



Safety Data Sheet

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Graco Pump Armor

MSD029ENNZ
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Graco (Graco Australia)

Chemwatch: 6101-74

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Safety Data Sheet according to the Health and Safety at Work (Hazardous Substances) Regulations 2017

Chemwatch Hazard Alert Code: 3

Issue Date: 06/02/2023

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L.GHS.NZL.EN.E

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

Product Identifier

Product name	Graco Pump Armour
Chemical Name	Not Applicable
Synonyms	243103, 245133, 243104, 244168, 258555, 248556, 253574, 16M816, 24D386, 16W448
Proper shipping name	TOXIC LIQUID, INORGANIC, N.O.S. (contains sodium nitrite)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Corrosion Inhibitor/Lubricant Use according to manufacturer's directions.
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Details of the manufacturer or supplier of the safety data sheet

Registered company name	Graco (Graco Australia)
Address	Suite 17, 2 Enterprise Drive Bundoora VIC 3083 Australia
Telephone	+61 3 9468 8500
Fax	+61 3 9468 8599
Website	www.graco.com
Email	ANZTA@graco.com

Emergency telephone number

Association / Organisation	Graco (Graco Australia)
Emergency telephone numbers	+61 3 9468 8500 (Mon-Fri 8am-18pm), +61 467 836 080 (After Hours)
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

Classification [1]	Acute Toxicity (Oral) Category 4, Serious Eye Damage/Eye Irritation Category 2, Germ Cell Mutagenicity Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI
Determined by Chemwatch using GHS/HSNO criteria	6.1D (oral), 6.4A, 6.6B, 6.9A, 9.1B

Label elements

Hazard pictogram(s)	
Signal word	Danger

Hazard statement(s)

H302	Harmful if swallowed.
H319	Causes serious eye irritation.
H341	Suspected of causing genetic defects.
H372	Causes damage to organs through prolonged or repeated exposure.
H411	Toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
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P260	Do not breathe mist/vapours/spray.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P314	Get medical advice/attention if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P330	Rinse mouth.

Precautionary statement(s) Storage

P405	Store locked up.
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Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
107-21-1	40-60	<u>ethylene glycol</u>
7758-11-4	0.1-1	<u>potassium phosphate, dibasic</u>
7632-00-0	0.1-1	<u>sodium nitrite</u>
3734-33-6	<0.1	<u>denatonium benzoate</u>
Legend:	1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▶ Immediately hold eyelids apart and flush the eye continuously with 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. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs: ▶ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▶ Quickly remove all contaminated clothing, including footwear. ▶ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▶ Transport to hospital, or doctor.
Inhalation	▶ 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, without delay.
Ingestion	▶ 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. ▶ Avoid giving milk or oils. ▶ Avoid giving alcohol.

Indication of any immediate medical attention and special treatment needed

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.

- ▶ Polyethylene glycols are generally poorly absorbed orally and are mostly unchanged by the kidney.
- ▶ Dermal absorption can occur across damaged skin (e.g. through burns) leading to increased osmolality, anion gap metabolic acidosis, elevated calcium, low ionised calcium, CNS

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depression and renal failure.

- Treatment consists of supportive care.

[Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 Firefighting measures

Extinguishing media

- Alcohol stable foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.
- Water spray or fog - Large fires only.

Special hazards arising from the substrate or mixture

Fire Incompatibility	▸ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ Avoid spraying water onto liquid pools. ▸ 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.
Fire/Explosion Hazard	▸ Combustible. ▸ Slight fire hazard when exposed to heat or flame. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke. ▸ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO₂) nitrogen oxides (NO_x) sulfur oxides (SO_x) metal oxides other pyrolysis products typical of burning organic material.

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. Slippery when spilt. ▸ 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	Environmental hazard - contain spillage. Chemical Class: alcohols and glycols For release onto land: recommended sorbents listed in order of priority.				
	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
	wood fiber - pillow	3	throw	pitchfork	R, P, DGC, RT
	treated wood fiber - pillow	3	throw	pitchfork	DGC, RT
	foamed glass - pillow	4	throw	pichfork	R, P, DGC, RT
	LAND SPILL - MEDIUM				
	cross-linked polymer - particulate	1	blower	skiploader	R,W, SS
	polypropylene - particulate	2	blower	skiploader	W, SS, DGC
	sorbent clay - particulate	2	blower	skiploader	R, I, W, P, DGC
	polypropylene - mat	3	throw	skiploader	DGC, RT

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expanded mineral - particulate	3	blower	skiploader	R, I, W, P, DGC
polyurethane - mat	4	throw	skiploader	DGC, RT

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

Slippery when spilt.

Moderate hazard.

- ▶ Clear area of personnel and move upwind.
- ▶ Alert Fire Brigade and tell them location and nature of hazard.
- ▶ Wear breathing apparatus plus protective gloves.
- ▶ Prevent, by any means available, spillage from entering drains or water course.
- ▶ No smoking, naked lights or ignition sources.
- ▶ Increase ventilation.
- ▶ Stop leak if safe to do so.
- ▶ Contain spill with sand, earth or vermiculite.
- ▶ Collect recoverable product into labelled containers for recycling.
- ▶ Absorb remaining product with sand, earth or vermiculite.
- ▶ Collect solid residues and seal in labelled drums for disposal.
- ▶ Wash area and prevent runoff into drains.
- ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ 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. ▶ Avoid smoking, naked lights or ignition sources. ▶ 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. ▶ 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.
Other information	Consider storage under inert gas. ▶ 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	▶ DO NOT use aluminium or galvanised containers ▶ Metal can or drum ▶ Packaging as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▶ Glycols and their ethers undergo violent decomposition in contact with 70% perchloric acid. This seems likely to involve formation of the glycol perchlorate esters (after scission of ethers) which are explosive, those of ethylene glycol and 3-chloro-1,2-propanediol being more powerful than glyceryl nitrate, and the former so sensitive that it explodes on addition of water. Metal nitrites: ▶ are incompatible with chlorates, hypophosphites, iodides, mercury salts, permanganates, sulfites, primary amines and amides, secondary amines and amides, ammonium salts, activated carbon, cyanogen compounds, thiocyanates, thiosulfates, cyanides, sodium amide, boron, acetanilide, antipyrine, tannic acid and cellulose ▶ react explosively with hydrazine and liquid ammonia . ▶ react explosively following fusion with metal cyanides ▶ react (often) with salts of nitrogenous bases to produce an unstable corresponding nitrite salt. Alcohols ▶ are incompatible with strong acids, acid chlorides, acid anhydrides, oxidising and reducing agents. ▶ reacts, possibly violently, with alkaline metals and alkaline earth metals to produce hydrogen ▶ react with strong acids, strong caustics, aliphatic amines, isocyanates, acetaldehyde, benzoyl peroxide, chromic acid, chromium oxide, dialkylzincs, dichlorine oxide, ethylene oxide, hypochlorous acid, isopropyl chlorocarbonate, lithium tetrahydroaluminate, nitrogen dioxide, pentafluoroguanidine, phosphorus halides, phosphorus pentasulfide, tangerine oil, triethylaluminium, triisobutylaluminium ▶ should not be heated above 49 deg. C. when in contact with aluminium equipment Ethylene glycol: ▶ reacts violently with oxidisers and oxidising acids, sulfuric acid, chlorosulfonic acid, chromyl chloride, perchloric acid ▶ forms explosive mixtures with sodium perchlorate

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- ▶ is incompatible with strong acids, caustics, aliphatic amines, isocyanates, chlorosulfonic acid, oleum, potassium bichromate, phosphorus pentasulfide, sodium chlorite

▶ Avoid strong acids, bases.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
New Zealand Workplace Exposure Standards (WES)	ethylene glycol	Ethylene glycol (vapour and mist)	Not Available	Not Available	50 ppm / 127 mg/m3	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
ethylene glycol	30 ppm	150 ppm	900 ppm
potassium phosphate, dibasic	16 mg/m3	180 mg/m3	1,100 mg/m3
potassium phosphate, dibasic	13 mg/m3	140 mg/m3	830 mg/m3
sodium nitrite	6.4 mg/m3	71 mg/m3	240 mg/m3

Ingredient	Original IDLH	Revised IDLH
ethylene glycol	Not Available	Not Available
potassium phosphate, dibasic	Not Available	Not Available
sodium nitrite	Not Available	Not Available
denatonium benzoate	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
potassium phosphate, dibasic	E	≤ 0.01 mg/m³
sodium nitrite	E	≤ 0.01 mg/m³
denatonium benzoate	E	≤ 0.01 mg/m³

Notes:

Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.

MATERIAL DATA

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in specific circumstances. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.	
	Type of Contaminant:	Air Speed:
	solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min)
	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)
	Within each range the appropriate value depends on:	
	Lower end of the range	Upper end of the range
	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
	3: Intermittent, low production.	3: High production, heavy use
	4: Large hood or large air mass in motion	4: Small hood-local control only
	Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or	

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	more when extraction systems are installed or used.
Personal protection	
Eye and face protection	▸ Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure. ▸ Chemical goggles whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted. ▸ Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection. ▸ Alternatively a gas mask may replace splash goggles and face shields. ▸ 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	▸ Elbow length PVC gloves NOTE: ▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. 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	▸ 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:

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Material	CPI
NATURAL RUBBER	A
NATURAL+NEOPRENE	A
NEOPRENE	A

Respiratory protection

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

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

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	A-AUS / Class1 P2	-

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NEOPRENE/NATURAL	A
NITRILE	A
NITRILE+PVC	A
PE/EVAL/PE	A
PVC	A
TEFLON	A
PVA	B

* 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.

up to 50	1000	-	A-AUS / Class 1 P2
up to 50	5000	Airline *	-
up to 100	5000	-	A-2 P2
up to 100	10000	-	A-3 P2
100+			Airline**

* - Continuous Flow ** - Continuous-flow or positive pressure demand

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

- ▶ 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 blue liquid with mild sweet odour; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	1.08
Odour	Slight, Sweet	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	~10-11	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	118	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	>1	VOC g/L	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation. The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other
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	route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Exposure to aliphatic alcohols with more than 3 carbons may produce central nervous system effects such as headache, dizziness, drowsiness, muscle weakness, delirium, CNS depression, coma, seizure, and neurobehavioural changes. Symptoms are more acute with higher alcohols. Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, respiratory depression secondary to CNS depression, pulmonary oedema, chemical pneumonitis and bronchitis. Cardiovascular involvement may result in arrhythmias and hypotension. Gastrointestinal effects may include nausea and vomiting. Kidney and liver damage may result following massive exposures. The alcohols are potential irritants being, generally, stronger irritants than similar organic structures that lack functional groups (e.g. alkanes) but are much less irritating than the corresponding amines, aldehydes or ketones. Alcohols and glycols (diols) rarely represent serious hazards in the workplace, because their vapour concentrations are usually less than the levels which produce significant irritation which, in turn, produce significant central nervous system effects as well. Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may produce severely toxic effects; these may be fatal.
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing. The toxic effects of glycols (dihydric alcohols), following ingestion are similar to those of alcohol, with depression of the central nervous system (CNS), nausea, vomiting and degenerative changes in liver and kidney. Effects on the nervous system characterise over-exposure to higher aliphatic alcohols. These include headache, muscle weakness, giddiness, ataxia, (loss of muscle coordination), confusion, delirium and coma. Gastrointestinal effects may include nausea, vomiting and diarrhoea. In the absence of effective treatment, respiratory arrest is the most common cause of death in animals acutely poisoned by the higher alcohols. Aspiration of liquid alcohols produces an especially toxic response as they are able to penetrate deeply in the lung where they are absorbed and may produce pulmonary injury. Those possessing lower viscosity elicit a greater response. The result is a high blood level and prompt death at doses otherwise tolerated by ingestion without aspiration. In general the secondary alcohols are less toxic than the corresponding primary isomers. As a general observation, alcohols are more powerful central nervous system depressants than their aliphatic analogues. In sequence of decreasing depressant potential, tertiary alcohols with multiple substituent OH groups are more potent than secondary alcohols, which, in turn, are more potent than primary alcohols. The potential for overall systemic toxicity increases with molecular weight (up to C7), principally because the water solubility is diminished and lipophilicity is increased. Within the homologous series of aliphatic alcohols, narcotic potency may increase even faster than lethality Only scanty toxicity information is available about higher homologues of the aliphatic alcohol series (greater than C7) but animal data establish that lethality does not continue to increase with increasing chain length. Aliphatic alcohols with 8 carbons are less toxic than those immediately preceding them in the series. 10 -Carbon n-decyl alcohol has low toxicity as do the solid fatty alcohols (e.g. lauryl, myristyl, cetyl and stearyl). However the rat aspiration test suggests that decyl and melted dodecyl (lauryl) alcohols are dangerous if they enter the trachea. In the rat even a small quantity (0.2 ml) of these behaves like a hydrocarbon solvent in causing death from pulmonary oedema. Primary alcohols are metabolised to corresponding aldehydes and acids; a significant metabolic acidosis may occur. Secondary alcohols are converted to ketones, which are also central nervous system depressants and which, in the case of the higher homologues persist in the blood for many hours. Tertiary alcohols are metabolised slowly and incompletely so their toxic effects are generally persistent. 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 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. Fatal poisonings in infants, resulting from ingestion of nitrates in water or spinach, have been reported. 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. for ethylene glycol: Ingestion symptoms include respiratory failure, central nervous depression, cardiovascular collapse, pulmonary oedema, acute kidney failure, and even brain damage. Ingestion of 100 ml has caused death. (ChemInfo) Toxicity of ethylene glycol to human (KB) cell cultures has been reported as less than that of ethanol. (NIOSH/TIC) Ethylene glycol produces a three-stage response with the severity of each stage dependent on the amount of ingestion. Hepatic damage is usually minimal. Central nervous system depression characterise the first 12 hours post ingestion. Transient exhalation occurs without the odour of ethanol. Gastrointestinal complaints include nausea and vomiting. Acidosis, coma, convulsions and myoclonic jerks may also be evident. The optic fundus is usually normal although the presence of papilloedema may confuse the presentation with that produced by methanol. Nystagmus and ophthalmoplegias may appear. Cardiopulmonary effects are seen 12-24 hours post-ingestion and are characterised by tachycardia, tachypnea, and mild hypertension. Congestive heart failure and circulatory collapse may occur in severe intoxications. Renal effects are seen 24-72 hours post-ingestion and are characterised by oliguria, flank pain, acute tubular necrosis, renal failure, and rarely, bone marrow arrest. Renal damage may be permanent. Toxic effects of ethylene glycol are similar to those produced by ethanol but ethylene glycol produces toxic metabolites. Metabolic acidosis and anion gap result primarily from glycolic acid formation and some lactic acid formation. The citric acid cycle is inhibited as a result of reduced NAD/NADH ratios and to a limited extent, the formation of oxalic acid, and to metabolic acidosis. Oxalate formation produces myocardial depression and acute tubular necrosis. Glycoaldehyde, glycolic acid and glyoxylic acid may contribute to CNS depression and may also produce renal toxicity by producing renal oedema. Hypocalcaemia may result from chelation by oxalate. Oxalic acid, glycoxalic acid, glycoaldehyde and formic acid appear to form to only a limited degree during intoxication. Oral administration to pregnant mice and rats produced birth defects amongst the off-spring.
Skin Contact	Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact. Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.

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	Skin contact with the material may produce toxic effects; systemic effects may result following absorption. The material may produce mild skin irritation; limited evidence or practical experience suggests, that the material either: ▶ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or ▶ produces significant, but mild, 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 (non allergic). 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.																
Eye	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.																
Chronic	Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure. Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: - clear evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of: - clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals. 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. 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. 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. 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. 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. NTP Technical Report Series No. 495, May 2001 Under certain conditions, nitrites can react with secondary amines, either alone or in biological systems, to form carcinogenic nitrosamines. 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. Human volunteers exposed to ethylene glycol, 20 to 22 hours/day at mean daily concentrations ranging from 1.4 to 27 ppm for about 4 weeks complained of throat irritation, mild headache and low backache. Complaints became marked when the concentration in the exposure chamber was raised above 56 mg/m³ for part of the day. The most common complaint was irritation of the upper respiratory tract. Concentrations above 80 ppm were intolerable with a burning sensation along the trachea and a burning cough. Excessively exposed workers have reported drowsiness.																
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	Oral (Rat) LD50: >500 mg/kg ^[1]	
sodium nitrite	TOXICITY	IRRITATION
	Inhalation(Rat) LC50: 0.006 mg/L4h ^[2]	Eye (rabbit): 500 mg/24hr - mild
	Oral (Rat) LD50: 180 mg/kg ^[2]	
denatonium benzoate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: adverse effect observed (irreversible damage) ^[1]
	Inhalation(Rat) LC50: 0.2 mg/l4h ^[1]	Skin: adverse effect observed (irritating) ^[1]
	Oral (Rabbit) LD50: 508 mg/kg ^[2]	
Legend:	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

ETHYLENE GLYCOL	[Estimated Lethal Dose (human) 100 ml; RTECS quoted by Orica] Substance is reproductive effector in rats (birth defects). Mutagenic to rat cells. 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. 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). 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. 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. 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. Cancer: No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol. 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.
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POTASSIUM PHOSPHATE, DIBASIC	No significant acute toxicological data identified in literature search.
SODIUM NITRITE	Tumorigenic - Carcinogenic by RTECS criteria. 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 in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies. The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
DENATONIUM BENZOATE	Somnolence, tremor, ataxia recorded. Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production. Most undiluted cationic surfactants satisfy the criteria for classification as Harmful (Xn) with R22 and as Irritant (Xi) for skin and eyes with R38 and R41. For quaternary ammonium compounds (QACs): Quaternary ammonium compounds (QACs) are cationic surfactants. They are synthetic organically tetra-substituted ammonium compounds, where the R substituents are alkyl or heterocyclic radicals (where hydrogen atoms remain unsubstituted, the term "secondary- or "tertiary-ammonium compounds" is preferred) . A common characteristic of these synthetic compounds is that one of the R's is a long-chain hydrophobic aliphatic residue The cationic surface active compounds are in general more toxic than the anionic and non-ionic surfactants. The positively-charged cationic portion is the functional part of the molecule and the local irritation effects of QACs appear to result from the quaternary ammonium cation. Due to their relative ability to solubilise phospholipids and cholesterol in lipid membranes, QACs affect cell permeability which may lead to cell death. Further QACs denature proteins as cationic materials precipitate protein and are accompanied by generalised tissue irritation. It has been suggested that the experimentally determined decrease in acute toxicity of QACs with chain lengths above C16 is due to decreased water solubility. In general it appears that QACs with a single long-chain alkyl groups are more toxic and irritating than those with two such substitutions, The straight chain aliphatic QACs have been shown to release histamine from minced guinea pig lung tissue However, studies with benzalkonium chloride have shown that the effect on histamine release depends on the concentration of the solution. When cell suspensions (11% mast cells) from rats were exposed to low concentrations, a decrease in histamine release was seen. When exposed to high concentrations the opposite result was obtained. In addition, QACs may show curare-like properties (specifically benzalkonium and cetylpyridinium derivatives, a muscular paralysis with no involvement of the central nervous system. This is most often associated with lethal doses Parenteral injections in rats, rabbits and dogs have resulted in prompt but transient limb paralysis and sometimes fatal paresis of the respiratory muscles. This effect seems to be transient. From human testing of different QACs the generalised conclusion is obtained that all the compounds investigated to date exhibit similar toxicological properties. Acute toxicity: Studies in rats have indicated poor intestinal absorption of QACs. Acute toxicity of QACs varies with the compound and, especially, the route of administration. For some substances the LD50 value is several hundreds times lower by the i.p. or i.v. than the oral route, whereas toxicities between the congeners only differ in the range of two to five times. At least some QACs are significantly more toxic in 50% dimethyl sulfoxide than in plain water when given orally Probably all common QAC derivatives produce similar toxic reactions, but as tested in laboratory animals the oral mean lethal dose varies with the compound . Oral toxicity: LD50 values for QACs have been reported within the range of 250-1000 mg/kg for rats, 150-1000 mg/kg for mice, 150-300 mg/kg for guinea pigs and about 500 mg/kg b.w. for rabbits and dogs . The ranges observed reflect differences in the study designs of these rather old experiments as well as differences between the various QACs. The oral route of administration was characterised by delayed deaths, gastrointestinal lesions and respiratory and central nervous system depression. It was also found that given into a full stomach, the QACs lead to lower mortality and fewer gastrointestinal symptoms. This support the suggestion of an irritating effect Dermal toxicity: It has been concluded that the maximum concentration that did not produce irritating effect on intact skin is 0.1%. Irritation became manifest in the 1-10% range. Concentrations below 0.1% have caused irritation in persons with contact dermatitis or broken skin. Although the absorption of QACs through normal skin probably is of less importance than by other routes , studies with excised guinea pig skin have shown that the permeability constants strongly depends on the exposure time and type of skin Sensitisation: Topical mucosal application of QACs may produce sensitisation. Reports on case stories and patch test have shown that compounds such as benzalkonium chloride , cetalkonium chloride and cetrimide may possibly act as sensitisers . However, in general it is suggested that QACs have a low potential for sensitising man It is difficult to distinguish between an allergic and an irritative skin reaction due to the inherent skin irritating effect of QACs. Long term/repeated exposure: Inhalation: A group of 196 farmers (with or without respiratory symptoms) were evaluated for the relationship between exposure to QACs (unspecified, exposure levels not given) and respiratory disorders by testing for lung function and bronchial responsiveness to histamine. After histamine provocation statistically significant associations were found between the prevalence of mild bronchial responsiveness (including asthma-like symptoms) and the use of QACs as disinfectant. The association seems even stronger in people without respiratory symptoms. Genetic toxicity: QACs have been investigated for mutagenicity in microbial test systems. In Ames tests using Salmonella typhimurium with and without metabolic activation no signs of mutagenicity has been observed. Negative results were also obtained in E. coli reversion and B. subtilis rec assays. However, for benzalkonium chloride also positive and equivocal results were seen in the B. subtilis rec assays.

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

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

SECTION 12 Ecological information

Toxicity

Continued...

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Graco Pump Armour	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
ethylene glycol	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	8050mg/l	4
	EC50	48h	Crustacea	>100mg/l	2
	EC50(ECx)	Not Available	Algae or other aquatic plants	6500-7500mg/l	1
	EC50	96h	Algae or other aquatic plants	6500-13000mg/l	1
potassium phosphate, dibasic	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	96h	Fish	100mg/l	2
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	LC50	96h	Fish	>100mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
sodium nitrite	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	672h	Fish	0.01mg/l	4
	EC50	96h	Algae or other aquatic plants	1600mg/l	4
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	LC50	96h	Fish	0.00016mg/l	4
	EC50	48h	Crustacea	ca.12.51mg/l	1
denatonium benzoate	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	>100mg/l	2
	NOEC(ECx)	48h	Crustacea	50mg/l	2
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	EC50	48h	Crustacea	>500mg/l	2
Legend:	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data				

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

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.

Methaemoglobinaemia 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.

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.

for ethylene glycol:

log Kow : -1.93- -1.36

Half-life (hr) air : 24

Henry's atm m3 /mol: 6.00E-08

BOD 5 : 0.15-0.81,12%

COD : 1.21-1.29

ThOD : 1.26

BCF : 10-190

In the atmosphere ethylene glycol exists mainly in the vapour phase. It is degraded in the atmosphere by reaction with photochemically produced hydroxy radicals (estimated half-life 24-50 hours).

Ethylene glycol does not concentrate in the food chain.

Environmental fate:

Ethylene glycol has a low vapour pressure (7.9 Pa at 20 C); it is expected to exist almost entirely in the vapour phase if released to the atmosphere. The Henry's law constant for ethylene glycol is 1.41×10^{-3} or 6.08×10^{-3} Pa.m³/mol, depending on method of calculation, indicating a low capacity for volatilisation from water bodies or soil surfaces.

Ethylene glycol adsorbed onto silica gel and irradiated with light (wavelength >290 nm) degraded by 12.1% over 17 h . Photodegradation is not expected, as the molecule should not absorb at these wavelengths; the mechanism of this breakdown is, therefore, unknown. Estimated half-life in the atmosphere for reaction with hydroxyl radicals from various reports is 2.1 days , 8-84 h or 1 day.

Ethylene glycol released to the atmosphere will be degraded by reaction with hydroxyl radicals; the half-life for the compound in this reaction has been estimated at between 0.3 and 3.5 days. No hydrolysis of ethylene glycol is expected in surface waters.

The compound has little or no capacity to bind to particulates and will be mobile in soil. Soil partition coefficients (log Koc) of 0-0.62 were determined. Migration rates in five soil types were measured at between 4 and 27 cm per 12 h

The low octanol/water partition coefficient (log Kow -1.93 to -1.36)and measured bioconcentration factors in a few organisms indicate low capacity for bioaccumulation.

Bioconcentration factors of 190 for the green algae (Chlorella fusca) , up to 0.27 in specific tissues of the crayfish (Procambarus sp.) , and 10 for the golden orfe (Leuciscus idus melanotus) confirm low bioaccumulation.

Ethylene glycol is readily biodegradable in standard tests using sewage sludge. Many studies show biodegradation under both aerobic and anaerobic conditions. Some studies suggest a lag phase before degradation, but many do not. Degradation occurs in both adapted and unadapted sludges. Rapid degradation has been reported in surface waters (less in salt water than in fresh water), groundwater, and soil inocula. Several strains of microorganisms capable of utilising ethylene glycol as a carbon source have been identified.

Ethylene glycol has been identified as a metabolite of the growth regulator ethylene in a number of higher plants and as naturally occurring in the edible fungus Tricholoma matsutake

Ecotoxicity:

Fish LC50 (96 h):118-550 mg/L

Ethylene glycol has generally low toxicity to aquatic organisms. Toxic thresholds for microorganisms are above 1000 mg/litre. EC50s for growth in microalgae are 6500 mg/litre or

Continued...

Graco Pump Armour

higher. Acute toxicity tests with aquatic invertebrates where a value could be determined show LC50s above 20 000 mg/litre, and those with fish show LC50s above 17 800 mg/litre. An amphibian test showed an LC50 for tadpoles at 17 000 mg/litre. A no-observed-effect concentration (NOEC) for chronic tests on daphnids of 8590 mg/litre (for reproductive end-points) has been reported. A NOEC following short-term exposure of fish has been reported at 15 380 mg/litre for growth. Tests using deicer containing ethylene glycol showed greater toxicity to aquatic organisms than observed with the pure compound, indicating other toxic components of the formulations. Laboratory tests exposing aquatic organisms to stream water receiving runoff from airports have demonstrated toxic effects and death. Field studies in the vicinity of an airport have reported toxic signs consistent with ethylene glycol poisoning, fish kills, and reduced biodiversity. These effects cannot definitively be ascribed to ethylene glycol. Terrestrial organisms are much less likely to be exposed to ethylene glycol and generally show low sensitivity to the compound. Concentrations above 100 000 mg/litre were needed to produce toxic effects on yeasts and fungi from soil. Very high concentrations and soaking of seeds produced inhibition of germination in some experiments; these are not considered of environmental significance. A no-observed-effect level (NOEL) for orally dosed ducks at 1221 mg/kg body weight and reported lethal doses for poultry at around 8000 mg/kg body weight indicate low toxicity to birds.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ethylene glycol	LOW (Half-life = 24 days)	LOW (Half-life = 3.46 days)
sodium nitrite	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
ethylene glycol	LOW (BCF = 200)
sodium nitrite	LOW (LogKOW = 0.0564)

Mobility in soil

Ingredient	Mobility
ethylene glycol	HIGH (KOC = 1)
sodium nitrite	LOW (KOC = 23.74)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	- Containers may still present a chemical hazard/ danger when empty. - Return to supplier for reuse/ recycling if possible. Otherwise: - If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. - Where possible retain label warnings and SDS and observe all notices pertaining to the product. 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. A Hierarchy of Controls seems to be common - the user should investigate: - Reduction - Reuse - Recycling - Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. DO NOT allow wash water from cleaning or process equipment to enter drains. - It may be necessary to collect all wash water for treatment before disposal. - In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. - Where in doubt contact the responsible authority. - Recycle wherever possible or consult manufacturer for recycling options. - Consult State Land Waste Authority for disposal. - Bury or incinerate residue at an approved site. - Recycle containers if possible, or dispose of in an authorised landfill.
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Ensure that the hazardous substance is disposed in accordance with the Hazardous Substances (Disposal) Notice 2017

Disposal Requirements

Packages that have been in direct contact with the hazardous substance must be only disposed if the hazardous substance was appropriately removed and cleaned out from the package. The package must be disposed according to the manufacturer's directions taking into account the material it is made of. Packages which hazardous content have been appropriately treated and removed may be recycled.

The hazardous substance must only be disposed if it has been treated by a method that changed the characteristics or composition of the substance and it is no longer hazardous. Only dispose to the environment if a tolerable exposure limit has been set for the substance.

Only deposit the hazardous substance into or onto a landfill or sewage facility or incinerator, where the hazardous substance can be handled and treated appropriately.

SECTION 14 Transport information

Labels Required

	
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Marine Pollutant	
HAZCHEM	2X

Land transport (UN)

UN number	3287	
UN proper shipping name	TOXIC LIQUID, INORGANIC, N.O.S. (contains sodium nitrite)	
Transport hazard class(es)	Class	6.1
	Subrisk	Not Applicable
Packing group	III	
Environmental hazard	Environmentally hazardous	
Special precautions for user	Special provisions	223; 274
	Limited quantity	5 L

Air transport (ICAO-IATA / DGR)

UN number	3287	
UN proper shipping name	Toxic liquid, inorganic, n.o.s. * (contains sodium nitrite)	
Transport hazard class(es)	ICAO/IATA Class	6.1
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	6L
Packing group	III	
Environmental hazard	Environmentally hazardous	
Special precautions for user	Special provisions	A3 A4 A137
	Cargo Only Packing Instructions	663
	Cargo Only Maximum Qty / Pack	220 L
	Passenger and Cargo Packing Instructions	655
	Passenger and Cargo Maximum Qty / Pack	60 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y642
	Passenger and Cargo Limited Maximum Qty / Pack	2 L

Sea transport (IMDG-Code / GGVSee)

UN number	3287	
UN proper shipping name	TOXIC LIQUID, INORGANIC, N.O.S. (contains sodium nitrite)	
Transport hazard class(es)	IMDG Class	6.1
	IMDG Subrisk	Not Applicable
Packing group	III	
Environmental hazard	Marine Pollutant	
Special precautions for user	EMS Number	F-A, S-A
	Special provisions	223 274
	Limited Quantities	5 L

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

Not Applicable

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

Product name	Group
ethylene glycol	Not Available
potassium phosphate, dibasic	Not Available
sodium nitrite	Not Available
denatonium benzoate	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
ethylene glycol	Not Available
potassium phosphate, dibasic	Not Available

Product name	Ship Type
sodium nitrite	Not Available
denatonium benzoate	Not Available

SECTION 15 Regulatory information

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

This substance is to be managed using the conditions specified in an applicable Group Standard

HSR Number	Group Standard
HSR002521	Animal Nutritional and Animal Care Products Group Standard 2020
HSR002530	Cleaning Products Subsidiary Hazard Group Standard 2020
HSR002535	Gases under Pressure Mixtures Subsidiary Hazard Group Standard 2020
HSR002503	Additives Process Chemicals and Raw Materials Subsidiary Hazard Group Standard 2020
HSR002606	Lubricants Lubricant Additives Coolants and Anti freeze Agents Subsidiary Hazard Group Standard 2020
HSR002612	Metal Industry Products Subsidiary Hazard Group Standard 2020
HSR002624	N.O.S. Subsidiary Hazard Group Standard 2020
HSR002638	Photographic Chemicals Subsidiary Hazard Group Standard 2020
HSR002644	Polymers Subsidiary Hazard Group Standard 2020
HSR002647	Reagent Kits Group Standard 2020
HSR002648	Refining Catalysts Group Standard 2020
HSR002653	Solvents Subsidiary Hazard Group Standard 2020
HSR002670	Surface Coatings and Colourants Subsidiary Hazard Group Standard 2020
HSR002684	Water Treatment Chemicals Subsidiary Hazard Group Standard 2020
HSR100425	Pharmaceutical Active Ingredients Group Standard 2020
HSR002600	Leather and Textile Products Subsidiary Hazard Group Standard 2020
HSR002544	Construction Products Subsidiary Hazard Group Standard 2020
HSR002549	Corrosion Inhibitors Subsidiary Hazard Group Standard 2020
HSR002552	Cosmetic Products Group Standard 2020
HSR002558	Dental Products Subsidiary Hazard Group Standard 2020
HSR002565	Embalming Products Subsidiary Hazard Group Standard 2020
HSR002571	Fertilisers Subsidiary Hazard Group Standard 2020
HSR002573	Fire Fighting Chemicals Group Standard 2021
HSR002578	Food Additives and Fragrance Materials Subsidiary Hazard Group Standard 2020
HSR002585	Fuel Additives Subsidiary Hazard Group Standard 2020
HSR002596	Laboratory Chemicals and Reagent Kits Group Standard 2020
HSR100757	Veterinary Medicines Limited Pack Size Finished Dose Group Standard 2020
HSR100758	Veterinary Medicines Non dispersive Closed System Application Group Standard 2020
HSR100759	Veterinary Medicines Non dispersive Open System Application Group Standard 2020
HSR100592	Agricultural Compounds Special Circumstances Group Standard 2020
HSR100756	Active Ingredients for Use in the Manufacture of Agricultural Compounds Group Standard 2020

Please refer to Section 8 of the SDS for any applicable tolerable exposure limit or Section 12 for environmental exposure limit.

ethylene glycol is found on the following regulatory lists

- Chemical Footprint Project - Chemicals of High Concern List
- New Zealand Approved Hazardous Substances with controls
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
- New Zealand Inventory of Chemicals (NZIoC)
- New Zealand Workplace Exposure Standards (WES)

potassium phosphate, dibasic is found on the following regulatory lists

- New Zealand Approved Hazardous Substances with controls
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
- New Zealand Inventory of Chemicals (NZIoC)

sodium nitrite is found on the following regulatory lists

- International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
- International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2A: Probably carcinogenic to humans
- New Zealand Approved Hazardous Substances with controls

- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
- New Zealand Inventory of Chemicals (NZIoC)

denatonium benzoate is found on the following regulatory lists

- New Zealand Approved Hazardous Substances with controls
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
- New Zealand Inventory of Chemicals (NZIoC)

Hazardous Substance Location

Subject to the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Quantities
Not Applicable	Not Applicable

Certified Handler

Subject to Part 4 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Class of substance	Quantities
Not Applicable	Not Applicable

Refer Group Standards for further information

Maximum quantities of certain hazardous substances permitted on passenger service vehicles

Subject to Regulation 13.14 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Gas (aggregate water capacity in mL)	Liquid (L)	Solid (kg)	Maximum quantity per package for each classification
Not Applicable	Not Applicable	Not Applicable	Not Applicable	Not Applicable

Tracking Requirements

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (ethylene glycol; potassium phosphate, dibasic; sodium nitrite; denatonium benzoate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (denatonium benzoate)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	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. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	06/02/2023
Initial Date	10/01/2007

SDS Version Summary

Version	Date of Update	Sections Updated
12.1	20/08/2021	Classification change due to full database hazard calculation/update.
13.1	06/02/2023	Supplier Information, Synonyms, Transport Information, Use, Name

Other information

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

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

Definitions and abbreviations

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

LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index
AIC: Australian Inventory of Industrial Chemicals
DSL: Domestic Substances List
NDSL: Non-Domestic Substances List
IECSC: Inventory of Existing Chemical Substance in China
EINECS: European INventory of Existing Commercial chemical Substances
ELINCS: European List of Notified Chemical Substances
NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory
KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals
PICCS: Philippine Inventory of Chemicals and Chemical Substances
TSCA: Toxic Substances Control Act
TCSI: Taiwan Chemical Substance Inventory
INSQ: Inventario Nacional de Sustancias Químicas
NCI: National Chemical Inventory
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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