>Major Spills</b>	<p>Moderate hazard.</p> <ul style="list-style-type: none"> <li>▶ Clear area of personnel and move upwind.</li> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ No smoking, naked lights or ignition sources.</li> <li>▶ Increase ventilation.</li> <li>▶ Stop leak if safe to do so.</li> <li>▶ Contain spill with sand, earth or vermiculite.</li> <li>▶ Collect recoverable product into labelled containers for recycling.</li> <li>▶ Absorb remaining product with sand, earth or vermiculite.</li> <li>▶ Collect solid residues and seal in labelled drums for disposal.</li> <li>▶ Wash area and prevent runoff into drains.</li> <li>▶ If contamination of drains or waterways occurs, advise emergency services.</li> </ul>
---------------------	---

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

**SECTION 7 Handling and storage**

**Precautions for safe handling**

<b>Safe handling</b>	<ul style="list-style-type: none"> <li>▶ <b>DO NOT allow clothing wet with material to stay in contact with skin</b></li> <li>▶ Avoid all personal contact, including inhalation.</li> <li>▶ Wear protective clothing when risk of exposure occurs.</li> <li>▶ Use in a well-ventilated area.</li> <li>▶ Prevent concentration in hollows and sumps.</li> <li>▶ <b>DO NOT enter confined spaces until atmosphere has been checked.</b></li> <li>▶ Avoid smoking, naked lights or ignition sources.</li> <li>▶ Avoid contact with incompatible materials.</li> <li>▶ When handling, <b>DO NOT eat, drink or smoke.</b></li> <li>▶ Keep containers securely sealed when not in use.</li> <li>▶ Avoid physical damage to containers.</li> <li>▶ Always wash hands with soap and water after handling.</li> <li>▶ Work clothes should be laundered separately.</li> <li>▶ Use good occupational work practice.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.</li> </ul>
<b>Other information</b>	<ul style="list-style-type: none"> <li>▶ Store in original containers.</li> <li>▶ Keep containers securely sealed.</li> <li>▶ No smoking, naked lights or ignition sources.</li> <li>▶ Store in a cool, dry, well-ventilated area.</li> <li>▶ Store away from incompatible materials and foodstuff containers.</li> <li>▶ Protect containers against physical damage and check regularly for leaks.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> </ul>

**Conditions for safe storage, including any incompatibilities**

<b>Suitable container</b>	<ul style="list-style-type: none"> <li>▶ Polyethylene or polypropylene container.</li> <li>▶ Packing as recommended by manufacturer.</li> <li>▶ Check all containers are clearly labelled and free from leaks.</li> </ul>
<b>Storage incompatibility</b>	<ul style="list-style-type: none"> <li>▶ Avoid reaction with oxidising agents, bases and strong reducing agents.</li> <li>▶ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.</li> </ul>

**SECTION 8 Exposure controls / personal protection**

**Control parameters**

**Occupational Exposure Limits (OEL)**

**INGREDIENT DATA**

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	hydrogen peroxide	Hydrogen peroxide	1 ppm / 1.4 mg/m3	Not Available	Not Available	Not Available

**Emergency Limits**

Ingredient	TEEL-1	TEEL-2	TEEL-3
hydrogen peroxide	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
alcohols C9-11 ethoxylated	Not Available	Not Available
hydrogen peroxide	75 ppm	Not Available
N,N-dimethyloctylamine N-oxide	Not Available	Not Available

### Occupational Exposure Banding


Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
alcohols C9-11 ethoxylated	E	≤ 0.1 ppm
N,N-dimethyloctylamine N-oxide	E	≤ 0.01 mg/m <sup>3</sup>

#### 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	<p>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.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>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.</p> <p>Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.</p> <p>An approved self contained breathing apparatus (SCBA) may be required in some situations.</p> <p>Provide adequate ventilation in warehouse or closed storage area. 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.</p>											
	<table border="1"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td> <td>0.25-0.5 m/s (50-100 f/min.)</td> </tr> <tr> <td>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)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table>	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.)	
	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.)											
<p>Within each range the appropriate value depends on:</p> <table border="1"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table>	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		
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											
<p>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.</p>												
Personal protection												

Hoover 1.47L Oxy Formula

<p><b>Eye and face protection</b></p>	<ul style="list-style-type: none"> <li>▶ Safety glasses with side shields.</li> <li>▶ Chemical goggles.</li> <li>▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]</li> </ul>
<p><b>Skin protection</b></p>	<p>See Hand protection below</p>
<p><b>Hands/feet protection</b></p>	<ul style="list-style-type: none"> <li>▶ Wear chemical protective gloves, e.g. PVC.</li> <li>▶ Wear safety footwear or safety gumboots, e.g. Rubber</li> </ul> <p><b>NOTE:</b></p> <ul style="list-style-type: none"> <li>▶ 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.</li> <li>▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.</li> </ul> <p>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.</p> <p>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.</p> <p>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.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> <li>- frequency and duration of contact,</li> <li>- chemical resistance of glove material,</li> <li>- glove thickness and</li> <li>- dexterity</li> </ul> <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> <li>- 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.</li> <li>- 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.</li> <li>- Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.</li> <li>- Contaminated gloves should be replaced.</li> </ul> <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> <li>- Excellent when breakthrough time &gt; 480 min</li> <li>- Good when breakthrough time &gt; 20 min</li> <li>- Fair when breakthrough time &lt; 20 min</li> <li>- Poor when glove material degrades</li> </ul> <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>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.</p> <p>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.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> <li>- 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.</li> <li>- 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</li> </ul> <p>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.</p>
<p><b>Body protection</b></p>	<p>See Other protection below</p>
<p><b>Other protection</b></p>	<ul style="list-style-type: none"> <li>▶ Overalls.</li> <li>▶ P.V.C apron.</li> <li>▶ Barrier cream.</li> <li>▶ Skin cleansing cream.</li> <li>▶ Eye wash unit.</li> </ul>

**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:  
Hoover 1.47L Oxy Formula

**Respiratory protection**

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

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

Material	CPI
NAT+NEOPR+NITRILE	A
NATURAL RUBBER	A
NATURAL+NEOPRENE	A
NEOPRENE	A
NEOPRENE/NATURAL	A
NITRILE	A
PVC	A

\* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	AB-AUS P2	-	AB-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AB-AUS / Class 1 P2	-
up to 100 x ES	-	AB-2 P2	AB-PAPR-2 P2 ^

^ - Full-face

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

- ▶ Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- ▶ The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- ▶ Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

## SECTION 9 Physical and chemical properties

### Information on basic physical and chemical properties

<b>Appearance</b>	Clear liquid; mixes with water.		
<b>Physical state</b>	Liquid	<b>Relative density (Agua= 1)</b>	1.015
<b>Odour</b>	Not Available	<b>Partition coefficient n-octanol / water</b>	Not Available
<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	Not Available
<b>pH (as supplied)</b>	4-6	<b>Decomposition temperature</b>	Not Available
<b>Melting point / freezing point (°C)</b>	Not Available	<b>Viscosity (cSt)</b>	Not Available
<b>Initial boiling point and boiling range (°C)</b>	100	<b>Molecular weight (g/mol)</b>	Not Applicable
<b>Flash point (°C)</b>	Not Available	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	<1 (Ether = 1)	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	Not Available	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	Not Available	<b>Surface Tension (dyn/cm or mN/m)</b>	Not Available
<b>Lower Explosive Limit (%)</b>	Not Available	<b>Volatile Component (%vol)</b>	Not Available
<b>Vapour pressure (kPa)</b>	Not Available	<b>Gas group</b>	Not Available
<b>Solubility in water</b>	Miscible	<b>pH as a solution (1%)</b>	Not Available
<b>Vapour density (Air = 1)</b>	>1	<b>VOC g/L</b>	Not Available

## SECTION 10 Stability and reactivity

<b>Reactivity</b>	See section 7
<b>Chemical stability</b>	<ul style="list-style-type: none"> <li>▶ Unstable in the presence of incompatible materials.</li> <li>▶ Product is considered stable.</li> <li>▶ Hazardous polymerisation will not occur.</li> </ul>

<b>Possibility of hazardous reactions</b>	See section 7
<b>Conditions to avoid</b>	See section 7
<b>Incompatible materials</b>	See section 7
<b>Hazardous decomposition products</b>	See section 5

## SECTION 11 Toxicological information

### Information on toxicological effects

<b>Inhaled</b>	<p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Inhalation of excessive levels of mist may result in headache, dizziness, vomiting, diarrhoea, irritability, insomnia and in extreme pulmonary oedema. Systemic poisoning due to hydrogen peroxide inhalation may cause tremors and numbness of the extremities, convulsions, pulmonary oedema, coma and shock. Hydrogen peroxide has poor warning properties. High concentrations of the vapour or mist are likely to cause extreme irritation of the nose and chest, cough, discomfort, shortness of breath, and inflammation of the nose and throat.</p>
<b>Ingestion</b>	<p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>Hydrogen peroxide may cause blistering and bleeding from the throat and stomach. Ingested hydrogen peroxide may evolve large quantities of oxygen which could hyper-distend the gastro-intestinal tract and may cause internal bleeding. Ingestion of large amounts of hydrogen peroxide causes chest and stomach pain, loss of consciousness, and motor disorders in humans and has caused mortality in experimental animals.</p> <p>Ingestion of hydrogen peroxide containing/ generating materials may cause nausea, vomiting and, possibly, internal bleeding. Rapid evolution of oxygen in the acid environment of the stomach (up to 10 times the volume of the ingested solution) may result in severe organ damage. Large doses are presumed to produce gastritis and oesophagitis. Cases of rupture of the colon, proctitis and ulcerative colitis have been reported following hydrogen peroxide enemas. Powders and tablets that generate hydrogen peroxide, such as Oxone (KHSO<sub>5</sub>), have caused oral and oesophageal burns when ingested.</p> <p>Large amounts of hydrogen peroxide taken orally by humans has caused apnea, dizziness, headache, tremors weakness or numbness in the extremities, convulsions, loss of consciousness and shock.</p> <p>Hydrogen peroxide concentrate is caustic and should not be tasted undiluted. Rats receiving 2.5% hydrogen peroxide (equivalent to approximately 3.5 g/kg/day) in their drinking water died within 43 days. Cases of rupture of the colon, inflammation of the anus or rectum, and ulcerative colitis have been reported following hydrogen peroxide enemas</p> <p>In five persons who accidentally drank about 50 mL of a 33% hydrogen peroxide solution, symptoms included stomach and chest pain, retention of breath, foaming at the mouth, and loss of consciousness. Later, motor and sensory disorders, fever, microhaemorrhages and moderate leucocytosis were noted. All recovered completely within 2-3 weeks.</p>
<b>Skin Contact</b>	<p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p> <p>Hydrogen peroxide as a topical gel is used to cleanse minor wounds or minor gum inflammation. In appropriate solution, hydrogen peroxide is used in topical and dental gels. Skin contact may cause bleaching, blistering and reddening. Short term contact results in temporary whitening and a tingling sensation due to diffusion of hydrogen peroxide into the skin. The characteristic whitening of the skin occurs after topical application of hydrogen peroxide (1-30%), which is believed the result of avascularity of the skin produced by oxygen bubbles acting microembolically in the capillaries. Concentrations above 50% are corrosive to skin.</p>
<b>Eye</b>	<p>Corneal ulcerations due to hydrogen peroxide exposure may not appear for up to a week after exposure; concentrations above 10% are corrosive to the eye.</p> <p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals.</p> <p>Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>
<b>Chronic</b>	<p>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.</p> <p>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.</p>



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.

On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

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.

Prolonged or repeated skin contact may cause degreasing with drying, cracking and dermatitis following.

Hydrogen peroxide as a human food additive is generally regarded as safe when used in certain limitations. In experimental animals, oral administration of hydrogen peroxide causes dental, liver, kidney, stomach, and intestinal damage. Inhalation exposure to hydrogen peroxide caused skin irritation and sneezing in dogs, and high mortality in mice.

Hydrogen peroxide added to food is affirmed to be generally regarded as safe (GRAS) by the U.S. FDA when used to treat certain foods in specified limitations [FDA 21 CFR 184.1366 (4/1/93)]. Hydrogen peroxide may be used as a component of articles for use in packaging, handling, transporting, or holding food in accordance with prescribed conditions [FDA 21 CFR 175.105 (4/1/93)].

Dose-related growth retardation, induction of dental caries, and pathological changes in the periodontium were observed in young male rats receiving 1.5% hydrogen peroxide as their drinking fluid (equivalent to approximately 2.1 g/kg/day)<sup>2</sup> for 8 weeks .

Effects observed in mice treated for 35 weeks with 0.15% hydrogen peroxide as their drinking fluid (equivalent to approximately 0.29 g/kg/day)<sup>3</sup> included degeneration of hepatic and renal tubular epithelial tissues, necrosis, inflammation, irregularities of tissue structure of the stomach wall, and hypertrophy of the small intestine wall. Concentrations in excess of 1% (equivalent to approximately 1.9 g/kg/day)<sup>4</sup> resulted in pronounced weight loss and death within two weeks. In a sequential study of mice treated with 0.4% hydrogen peroxide in drinking water (equivalent to approximately 0.76 g/kg/day)<sup>5</sup>, gastric erosion was observed at 30 days and was present consistently throughout the 108 week study period.

Dogs exposed 6 hours/day, 5 days/week for 6 months at an average vapour concentration of 7 ppm (9.73 mg/3 ) of 90% hydrogen peroxide, developed skin irritation, sneezing, lacrimation, and bleaching of the hair. Autopsy disclosed pulmonary irritation and greatly thickened skin, but no hair follicle destruction. No significant changes in blood or urinary parameters were observed .

Following eight 6-hour exposures to hydrogen peroxide at a concentration of 79 mg/m<sup>3</sup> (56.88 ppm), 7/9 mice died. Following exposure to hydrogen peroxide at 93 mg/m<sup>3</sup>, 6 hours/day, 5 days/week for 30 exposures, 1/10 rats died

Hoover 1.47L Oxy Formula	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
alcohols C9-11 ethoxylated	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >2000 mg/kg <sup>[2]</sup>	Eye (human): SEVERE
	Inhalation(Rat) LC50; >1.6 mg/l <sup>[1]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
	Oral(Rat) LD50; >5050 mg/kg <sup>[1]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
		Skin: SEVERE
hydrogen peroxide	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >2000 mg/kg <sup>[1]</sup>	Not Available
	Inhalation(Rat) LC50; 2 mg/l <sup>[2]</sup>	
	Oral(Rat) LD50; >225 mg/kg <sup>[2]</sup>	
N,N-dimethyloctylamine N-oxide	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral(Rat) LD50; >5000 mg/kg <sup>[1]</sup>	Not Available
<b>Legend:</b>	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	

#### ALCOHOLS C9-11 ETHOXYLATED

Somnolence, ataxia, diarrhoea recorded.

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaosaheptacosan-1-ol ) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture .

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.

Allergic Contact Dermatitis—Formation, Structural Requirements, and Reactivity of Skin Sensitizers.

Ann-Therese Karlberg et al; Chem. Res. Toxicol. 2008, 21, 53-69

Polyethylene glycols (PEGs) have a wide variety of PEG-derived mixtures due to their readily linkable terminal primary hydroxyl groups in combination with many possible compounds and complexes such as ethers, fatty acids, castor oils, amines, propylene glycols, among other derivatives. PEGs and their derivatives are broadly utilized in cosmetic products as surfactants, emulsifiers, cleansing agents, humectants, and skin conditioners.

PEGs and PEG derivatives were generally regulated as safe for use in cosmetics, with the conditions that impurities and by-products, such as ethylene oxides and 1,4-dioxane, which are known carcinogenic materials, should be removed before they are mixed in cosmetic formulations.

Most PEGs are commonly available commercially as mixtures of different oligomer sizes in broadly- or narrowly-defined molecular weight (MW) ranges. For instance, PEG-10,000 typically designates a mixture of PEG molecules ( $n = 195$  to 265) having an average MW of 10,000. PEG is also known as polyethylene oxide (PEO) or polyoxyethylene (POE), with the three names being chemical synonyms. However, PEGs mainly refer to oligomers and polymers with molecular masses below 20,000 g/mol, while PEOs are polymers with molecular masses above 20,000 g/mol, and POEs are polymers of any molecular mass. Relatively small molecular weight PEGs are produced by the chemical reaction between ethylene oxide and water or ethylene glycol (or other ethylene glycol oligomers), as catalyzed by acidic or basic catalysts. To produce PEO or high-molecular weight PEGs, synthesis is performed by suspension polymerization. It is necessary to hold the growing polymer chain in solution during the course of the poly-condensation process. The reaction is catalyzed by magnesium-, aluminum-, or calcium-organoelement compounds. To prevent coagulation of polymer chains in the solution, chelating additives such as dimethylglyoxime are used.

Safety Evaluation of Polyethylene 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>

Human beings have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents, and other cleaning products. Exposure to these chemicals can occur through ingestion, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that volumes well above a reasonable intake level would have to occur to produce any toxic response. Moreover, no fatal case of poisoning with alcohol ethoxylates has ever been reported. Multiple studies investigating the acute toxicity of alcohol ethoxylates have shown that the use of these compounds is of low concern in terms of oral and dermal toxicity.

Clinical animal studies indicate these chemicals may produce gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhea, and lethargy. Similarly, slight to severe irritation of the skin or eye was generated when undiluted alcohol ethoxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotoxin, carcinogen, or mutagen (HERA 2007). No information was available on levels at which these effects might occur, though toxicity is thought to be substantially lower than that of nonylphenol ethoxylates.

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture.

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.

Alcohol ethoxylates are according to CESIO (2000) classified as Irritant or Harmful depending on the number of EO-units:

EO < 5 gives Irritant (Xi) with R38 (Irritating to skin) and R41 (Risk of serious damage to eyes)

EO > 5-15 gives Harmful (Xn) with R22 (Harmful if swallowed) - R38/41

EO > 15-20 gives Harmful (Xn) with R22-41

>20 EO is not classified (CESIO 2000)

Oxo-AE, C13 EO10 and C13 EO15, are Irritating (Xi) with R36/38 (Irritating to eyes and skin).

AE are not included in Annex 1 of the list of dangerous substances of the Council Directive 67/548/EEC

In general, alcohol ethoxylates (AE) are readily absorbed through the skin of guinea pigs and rats and through the gastrointestinal mucosa of rats. AE are quickly eliminated from the body through the urine, faeces, and expired air (CO<sub>2</sub>). 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<sub>2</sub>). The metabolism of C12 AE yields PEG, carboxylic acids, and CO<sub>2</sub> as metabolites. The LD<sub>50</sub> values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.

The ability of nonionic surfactants to cause a swelling of the stratum corneum of guinea pig skin has been studied. The swelling mechanism of the skin involves a combination of ionic binding of the hydrophilic group as well as hydrophobic interactions of the alkyl chain with the substrate. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants. A substantial amount of toxicological data and information in vivo and in vitro demonstrates that there is no evidence for alcohol ethoxylates (AEs) being genotoxic, mutagenic or carcinogenic. No adverse reproductive or developmental effects were observed. The majority of available toxicity studies revealed NOAELs in excess of 100 mg/kg bw/d but the lowest NOAEL for an individual AE was established to be 50 mg/kg

bw/day. This value was subsequently considered as a conservative, representative value in the risk assessment of AE. The effects were restricted to changes in organ weights with no histopathological organ changes with the exception of liver hypertrophy (indicative of an adaptive response to metabolism rather than a toxic effect). It is noteworthy that there was practically no difference in the NOAEL in oral studies of 90-day or 2 years of duration in rats. A comparison of the aggregate consumer exposure and the systemic NOAEL (taking into account an oral absorption value of 75%) results in a Margin of Exposure of 5,800. Taking into account the conservatism in the exposure assessment and the assigned systemic NOAEL, this margin of exposure is considered more than adequate to account for the inherent uncertainty and variability of the hazard database and inter and intra-species extrapolations.

AEs are not contact sensitizers. Neat AE are irritating to eyes and skin. The irritation potential of aqueous solutions of AEs depends on concentrations. Local dermal effects due to direct or indirect skin contact in certain use scenarios where the products are diluted are not of concern as AEs are not expected to be irritating to the skin at in-use concentrations. Potential irritation of the respiratory tract is not a concern given the very low levels of airborne AE generated as a consequence of spray cleaner aerosols or laundry powder detergent dust.

In summary, the human health risk assessment has demonstrated that the use of AE in household laundry and cleaning detergents is safe and does not cause concern with regard to consumer use.

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/cm<sup>2</sup>/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/cm<sup>2</sup>/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 than 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. TGME 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.

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

The material may produce severe 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) thickening of the epidermis.

Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

For hydrogen peroxide:

Hazard increases with peroxide concentration, high concentrations contain an additive stabiliser.

#### Pharmacokinetics

Hydrogen peroxide is a normal product of metabolism. It is readily decomposed by catalase in normal cells. In experimental animals exposed to hydrogen peroxide, target organs affected include the lungs, intestine, thymus, liver, and kidney, suggesting its distribution to those sites.

Hydrogen peroxide has been detected in breath.

- ▶ Absorption: Hydrogen peroxide is decomposed in the bowel before absorption. When applied to tissue, solutions of hydrogen peroxide have poor penetrability.
- ▶ Distribution: Hydrogen peroxide is produced metabolically in intact cells and tissues. It is formed by reduction of oxygen either directly in a two-electron transfer reaction, often catalysed by flavoproteins, or by an initial one-electron step to O<sub>2</sub> followed by dismutation to hydrogen peroxide.
- ▶ Hydrogen peroxide has been detected in serum and in intact liver. based on the results of toxicity studies, the lungs, intestine, thymus, liver, and kidney may be distribution sites. In rabbits and cats that died after intravenous administration of hydrogen peroxide, the lungs were pale and emphysematous. Following intraperitoneal injection of hydrogen peroxide in mice, pyknotic nuclei were induced in the intestine and thymus (IARC 1985). Degeneration of hepatic and renal tubular epithelial tissue was observed following oral administration of hydrogen peroxide to mice.
- ▶ Metabolism: Glutathione peroxidase, responsible for decomposing hydrogen peroxide, is present in normal human tissues (IARC 1985). When hydrogen peroxide comes in contact with catalase, an enzyme found in blood and most tissues, it rapidly decomposes into oxygen and water.
- ▶ Excretion: Hydrogen peroxide has been detected in human breath at levels ranging from 1.0+/-0.5 g/L to 0.34+/-0.17 g/L.

#### Carcinogenicity

Gastric and duodenal lesions including adenomas, carcinomas, and adenocarcinomas have been observed in mice treated orally with hydrogen peroxide. Marked strain differences in the incidence of tumors have been observed. Papilloma development has been observed in mice treated by dermal application.

#### Genotoxicity

Hydrogen peroxide induced DNA damage, sister chromatid exchanges and chromosomal aberrations in mammalian cells *in vitro*. Hydrogen peroxide induced DNA damage in bacteria (*E. coli*), and was mutagenic to bacteria (*Salmonella typhimurium*) and the fungi, *Neurospora crassa* and *Aspergillus chevallieri*, but not to *Streptomyces griseoflavus*. It was not mutagenic to *Drosophila melanogaster* or to mammalian cells *in vitro*.

#### Developmental Toxicity

Malformations have been observed in chicken embryos treated with hydrogen peroxide, but experiments with mice and rats have been negative.

Female rats that received 0.45% hydrogen peroxide (equivalent to approximately 630 mg/kg/day) as the sole drinking fluid for five weeks produced normal litters when mated with untreated males.

Doses of 1.4 to 11 mol/egg hydrogen peroxide (purity 30%) dissolved in water were injected into the airspace of groups of 20-30 white leghorn chicken eggs on day 3 of incubation.

Embryos were examined on day 14. The incidence of embryonic deaths and malformations was dose-related and detected at doses of 2.8 mol/egg and above. The combined ED50 was 2.7 mol/egg.

#### Reproductive Toxicity

A 1% solution of hydrogen peroxide (equivalent to 1900 mg/kg/day) given as the sole drinking fluid to three-month-old male mice for 7-28 days did not cause infertility.

## HYDROGEN PEROXIDE

	<p>The substance is classified by IARC as Group 3:  <b>NOT</b> classifiable as to its carcinogenicity to humans.  Evidence of carcinogenicity may be inadequate or limited in animal testing.</p>		
<p><b>N,N-DIMETHYLOCTYLAMINE N-OXIDE</b></p>	<p>For amine oxides (AOs):  Substantial data exist for mammalian toxicity by <i>in vitro</i> and <i>in vivo</i> testing. Amine oxides are produced, and transported in aqueous solutions that are 25-35% concentration and most tests were conducted with aqueous solutions in that concentration range. Sometimes aqueous formulations were tested where the AO was at lesser concentrations than 25-35%. Whatever concentration were tested, results are reported below for the active ingredient, amine oxide, in mg AO/kg bw for dermal and oral acute toxicity results and mg AO/kg bw/day for repeated dose studies.  Toxicokinetic and metabolism studies indicate AOs are extensively metabolised and readily excreted after oral administration. Amine oxide was readily absorbed dermally by rats, mice and rabbits after 24 to 72 hours of exposure. After 8 hours of dermal exposure, humans absorbed &lt;1%.</p> <p><b>Acute toxicity:</b> In rat oral acute toxicity limit tests, no deaths occurred at single doses of 600 mg C10-16 AO/kg bw or less (for CAS No 70592-80-2). In multi-dose studies, acute oral LD50 values for rats ranged from 846 mg AO/kg bw to 3873 mg AO/kg bw (both values for CAS No 61788-90-7), with several other AOs having rat oral LD50s falling within this range. In single dose acute dermal toxicity limit tests, no deaths occurred at a dose of 520 mg AO/kg bw (CAS No 70592-80-2). This dose was equivalent to 2 mL/kg of a 30% formulation. There were no deaths observed in a rat acute inhalation study to aerosol droplets of a consumer product providing a dose of 0.016 mg AO/L.</p> <p>In a series of studies on rabbits, AOs of varying chain length showed consistent results and all</p> <ul style="list-style-type: none"> <li>▸ were not irritating to the skin or eyes at low concentrations (1%),</li> <li>▸ were moderately irritating at 5%, and</li> <li>▸ more severely irritating when tested as produced (e.g., ~30% aqueous solutions).</li> </ul> <p>In studies that included rinsing, eye irritation effects diminished with rinsing after 30 seconds of exposure and were slight with rinsing after 4 seconds of exposure. In Draize rabbit eye irritation tests using ~30% AO solutions, rabbits experienced severe to moderate irritation. (The maximum concentration of AO is 10% active in consumer products.) Accidental eye exposure in manufacturing employee incidents and consumer incidents established that eye irritation effects of exposure during manufacturing and use of products containing AO and other surfactants are moderate, transient and reversible  There is no indication of skin sensitisation for the AO category based on the available animal and human data.</p> <p><b>Repeat dose toxicity:</b> In four repeated-dose studies with rats and mice exposed to AO via oral and dermal routes (all with CAS No 70592-80-2), three dermal studies were designed to assess the effect of repeated exposure on skin at maximum doses of 1.5 mg AO/kg-bw/day. Higher doses were tested in a 90-day dietary study with rabbits. No treatment related clinical chemistry, hematology and histopathological changes were observed. In these studies, LOAELs ranged from 87 to 150 mg AO/kg bw/day with the highest oral NOAEL below the lowest LOAEL as 80 mg AO/kg bw/day. Signs of toxicity observed in the oral study included suppressed mean body weight gain, lenticular opacities and diarrhea; in the dermal studies, local dermal irritation was evident.</p> <p><b>Genetic toxicity:</b> In five <i>in vitro</i> bacterial (<i>Salmonella</i>) mutagenicity studies, AO shows no evidence of mutagenicity either with or without S9 metabolic activation at concentrations up to 250 ug/plate (higher concentrations caused cytotoxicity).  Three <i>in vivo</i> studies investigated clastogenic effects on a close structural analog of the category, 1-(methyl dodecyl)dimethylamine-N-oxide including: a mouse micronucleus, a Chinese hamster micronucleus and a Chinese hamster cytogenetics study. These studies were all negative showing no increase in micronuclei or chromosome aberrations.  An <i>in vivo</i> mouse dominant lethal assay showed no evidence of heritable effects. Two AOs (CAS No 1643-20-5 and CAS No 3332-27-2) were negative in an <i>in vitro</i> cell transformation assay tested at concentrations up to 20 ug/ml.</p> <p><b>Carcinogenicity:</b> The carcinogenic potential of amine oxides has been thoroughly investigated in three carcinogenicity studies in rats or mice by dermal, dietary, or drinking water routes. In all cases the substances demonstrated no evidence of a carcinogenic response.</p> <p><b>Reproductive and developmental toxicity:</b> No evidence of reproductive toxicity or fertility effects was observed in a study in which rats were given dietary doses of AO in the diet over two generations (CAS No 1643-20-5). No macroscopic or histopathological changes were attributable to treatment with the test substance. The maternal NOAEL from this reproductive study was &gt;40 mg AO/kg bw/day, which was the highest dose tested. At all treatment levels, the rate of bodyweight gain for the F1 and F2 offspring was reduced during the lactation period, however, this reduction was not greater than 10%. This effect appeared to be dose-related, but was not statistically significant until after weaning in the mid and high dose levels. This was not considered an adverse effect since the body weight change only reached statistical significance when the rat pups were getting the majority of their calories from solid food (Developmental NOAEL &gt;40 mg/kg bw/day).  In three developmental toxicity studies via gavage in rats and rabbits (with CAS No 1643-20-5 &amp; 70592-80-2), effects such as decreased foetal weight or delayed ossification, were most often observed only at maternally toxic doses and were associated with the irritation effects of AO on the gastrointestinal tract. No decreases in litter size, no changes in litter parameters, no malformations or significant differences in skeletal defects were observed at oral doses up to 25 mg/kg bw/day in rats (based on decreased foetal weight at 100 mg/kg bw/day) and &gt;160 mg/kg bw/day in rabbits (the highest dose tested).</p>		
<p><b>HYDROGEN PEROXIDE &amp; N,N-DIMETHYLOCTYLAMINE N-OXIDE</b></p>	<p>No significant acute toxicological data identified in literature search.</p>		
<p><b>Acute Toxicity</b></p>	<p>✘</p>		<p><b>Carcinogenicity</b></p> <p>✘</p>
<p><b>Skin Irritation/Corrosion</b></p>	<p>✔</p>		<p><b>Reproductivity</b></p> <p>✘</p>
<p><b>Serious Eye Damage/Irritation</b></p>	<p>✔</p>		<p><b>STOT - Single Exposure</b></p> <p>✘</p>
<p><b>Respiratory or Skin sensitisation</b></p>	<p>✘</p>		<p><b>STOT - Repeated Exposure</b></p> <p>✘</p>
<p><b>Mutagenicity</b></p>	<p>✘</p>		<p><b>Aspiration Hazard</b></p> <p>✘</p>

Hoover 1.47L Oxy Formula

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

**SECTION 12 Ecological information**

**Toxicity**

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
Hoover 1.47L Oxy Formula	Not Available	Not Available	Not Available	Not Available	Not Available
alcohols C9-11 ethoxylated	LC50	96	Fish	5-7mg/l	2
	EC50	48	Crustacea	2.5mg/l	2
	NOEC(ECx)	720	Fish	0.11-0.28mg/l	2
	EC50	96	Algae or other aquatic plants	1.4mg/l	2
hydrogen peroxide	EC50(ECx)	48	Crustacea	0.029-0.039mg/L	4
	LC50	96	Fish	0.078-0.25mg/L	4
	EC50	48	Crustacea	0.029-0.039mg/L	4
	EC50	72	Algae or other aquatic plants	0.69mg/l	4
	EC50	96	Algae or other aquatic plants	2.27mg/l	4
N,N-dimethyloctylamine N-oxide	EC50	48	Crustacea	130mg/l	2
	LC50	96	Fish	>100mg/l	2
	EC10(ECx)	72	Algae or other aquatic plants	10mg/l	2
	EC50	72	Algae or other aquatic plants	20mg/l	2

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

**DO NOT** discharge into sewer or waterways.

**Persistence and degradability**

Ingredient	Persistence: Water/Soil	Persistence: Air
hydrogen peroxide	LOW	LOW
N,N-dimethyloctylamine N-oxide	LOW	LOW

**Bioaccumulative potential**

Ingredient	Bioaccumulation
hydrogen peroxide	LOW (LogKOW = -1.571)
N,N-dimethyloctylamine N-oxide	LOW (LogKOW = 2.7086)

**Mobility in soil**

Ingredient	Mobility
hydrogen peroxide	LOW (KOC = 14.3)
N,N-dimethyloctylamine N-oxide	LOW (KOC = 1613)

## SECTION 13 Disposal considerations

### Waste treatment methods

<b>Product / Packaging disposal</b>	<p>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.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> <li>▸ Reduction</li> <li>▸ Reuse</li> <li>▸ Recycling</li> <li>▸ Disposal (if all else fails)</li> </ul> <p>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.</p> <ul style="list-style-type: none"> <li>▸ <b>DO NOT allow wash water from cleaning or process equipment to enter drains.</b></li> <li>▸ It may be necessary to collect all wash water for treatment before disposal.</li> <li>▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.</li> <li>▸ Where in doubt contact the responsible authority.</li> <li>▸ Recycle wherever possible or consult manufacturer for recycling options.</li> <li>▸ Consult State Land Waste Authority for disposal.</li> <li>▸ Bury or incinerate residue at an approved site.</li> <li>▸ Recycle containers if possible, or dispose of in an authorised landfill.</li> </ul>
-------------------------------------	---

## SECTION 14 Transport information

### Labels Required

<b>Marine Pollutant</b>	NO
<b>HAZCHEM</b>	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
alcohols C9-11 ethoxylated	Not Available
hydrogen peroxide	Not Available
N,N-dimethyloctylamine N-oxide	Not Available

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

Product name	Ship Type
alcohols C9-11 ethoxylated	Not Available
hydrogen peroxide	Not Available
N,N-dimethyloctylamine N-oxide	Not Available

## SECTION 15 Regulatory information

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

alcohols C9-11 ethoxylated is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

hydrogen peroxide is found on the following regulatory lists

Hoover 1.47L Oxy Formula

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

Australian Inventory of Industrial Chemicals (AIIC)

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

**N,N-dimethyloctylamine N-oxide is found on the following regulatory lists**

Not Applicable

**National Inventory Status**

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (N,N-dimethyloctylamine N-oxide)
Canada - DSL	Yes
Canada - NDSL	No (alcohols C9-11 ethoxylated; hydrogen peroxide; N,N-dimethyloctylamine N-oxide)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (alcohols C9-11 ethoxylated; N,N-dimethyloctylamine N-oxide)
Japan - ENCS	No (alcohols C9-11 ethoxylated)
Korea - KECI	No (N,N-dimethyloctylamine N-oxide)
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (N,N-dimethyloctylamine N-oxide)
Vietnam - NCI	Yes
Russia - FBEPH	No (alcohols C9-11 ethoxylated; N,N-dimethyloctylamine N-oxide)
<b>Legend:</b>	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

**SECTION 16 Other information**

<b>Revision Date</b>	12/03/2021
<b>Initial Date</b>	12/03/2021

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

This document is copyright.

Apart from any fair dealing for the purposes of private study, research, review or criticism, as permitted under the Copyright Act, no part may be reproduced by any process without written permission from CHEMWATCH.

TEL (+61 3) 9572 4700.