

CRC (NZ) 3071 Glass Cleaner (Bulk)

CRC Industries (CRC Industries New Zealand)

Chemwatch Hazard Alert Code: 2

Chemwatch: 4546-90

Version No: 9.1.1.1

Safety Data Sheet according to WHS and ADG requirements

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SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	CRC (NZ) 3071 Glass Cleaner (Bulk)
Synonyms	window cleaner
Other means of identification	Not Available

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

Relevant identified uses	Commercial glass cleaner.
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Details of the supplier of the safety data sheet

Registered company name	CRC Industries (CRC Industries New Zealand)
Address	10 Highbrook Drive East Tamaki Auckland New Zealand
Telephone	+64 9 272 2700
Fax	+64 9 274 9696
Website	www.crc.co.nz
Email	customerservices@crc.co.nz

Emergency telephone number


Association / Organisation	CRC Industries (CRC Industries New Zealand)
Emergency telephone numbers	NZ Poisons Centre 0800 POISON (0800 764 766)
Other emergency telephone numbers	111 (NZ Emergency Services)

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Acute Aquatic 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	WARNING

Hazard statement(s)

H315	Causes skin irritation.
H319	Causes serious eye irritation.
H402	Harmful to aquatic life.

Precautionary statement(s) Prevention

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

Precautionary statement(s) Response

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

Continued...

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P332+P313 | If skin irritation occurs: Get medical advice/attention.

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

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

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
64-17-5	1-9	<u>ethanol</u>
111-76-2	1-9	<u>ethylene glycol monobutyl ether</u>
7632-00-0	<1	<u>sodium nitrite</u>
127087-87-0	<1	<u>4-nonylphenol, branched, ethoxylated</u>
1336-21-6	<1	<u>ammonium hydroxide</u>
Not Available	<1	additive, unregulated
7732-18-5	>60	<u>water</u>

SECTION 4 FIRST AID MEASURES

Description of first aid measures

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

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

Followed acute or short term repeated exposures to ethylene glycol monoalkyl ethers and their acetates:

- ▶ Hepatic metabolism produces ethylene glycol as a metabolite.
- ▶ Clinical presentation, following severe intoxication, resembles that of ethylene glycol exposures.
- ▶ Monitoring the urinary excretion of the alkoxyacetic acid metabolites may be a useful indication of exposure.

[Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 FIREFIGHTING MEASURES

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.

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	<ul style="list-style-type: none"> ▶ Wear breathing apparatus plus protective gloves in the event of a fire. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Use fire fighting procedures suitable for surrounding area. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ The material is not readily combustible under normal conditions. ▶ However, it will break down under fire conditions and the organic component may burn. ▶ Not considered to be a significant fire risk. ▶ Heat may cause expansion or decomposition with violent rupture of containers. ▶ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. <p>Decomposes on heating and produces toxic fumes of: carbon dioxide (CO₂) other pyrolysis products typical of burning organic material. May emit poisonous fumes.</p>
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	<p>Slippery when spilt.</p> <ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb spill with sand, earth, inert material or vermiculite. ▶ Wipe up. ▶ Place in a suitable, labelled container for waste disposal. 																																																																											
Major Spills	<p>Chemical Class: bases For release onto land: recommended sorbents listed in order of priority.</p> <table border="1"> <thead> <tr> <th>SORBENT TYPE</th> <th>RANK</th> <th>APPLICATION</th> <th>COLLECTION</th> <th>LIMITATIONS</th> </tr> </thead> <tbody> <tr> <td colspan="5">LAND SPILL - SMALL</td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>1</td> <td>shovel</td> <td>shovel</td> <td>R,W,SS</td> </tr> <tr> <td>cross-linked polymer - pillow</td> <td>1</td> <td>throw</td> <td>pitchfork</td> <td>R, DGC, RT</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>2</td> <td>shovel</td> <td>shovel</td> <td>R, I, P</td> </tr> <tr> <td>foamed glass - pillow</td> <td>2</td> <td>throw</td> <td>pitchfork</td> <td>R, P, DGC, RT</td> </tr> <tr> <td>expanded minerals - particulate</td> <td>3</td> <td>shovel</td> <td>shovel</td> <td>R, I, W, P, DGC</td> </tr> <tr> <td>foamed glass - particulate</td> <td>4</td> <td>shovel</td> <td>shovel</td> <td>R, W, P, DGC,</td> </tr> <tr> <td colspan="5">LAND SPILL - MEDIUM</td> </tr> <tr> <td>cross-linked polymer -particulate</td> <td>1</td> <td>blower</td> <td>skiploader</td> <td>R,W, SS</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>2</td> <td>blower</td> <td>skiploader</td> <td>R, I, P</td> </tr> <tr> <td>expanded mineral - particulate</td> <td>3</td> <td>blower</td> <td>skiploader</td> <td>R, I,W, P, DGC</td> </tr> <tr> <td>cross-linked polymer - pillow</td> <td>3</td> <td>throw</td> <td>skiploader</td> <td>R, DGC, RT</td> </tr> <tr> <td>foamed glass - particulate</td> <td>4</td> <td>blower</td> <td>skiploader</td> <td>R, W, P, DGC</td> </tr> <tr> <td>foamed glass - pillow</td> <td>4</td> <td>throw</td> <td>skiploader</td> <td>R, P, DGC., RT</td> </tr> </tbody> </table> <p>Legend DGC: Not effective where ground cover is dense R; Not reusable I: Not incinerable P: Effectiveness reduced when rainy RT:Not effective where terrain is rugged SS: Not for use within environmentally sensitive sites W: Effectiveness reduced when windy Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control; R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988</p> <p>Slippery when spilt. Moderate hazard.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Neutralise/decontaminate residue (see Section 13 for specific agent). ▶ Collect solid residues and seal in labelled drums for disposal. 	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS	LAND SPILL - SMALL					cross-linked polymer - particulate	1	shovel	shovel	R,W,SS	cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT	sorbent clay - particulate	2	shovel	shovel	R, I, P	foamed glass - pillow	2	throw	pitchfork	R, P, DGC, RT	expanded minerals - particulate	3	shovel	shovel	R, I, W, P, DGC	foamed glass - particulate	4	shovel	shovel	R, W, P, DGC,	LAND SPILL - MEDIUM					cross-linked polymer -particulate	1	blower	skiploader	R,W, SS	sorbent clay - particulate	2	blower	skiploader	R, I, P	expanded mineral - particulate	3	blower	skiploader	R, I,W, P, DGC	cross-linked polymer - pillow	3	throw	skiploader	R, DGC, RT	foamed glass - particulate	4	blower	skiploader	R, W, P, DGC	foamed glass - pillow	4	throw	skiploader	R, P, DGC., RT
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- ▶ Wash area and prevent runoff into drains.
- ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.
- ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ DO NOT allow material to contact humans, exposed food or food utensils. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	<ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Polyethylene or polypropylene container. ▶ Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	None known

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	ethanol	Ethyl alcohol	1000 ppm / 1880 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	ethylene glycol monobutyl ether	2-Butoxyethanol	20 ppm / 96.9 mg/m ³	242 mg/m ³ / 50 ppm	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
ethanol	Ethanol: (Ethyl alcohol)	Not Available	Not Available	15000* ppm
ethylene glycol monobutyl ether	Butoxyethanol, 2-; (Glycol ether EB)	60 ppm	120 ppm	700 ppm
sodium nitrite	Sodium nitrite	6.4 mg/m ³	71 mg/m ³	240 mg/m ³
4-nonylphenol, branched, ethoxylated	Nonylphenol, 4-, branched, ethoxylated	30 mg/m ³	330 mg/m ³	2,000 mg/m ³
4-nonylphenol, branched, ethoxylated	Nonylphenoxypolyethoxyethanol	30 mg/m ³	330 mg/m ³	2,000 mg/m ³
ammonium hydroxide	Ammonium hydroxide	61 ppm	330 ppm	2,300 ppm

Ingredient	Original IDLH	Revised IDLH
ethanol	3,300 ppm	Not Available
ethylene glycol monobutyl ether	700 ppm	Not Available
sodium nitrite	Not Available	Not Available
4-nonylphenol, branched, ethoxylated	Not Available	Not Available
ammonium hydroxide	Not Available	Not Available
water	Not Available	Not Available

OCCUPATIONAL EXPOSURE BANDING

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
sodium nitrite	E	≤ 0.01 mg/m ³
4-nonylphenol, branched, ethoxylated	E	≤ 0.1 ppm

ammonium hydroxide	E	≤ 0.1 ppm
Notes:	<i>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.</i>	

MATERIAL DATA

Odour Safety Factor(OSF) OSF=0.0023 (polyethylene glycol mono-p-nonylphenyl ether, branched)

For ethylene glycol monobutyl ether (2-butoxyethanol)

Odour Threshold Value: 0.10 ppm (detection), 0.35 ppm (recognition)

Although rats appear to be more susceptible than other animals anaemia is not uncommon amongst humans following exposure. The TLV reflects the need to maintain exposures below levels found to cause blood changes in experimental animals. It is concluded that this limit will reduce the significant risk of irritation, haematologic effects and other systemic effects observed in humans and animals exposed to higher vapour concentrations. The toxic effects typical of some other glycol ethers (pancytopenia, testis atrophy and teratogenic effects) are not found with this substance.

Odour Safety Factor (OSF)

OSF=2E2 (2-BUTOXYETHANOL)

for exposure to ammonia gas/ vapours:

Odour Threshold Value: Variously reported as 0.019 ppm and 55 ppm; AIHA Value 16.7 ppm (detection)

NOTE: Detector tubes for ammonia, measuring in excess of 1 ppm, are commercially available.

The TLV-TWA is thought to be protective against irritation of the eyes and respiratory tract and minimise discomfort among workers that are not inured to its effects and systemic damage. Acclimatised persons are able to tolerate prolonged exposures of up to 100 ppm without symptoms. Marked irritation has been seen in persons exposed to ammonia concentrations between 50 and 100 ppm only when the exposures involved sudden concentration peaks which do not permit short-term acclimatisation. The detoxification capacity of the liver is significant since the amount of ammonia formed endogenously in the intestines markedly exceeds that from external sources.

Human exposure effects, at vapour concentrations of about:

Concentration (ppm)	Possible Effects
5	minimal irritation
9-50	nasal dryness, olfactory fatigue and moderate irritation
125-137	definite nose, throat and chest irritation
140	slight eye irritation
150	laryngeal spasm
500	30 minute exposures may produce cyclic hypernea, increased blood pressure and pulse rate, and upper respiratory tract irritation which may persist for 24 hours
700	immediate eye irritation
	dyspnea, convulsive coughing, chest pain, respiratory spasm, pink frothy sputum, rapid asphyxia and delayed pulmonary oedema which may be fatal. Other effects include runny nose, swelling of the lips, restlessness, headache, salivation, nausea, vomiting, glottal oedema, pharyngitis, tracheitis, and speech difficulties.
1,500-10,000	Bronchopneumonia, asphyxiation due to spasms, inflammation, and oedema of the larynx, may be fatal. Residual effects include hoarseness, productive cough, and decreased respiratory function
	severe eye irritation, with swelling of the eyelids, lachrymation, blepharospasm, palpebral oedema, increased intraocular pressure, oval semi-dilated, fixed pupils, corneal ulceration (often severe) and temporary blindness. Depending on duration of exposure, there may be destruction of the epithelium, corneal and lenticular opacification, and iritis accompanied by hypopyon or haemorrhage and possible loss of pigment from the posterior layer of the iris. Less severe damage is often resolved. In the case of severe damage, symptoms may be delayed; late complications including persistent oedema, vascularisation and corneal scarring, permanent opacity, acute angle glaucoma, staphyloma, cataract, and atrophy of the retina, iris, and symblepharon.
>2,500	Long-term exposure to sub-acute concentrations or single exposures to high concentrations may produce chronic airway dysfunction, alveolar disease, bronchiolitis, bronchiectasis, emphysema and anxiety neuroses

Odour Safety Factor(OSF)

OSF=3.8 (AMMONIA)

For ethanol:

Odour Threshold Value: 49-716 ppm (detection), 101 ppm (recognition)

Eye and respiratory tract irritation do not appear to occur at exposure levels of less than 5000 ppm and the TLV-TWA is thought to provide an adequate margin of safety against such effects. Experiments in man show that inhalation of 1000 ppm caused slight symptoms of poisoning and 5000 ppm caused strong stupor and morbid sleepiness. Subjects exposed to 5000 ppm to 10000 ppm experienced smarting of the eyes and nose and coughing. Symptoms disappeared within minutes. Inhalation also causes local irritating effects to the eyes and upper respiratory tract, headaches, sensation of heat intraocular tension, stupor, fatigue and a need to sleep. At 15000 ppm there was continuous lachrymation and coughing.

None assigned for mixture or identified for ingredient(s).

Exposed individuals are **NOT** reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

Class OSF Description

A	550	Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities
B	26-550	As "A" for 50-90% of persons being distracted
C	1-26	As "A" for less than 50% of persons being distracted
D	0.18-1	10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached
E	<0.18	As "D" for less than 10% of persons aware of being tested

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.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in special circumstances. If risk of</p>
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overexposure exists, wear approved respirator. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. Provide adequate ventilation in warehouses and enclosed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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

Within each range the appropriate value depends on:

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

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

Personal protection



Eye and face protection

- ▶ Safety glasses with side shields.
- ▶ Chemical goggles.
- ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection

See Hand protection below

Hands/feet protection

- ▶ Wear chemical protective gloves, e.g. PVC.
 - ▶ Wear safety footwear or safety gumboots, e.g. Rubber
- The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
- The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
- Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
- Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
- frequency and duration of contact,
 - chemical resistance of glove material,
 - glove thickness and
 - dexterity
- Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
 - When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
 - Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
 - Contaminated gloves should be replaced.
- As defined in ASTM F-739-96 in any application, gloves are rated as:
- Excellent when breakthrough time > 480 min
 - Good when breakthrough time > 20 min
 - Fair when breakthrough time < 20 min
 - Poor when glove material degrades
- For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.
- It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.
- Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.
- Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:
- Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.
 - Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential
- Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed

	moisturiser is recommended.
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C. apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit.

Recommended material(s)**GLOVE SELECTION INDEX**

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:

CRC (NZ) 3071 Glass Cleaner (Bulk)

Material	CPI
BUTYL	A
NEOPRENE	B
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
SARANEX-23	C
VITON	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

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

Appearance	Clear colourless liquid with a slight ammoniacal odour; miscible with water.		
Physical state	Liquid	Relative density (Water = 1)	0.98
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Applicable	Decomposition temperature	Not Available
Melting point / freezing point (°C)	<0	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	100	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	As for water	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	100
Vapour pressure (kPa)	As for water	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	As for water	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	<p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. Inhalation hazard is increased at higher temperatures.</p> <p>The highly irritant properties of ammonia vapour result as the gas dissolves in mucous fluids and forms irritant, even corrosive solutions. Inhalation of the ammonia fumes causes coughing, vomiting, reddening of lips, mouth, nose, throat and conjunctiva while higher concentrations can cause temporary blindness, restlessness, tightness in the chest, pulmonary oedema (lung damage), weak pulse and cyanosis. Inhalation of high concentrations of vapour may cause breathing difficulty, tightness in chest, pulmonary oedema and lung damage. Brief exposure to high concentrations > 5000 ppm may cause death due to asphyxiation (suffocation) or fluid in the lungs. Prolonged or regular minor exposure to the vapour may cause persistent irritation of the eyes, nose and upper respiratory tract. Massive ammonia exposures may produce chronic airway hyperactivity and asthma with associated pulmonary function changes. The average nasal retention of ammonia by human subjects was found to be 83%.</p>
Ingestion	<p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>The lethal oral dose of nitrite for adults has been variously reported to be between 0.7 and 6 g NO₂⁻ (approximately 10 to 100 mg NO₂⁻/kg). Lower doses may apply for children (especially neonates), the elderly and people with certain enzyme deficiencies. The first symptoms of oral nitrite poisoning develop within 15 to 45 minutes</p> <p>In humans, inorganic nitrites produce smooth muscle relaxation, methaemoglobinaemia and cyanosis. The primary effect of nitrite intoxication in animals is methaemoglobinaemia whilst secondary effects include vasodilation, relaxation of smooth muscle and lowering of blood pressure. Other nitrite-induced toxic effects include abdominal pain, diarrhoea, atrophied intestinal villi and apoptotic cell death in the intestinal crypts. Nitrite may also cause sudden fall in blood pressure due to its vasodilating properties. Nitrite has vasodilating properties, probably through transformation into nitric oxide (NO) or a NO-containing molecule acting as a signal factor for smooth muscle relaxation.</p> <p>Fatal poisonings in infants, resulting from ingestion of nitrates in water or spinach, have been reported.</p> <p>When sodium nitrite was administered in drinking water for 6 weeks (0.06-1%), mice showed a slight degeneration and spotty necrosis of hepatocytes and haemosiderin deposition in the liver, spleen and lymph nodes, indicating haemolysis. At 2%, mice died within 3 weeks. In rats, subject to the same treatment regime, abnormal blood and spleen colours, due to MHG, were seen in 0.5% and 1.0% treatment groups. Hepatic microsomal lipoperoxidation (as measured by malondialdehyde formation) was increased in male rats given 0.2% sodium nitrite in drinking water. Liver lysosomal enzymes (acid phosphatase and cathepsin) and superoxide dismutase activities were also increased. This data suggests that the nitrite stimulates generation of superoxide radicals in the liver causing damage to cellular and subcellular membranes. Decreased plasma vitamin E and greater reduced glutathione-per erythrocyte were also reported in male rats receiving sodium nitrite in drinking water.</p>
Skin Contact	<p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</p> <p>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 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. Open cuts, abraded or irritated skin should not be exposed to this material</p>
Eye	The liquid may produce eye discomfort causing transient smarting, blinking
Chronic	<p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>Prolonged or repeated minor exposure to ammonia gas/vapour may cause long-term irritation to the eyes, nose and upper respiratory tract. Repeated exposure or prolonged contact may produce dermatitis, and conjunctivitis.</p> <p>Other effects may include ulcerative changes to the mouth and bronchial and gastrointestinal disturbances. Adaptation to usually irritating concentrations may result in tolerance. In animals, repeated exposures to sub-lethal levels produces adverse effects on the respiratory tract, liver, kidneys and spleen. Exposure at 675 ppm for several weeks produced eye irritation in dogs and rabbits; corneal opacity, covering between a quarter to one half of the total surface area, was evident in rabbits.</p> <p>Long-term exposure to ethanol may result in progressive liver damage with fibrosis or may exacerbate liver injury caused by other agents.</p> <p>Repeated ingestion of ethanol by pregnant women may adversely affect the central nervous system of the developing foetus, producing effects collectively described as foetal alcohol syndrome. These include mental and physical retardation, learning disturbances, motor and language deficiency, behavioural disorders and reduced head size.</p> <p>Consumption of ethanol (in alcoholic beverages) may be linked to the development of Type I hypersensitivities in a small number of individuals. Symptoms, which may appear immediately after consumption, include conjunctivitis, angioedema, dyspnoea, and urticarial rashes. The causative agent may be acetic acid, a metabolite (1).</p> <p>(1) Boehncke W.H., & H.Gall, Clinical & Experimental Allergy, 26, 1089-1091, 1996</p> <p>The major concern of possible long-term effects of exposure to nitrate and nitrite is associated with formation of nitroso compounds, many of which are carcinogenic. This formation may take place wherever nitrite and nitrosable compounds are present, but it is favoured by acidic conditions or the presence of some bacteria. The gastrointestinal tract and especially the stomach is regarded as the main formation site, but nitrosation reactions can also take place in an infected urinary bladder.</p> <p>Nitrite is mutagenic in a number of in vitro assays against microorganisms or cultured mammalian cells. Nitrates show no mutagenic activity in microbial tests under aerobic conditions. Activity has been reported under anaerobic conditions, probably due to reduction of nitrate into nitrite</p> <p>The mutagenic effects of nitrites were observed in an in vivo and in vitro experiment using Syrian hamsters. In vivo assays have been equivocal, both positive and negative results having been reported</p> <p>Exposure to sodium nitrite in drinking water resulted in an increased incidence of epithelial hyperplasia in the forestomach of male and female rats and in the glandular stomach of male mice.</p> <p>There was equivocal evidence of carcinogenic activity of sodium nitrite in female B6C3F1 mice based on the positive trend in the incidences of squamous cell papillomas or carcinomas (combined) of the forestomach. There was no evidence of carcinogenic activity in male and female F344/N rats or B6C3F1 male mice exposed to 750, 1500 or 3000 ppm.</p> <p>NTP Technical Report Series No. 495, May 2001</p> <p>Under certain conditions, nitrites can react with secondary amines, either alone or in biological systems, to form carcinogenic nitrosamines.</p>

CRC (NZ) 3071 Glass Cleaner (Bulk)

Sodium nitrite (60 mg/kg) administered in drinking water to pregnant guinea pigs produced maternal anaemia and increased the incidences of abortion and foetal mortality. Administration of 2000-3000 mg/l sodium nitrite in drinking water, to pregnant rats, produced 30-53% foetal mortality. In rat dams given 0.025-0.5% in feed, sodium nitrite caused an increase in foetal and pup mortality and decreases in pre-weanling body weights. 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.

CRC (NZ) 3071 Glass Cleaner (Bulk)	TOXICITY	IRRITATION
	Not Available	Not Available
ethanol	TOXICITY	IRRITATION
	Inhalation (rat) LC50: 124.7 mg/l/4H ^[2]	Eye (rabbit): 500 mg SEVERE
	Oral (rat) LD50: =1501 mg/kg ^[2]	Eye (rabbit):100mg/24hr-moderate
		Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit):20 mg/24hr-moderate
	Skin (rabbit):400 mg (open)-mild	
	Skin: no adverse effect observed (not irritating) ^[1]	
ethylene glycol monobutyl ether	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 100 mg SEVERE
	Inhalation (rat) LC50: 449.48655 mg/l/4H ^[2]	Eye (rabbit): 100 mg/24h-moderate
	Oral (rat) LD50: 250 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit): 500 mg, open; mild
	Skin: adverse effect observed (irritating) ^[1]	
	Skin: no adverse effect observed (not irritating) ^[1]	
sodium nitrite	TOXICITY	IRRITATION
	Inhalation (rat) LC50: 0.0055 mg/l/4H ^[2]	Eye (rabbit): 500 mg/24hr - mild
	Oral (rat) LD50: =85 mg/kg ^[2]	
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]	
ammonium hydroxide	TOXICITY	IRRITATION
	Inhalation (rat) LC50: 1997.718 mg/l/4h ^[2]	Eye (rabbit): 0.25 mg SEVERE
	Oral (rat) LD50: 350 mg/kg ^[2]	Eye (rabbit): 1 mg/30s SEVERE
water	TOXICITY	IRRITATION
	Oral (rat) LD50: >90000 mg/kg ^[2]	Not Available

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

CRC (NZ) 3071 Glass Cleaner (Bulk)	Not available for mixture or identified for ingredient(s).
ETHANOL	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 of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
ETHYLENE GLYCOL MONOBUTYL ETHER	NOTE: Changes in kidney, liver, spleen and lungs are observed in animals exposed to high concentrations of this substance by all routes. ** ASCC (NZ) SDS For ethylene glycol monoalkyl ethers and their acetates (EGMAEs): Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates. EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers. Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitizers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general.

Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE *in vitro* than those of rats.

Repeat dose toxicity: The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA *in vitro* and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA *in vitro*.

Mutagenicity: In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in *S. typhimurium* strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. *In vitro* cytogenetic and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and *in vivo* micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.

Carcinogenicity: In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity

Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes).

Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m³ and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m³), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m³), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m³) indicate that the members of the category are not teratogenic.

The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m³ (rabbit-EGPE), 100 ppm or 425 mg/m³ (rat-EGPE), 50 ppm or 241 mg/m³ (rat EGBE) and 100 ppm or 483 mg/m³ (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m³ (rat and rabbit-EGHE).

Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetotoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species.

At least one researcher has stated that the reproductive effects were less than that of other monoalkyl ethers of ethylene glycol.

Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility.

Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions (1). The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of erythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration -dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma.

1: NTP Toxicology Program Technical report Series 484, March 2000.

For ethylene glycol:

Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO₂, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO₂, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

Respiratory Effects. Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning. The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning. Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases).

Cardiovascular Effects. Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12- 24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol. Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases. Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.

Gastrointestinal Effects. Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition.

Musculoskeletal Effects. Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tenderness and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia.

Hepatic Effects. Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol.

Renal Effects. Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, anuria, and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near normal renal function can return with adequate supportive therapy.

Metabolic Effects. One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid.

	<p>Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate).</p> <p>Neurological Effects: Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion. Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months.</p> <p>Reproductive Effects: Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration.</p> <p>Developmental Effects: The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embryotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight.</p> <p>Cancer: No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol.</p> <p>Genotoxic Effects: Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available <i>in vivo</i> and <i>in vitro</i> laboratory studies provide consistently negative genotoxicity results for ethylene glycol.</p>
<p>SODIUM NITRITE</p>	<p>Tumorigenic - Carcinogenic by RTECS criteria.</p> <p>Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells <i>in vivo</i>. Such findings are often supported by positive results from <i>in vitro</i> mutagenicity studies.</p> <p>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
<p>4-NONYLPHENOL, BRANCHED, ETHOXYLATED</p>	<p>For nonylphenol and its compounds:</p> <p>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 endogenous hormone for binding with the estrogen receptors ERalpha and ERbeta.</p> <p>Effects in pregnant women.</p> <p>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.</p> <p>Nonylphenol has also been shown to affect cytokine signaling molecule secretions in the human placenta. <i>In vitro</i> 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.</p> <p>Effects on metabolism</p> <p>Nonylphenol has been shown to act as an obesity enhancing chemical or obesogen, though it has paradoxically been shown to have anti-obesity 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 to 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.</p> <p>Cancer</p> <p>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</p> <p>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.</p> <p>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.</p> <p>On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autooxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.</p> <p>Allergic Contact Dermatitis—Formation, Structural Requirements, and Reactivity of Skin Sensitizers.</p> <p>Ann-Therese Karlberg et al; Chem. Res. Toxicol.2008,21,53-69</p> <p>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.</p> <p>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</p>

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 autooxidation 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 (CO₂). Orally dosed AE was absorbed rapidly and extensively in rats, and more than 75% of the dose was absorbed. When applied to the skin of humans, the doses were absorbed slowly and incompletely (50% absorbed in 72 hours). Half of the absorbed surfactant was excreted promptly in the urine and smaller amounts of AE appeared in the faeces and expired air (CO₂). The metabolism of C12 AE yields PEG, carboxylic acids, and CO₂ as metabolites. The LD50 values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.

The ability of nonionic surfactants to cause a swelling of the stratum corneum of guinea pig skin has been studied. The swelling mechanism of the skin involves a combination of ionic binding of the hydrophilic group as well as hydrophobic interactions of the alkyl chain with the substrate. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants. A substantial amount of toxicological data and information in vivo and in vitro demonstrates that there is no evidence for alcohol ethoxylates (AEs) being genotoxic, mutagenic or carcinogenic. No adverse reproductive or developmental effects were observed. The majority of available toxicity studies revealed NOAELs in excess of 100 mg/kg bw/d but the lowest NOAEL for an individual AE was established to be 50 mg/kg bw/day. This value was subsequently considered as a conservative, representative value in the risk assessment of AE. The effects were restricted to changes in organ weights with no histopathological organ changes with the exception of liver hypertrophy (indicative of an adaptive response to metabolism rather than a toxic effect). It is noteworthy that there was practically no difference in the NOAEL in oral studies of 90-day or 2 years of duration in rats. A comparison of the aggregate consumer exposure and the systemic NOAEL (taking into account an oral absorption value of 75%) results in a Margin of Exposure of 5,800. Taking into account the conservatism in the exposure assessment and the assigned systemic NOAEL, this margin of exposure is considered more than adequate to account for the inherent uncertainty and variability of the hazard database and inter and intra-species extrapolations.

AEs are not contact 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/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

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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. 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 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.

Nonylphenol was not mutagenic to Salmonella typhimurium, TA100, TA1535, TA98, TA1537 and Escherichia coli WP2 uvrA, with or without an exogenous metabolic activation system.

Nonylphenol induced neither structural chromosomal aberrations nor polyploidy in CHL/IU cells, in the absence or presence of an exogenous metabolic activation system.

for linear material: Maternal effects, effects on fertility recorded.

AMMONIUM HYDROXIDE

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.

WATER

No significant acute toxicological data identified in literature search.

ETHYLENE GLYCOL MONOBUTYL ETHER & 4-NONYLPHENOL, BRANCHED, ETHOXYLATED & AMMONIUM HYDROXIDE

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

ETHYLENE GLYCOL MONOBUTYL ETHER & 4-NONYLPHENOL, BRANCHED, ETHOXYLATED

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

Acute Toxicity	✗	Carcinogenicity	✗
Skin Irritation/Corrosion	✓	Reproductivity	✗
Serious Eye Damage/Irritation	✓	STOT - Single Exposure	✗

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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

CRC (NZ) 3071 Glass Cleaner (Bulk)	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
ethanol	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	11-mg/L	2
	EC50	48	Crustacea	2mg/L	4
	EC50	96	Algae or other aquatic plants	17.921mg/L	4
NOEC	2016	Fish	0.000375mg/L	4	
ethylene glycol monobutyl ether	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1-700mg/L	2
	EC50	48	Crustacea	ca.1-800mg/L	2
	EC50	72	Algae or other aquatic plants	1-840mg/L	2
NOEC	24	Crustacea	>1-mg/L	2	
sodium nitrite	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.048mg/L	4
	EC50	48	Crustacea	ca.12.5100mg/L	1
	EC50	96	Algae or other aquatic plants	12.537mg/L	3
NOEC	96	Fish	0.02mg/L	4	
4-nonylphenol, branched, ethoxylated	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>10mg/L	2
	EC50	48	Crustacea	14mg/L	2
	EC50	96	Algae or other aquatic plants	12mg/L	2
NOEC	96	Algae or other aquatic plants	8mg/L	2	
ammonium hydroxide	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	15mg/L	4
NOEC	72	Fish	3.5mg/L	4	
water	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	897.520mg/L	3
EC50	96	Algae or other aquatic plants	8768.874mg/L	3	

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

Harmful to aquatic organisms.

For ethylene glycol monoalkyl ethers and their acetates:

Members of this category include ethylene glycol propyl ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE)

Environmental fate:

The ethers, like other simple glycol ethers possess no functional groups that are readily subject to hydrolysis in the presence of waters. The acetates possess an ester group that hydrolyses in neutral ambient water under abiotic conditions.

Level III fugacity modeling indicates that category members, when released to air and water, will partition predominately to water and, to a lesser extent, to air and soil. Estimates of soil and sediment partition coefficients (Kocs ranging from 1- 10) suggest that category members would exhibit high soil mobility. Estimated bioconcentration factors (log BCF) range from 0.463 to 0.732. Biodegradation studies indicate that all category members are readily biodegradable. The physical chemistry and environmental fate properties indicate that category members will not persist or bioconcentrate in the environment.

Ecotoxicity:

Glycol ether acetates do not hydrolyse rapidly into their corresponding glycol ethers in water under environmental conditions. The LC50 or EC50 values for EGHE are lower than those for EGPE and EGBE (which have shorter chain lengths and lower log Kow values). Overall, the LC50 values for the glycol ethers in aquatic species range from 94 to > 5000 mg/L. For EGHE, the 96-hour LC50 for *Brachydanio rerio* (zebra fish) is between 94 and mg/L, the 48-hour EC50 for *Daphnia magna* was 145 mg/L and the 72-hour EC50 values for biomass and growth rate of algae (*Scenedesmus subspicatus*) were 98 and 198 mg/L, respectively. LC50/EC50 values for EGPE and EGBE in aquatic species are 835 mg/l or greater.

Aquatic toxicity data for EGBEA show a 96-hour LC50 of 28.3 mg/L for rainbow trout (*Oncorhynchus mykiss*), a 48-hour LC50 of 37-143 mg/L for *Daphnia magna*, a 72-hour EC50 of greater than 500 mg/L for biomass or growth rate of algae (*Scenedesmus subspicatus* and *Pseudokirchneriella subcapitata*, respectively), and a 7-day EC10 of 30.4 mg/L and a NOEC of 16.4 mg/L for reproduction in *Ceriodaphnia dubia*.

The nitrates are of environmental concern because of their high water solubility and consequent leaching, diffusion, and environmental mobility in soil and water. Nitrate can contaminate groundwater to unacceptable levels. Nitrite is formed from nitrate or ammonium ion by micro-organisms in soil, water, sewage and the alimentary tract. The concern with nitrate in the environment is related to its conversion to nitrite.

Methaemoglobinemia is caused following exposure to high levels of nitrite and produces difficulties in oxygen transport in the blood. Thousands of cases involving poisoning of infants, particularly in rural areas, have been reported as a result of drinking nitrate rich well-water.

Continued...

Other concerns deriving from exposure to environmental nitrates relate to the production of nitrosamines following the reaction of food nitrites and secondary amines. Other nitroso-compounds may result following reaction with nitrites and amides, ureas, carbamates and other nitrogenous compounds. Nitrosamines produce liver damage, haemorrhagic lung lesions, convulsions and coma in rats, and teratogenic effects in experimental animals.

The N-nitroso class of compounds include potent carcinogens and mutagens: induction of tumors by single doses of N-nitroso compounds testify to this.

In air ammonia is persistent whilst, in water, it biodegrades rapidly to nitrate, producing a high oxygen demand. Ammonia is strongly adsorbed to soil. Ammonia is non-persistent in water (half-life 2 days) and is moderately toxic to fish under normal temperature and pH conditions. Ammonia is harmful to aquatic life at low concentrations but does not concentrate in the food chain. Ammonium ions may be toxic to fish at 0.3 mg/l

Drinking Water Standards:

0.5 mg/l (UK max.)

1.5 mg/l (WHO Levels)

Soil Guidelines: none available.

Air Quality Standards: none available.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ethanol	LOW (Half-life = 2.17 days)	LOW (Half-life = 5.08 days)
ethylene glycol monobutyl ether	LOW (Half-life = 56 days)	LOW (Half-life = 1.37 days)
sodium nitrite	LOW	LOW
water	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
ethanol	LOW (LogKOW = -0.31)
ethylene glycol monobutyl ether	LOW (BCF = 2.51)
sodium nitrite	LOW (LogKOW = 0.0564)
water	LOW (LogKOW = -1.38)

Mobility in soil

Ingredient	Mobility
ethanol	HIGH (KOC = 1)
ethylene glycol monobutyl ether	HIGH (KOC = 1)
sodium nitrite	LOW (KOC = 23.74)
water	LOW (KOC = 14.3)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	<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"> ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) <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"> ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority. ▶ Recycle wherever possible. ▶ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▶ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible material). ▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.
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SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

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

Not Applicable

SECTION 15 REGULATORY INFORMATION**Safety, health and environmental regulations / legislation specific for the substance or mixture****ETHANOL IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

ETHYLENE GLYCOL MONOBUTYL ETHER IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

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

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

SODIUM NITRITE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2

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

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7

4-NONYLPHENOL, BRANCHED, ETHOXYLATED IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

Chemical Footprint Project - Chemicals of High Concern List

AMMONIUM HYDROXIDE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

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

WATER IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

National Inventory Status

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (4-nonylphenol, branched, ethoxylated; ethanol; water; sodium nitrite; ammonium hydroxide; ethylene glycol monobutyl ether)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (4-nonylphenol, branched, ethoxylated)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - ARIPS	Yes
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing (see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Revision Date	01/11/2019
Initial Date	01/11/2009

SDS Version Summary

Version	Issue Date	Sections Updated
8.1.1.1	05/09/2017	Supplier Information
9.1.1.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification

Other information

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

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

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit.
IDLH: Immediately Dangerous to Life or Health Concentrations
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index

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