Folk Art Dragonfly Paints

Jasco Pty Limited

Chemwatch Hazard Alert Code: 3

Chemwatch: **7912-61**
Version No: **3.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Issue Date: **04/10/2024**
Print Date: **04/10/2024**
L.GHS.AUS.EN.E

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

Product Identifier

Product name	Folk Art Dragonfly Paints
Chemical Name	Not Applicable
Synonyms	Not Available
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Use according to manufacturer's directions.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**Jasco Pty Limited**
Address	1-5 Commercial Road Kingsgrove NSW 2208 Australia
Telephone	+61 2 9807 1555
Fax	Not Available
Website	www.jasco.com.au
Email	quickinfo@jasco.com.au

Emergency telephone number

Association / Organisation	**Australian Poisons Centre**
Emergency telephone numbers	**13 11 26 (24/7)**
Other emergency telephone numbers	**Not Available**

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Serious Eye Damage/Eye Irritation Category 2A, Germ Cell Mutagenicity Category 2, Carcinogenicity Category 1A, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI***

Label elements

Hazard pictogram(s)	
Signal word	**Danger**

Hazard statement(s)

H319	Causes serious eye irritation.
H341	Suspected of causing genetic defects.
H350	May cause cancer.
H412	Harmful to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

Page 1 continued...

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.
P337+P313	If eye irritation persists: Get medical advice/attention.

Precautionary statement(s) Storage

P405	Store locked up.

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
57-55-6	1-10	propylene glycol
34590-94-8	1-10	dipropylene glycol monomethyl ether
78330-21-9	1-10	alcohols C11-14-iso-, C13-rich, ethoxylated
13463-67-7	1-10	titanium dioxide
25322-69-4	<1	polypropylene glycol
127087-87-0	<1	4-nonylphenol, branched, ethoxylated
577-11-7	<1	sodium dioctyl sulfosuccinate
13463-41-7	<1	zinc pyrithione
124-68-5	<1	monoisobutanolamine
Not Available	balance	Ingredients determined not to be hazardous
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available***	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ 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	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary.
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.

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.
Treat symptomatically.
To treat poisoning by the higher aliphatic alcohols (up to C7):

- Gastric lavage with copious amounts of water.
- It may be beneficial to instill 60 ml of mineral oil into the stomach.
- Oxygen and artificial respiration as needed.
- Electrolyte balance: it may be useful to start 500 ml. M/6 sodium bicarbonate intravenously but maintain a cautious and conservative attitude toward electrolyte replacement unless shock or severe acidosis threatens.
- To protect the liver, maintain carbohydrate intake by intravenous infusions of glucose.
- Haemodialysis if coma is deep and persistent. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products, Ed 5)

BASIC TREATMENT

- Establish a patent airway with suction where necessary.
- Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- Monitor and treat, where necessary, for shock.
- Monitor and treat, where necessary, for pulmonary oedema.
- Anticipate and treat, where necessary, for seizures.
- **DO NOT** use emetics. Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.

Continued...

- Give activated charcoal.

ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- If the patient is hypoglycaemic (decreased or loss of consciousness, tachycardia, pallor, dilated pupils, diaphoresis and/or dextrose strip or glucometer readings below 50 mg), give 50% dextrose.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Treat seizures with diazepam.
- Proparacaine hydrochloride should be used to assist eye irrigation.

EMERGENCY DEPARTMENT

- Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- Acidosis may respond to hyperventilation and bicarbonate therapy.
- Haemodialysis might be considered in patients with severe intoxication.
- Consult a toxicologist as necessary. BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

For C8 alcohols and above.
Symptomatic and supportive therapy is advised in managing patients.

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

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 (CO_2) nitrogen oxides (NOx) sulfur oxides (SOx) metal oxides other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.
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	▸ Remove all ignition sources. ▸ 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	Chemical Class: alcohols and glycols For release onto land: recommended sorbents listed in order of priority. SORBENT TYPE | RANK | APPLICATION | COLLECTION | LIMITATIONS

Continued...

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
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
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

- Store in original containers.
- Keep containers securely sealed.
- No smoking, naked lights or ignition sources.
- 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

- Metal can or drum
- Packaging as recommended by manufacturer.
- Check all containers are clearly labelled and free from leaks.

Storage incompatibility

For frits:

- Avoid storage with hydrogen fluoride/ hydrofluoric acid, oxygen difluoride, manganese trifluoride, fluorine and other fluorine containing compounds, manganese trioxide, chlorates, chlorine trifluoride, chlorine trioxide, strong alkalis, metal oxides, concentrated orthophosphoric acid or vinyl acetate.
- 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.

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

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- 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

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	propylene glycol	Propane-1,2-diol: particulates only	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	propylene glycol	Propane-1,2-diol total: (vapour & particulates)	150 ppm / 474 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	dipropylene glycol monomethyl ether	(2-Methoxymethylethoxy) propanol	50 ppm / 308 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	titanium dioxide	Titanium dioxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.

Ingredient	Original IDLH	Revised IDLH
propylene glycol	Not Available	Not Available
dipropylene glycol monomethyl ether	600 ppm	Not Available
alcohols C11-14-iso-, C13-rich, ethoxylated	Not Available	Not Available
titanium dioxide	5,000 mg/m3	Not Available
polypropylene glycol	Not Available	Not Available
4-nonylphenol, branched, ethoxylated	Not Available	Not Available
sodium dioctyl sulfosuccinate	Not Available	Not Available
zinc pyrithione	Not Available	Not Available
monoisobutanolamine	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
alcohols C11-14-iso-, C13-rich, ethoxylated	E	≤ 0.1 ppm
4-nonylphenol, branched, ethoxylated	E	≤ 0.1 ppm
sodium dioctyl sulfosuccinate	E	≤ 0.01 mg/m³
zinc pyrithione	E	≤ 0.01 mg/m³
monoisobutanolamine	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.

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.
An approved self contained breathing apparatus (SCBA) may be required in some situations.
Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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:

Continued...

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.

Individual protection measures, such as personal protective equipment

Eye and face protection

- Safety glasses with side shields.
- Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].

Skin protection

See Hand protection below

Hands/feet protection

- Wear chemical protective gloves, e.g. PVC.
- Wear safety footwear or safety gumboots, e.g. Rubber

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:
Folk Art Dragonfly Paints

Material	CPI

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.

Continued...

PE/EVAL/PE	A

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Ansell Glove Selection

Glove *— In order of recommendation*
AlphaTec® Solvex® 37-675
MICROFLEX® 93-260
AlphaTec® 15-554
AlphaTec® Solvex® 37-185
AlphaTec® 38-612
AlphaTec® 58-008
AlphaTec® 58-530B
AlphaTec® 58-530W
AlphaTec® 58-735
AlphaTec® 79-700

The suggested gloves for use should be confirmed with the glove supplier.

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	Liquid.

Physical state	Liquid	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**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)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Not Available	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available
Heat of Combustion (kJ/g)	Not Available	**Ignition Distance (cm)**	Not Available
Flame Height (cm)	Not Available	**Flame Duration (s)**	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	**Enclosed Space Ignition Deflagration Density (g/m3)**	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

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Information on toxicological effects

Inhaled	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 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.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. 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 he 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. Ingestion of propylene glycol produced reversible central nervous system depression in humans following ingestion of 60 ml. Symptoms included increased heart-rate (tachycardia), excessive sweating (diaphoresis) and grand mal seizures in a 15 month child who ingested large doses (7.5 ml/day for 8 days) as an ingredient of vitamin preparation. Excessive repeated ingestions may cause hypoglycaemia (low levels of glucose in the blood stream) among susceptible individuals; this may result in muscular weakness, incoordination and mental confusion. Very high doses given during feeding studies to rats and dogs produce central nervous system depression (although one-third of that produced by ethanol), haemolysis and insignificant kidney changes. In humans propylene glycol is partly excreted unchanged in the urine and partly metabolised as lactic and pyruvic acid. Lactic acidosis may result.
Skin Contact	Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions. Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. 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 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. Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man.
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 temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	On the basis of epidemiological data, it has been concluded that prolonged inhalation of the material, in an occupational setting, may produce cancer in humans. Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure. Harmful: 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. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects. Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects. 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. Propylene glycol is though, by some, to be a sensitising principal following the regular use of topical creams by eczema patients. A study of 866 persons using a formulation containing propylene glycol in a patch test indicated that propylene glycol caused primary irritation in 16% of exposed individuals probably caused by dehydration. Undiluted propylene glycol was tested on 1556 persons in a 24 hour patch test. 12.5% showed reactions which were largely toxic (70%) or allergic in nature (30%). Reaction responses reached their maximum on the second day or later. Reactions were seasonal in nature ranging from 17.8% in winter to 9.2% in other seasons. In a patch-test using 25 standard allergens conducted on 500 individuals, propylene glycol ranked fourth in sensitising response. 84 subjects were patch tested using 100%

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propylene glycol. as well as 2% and 5% in water. With undiluted material, 15% demonstrated a reaction, with 40% of the reactions being allergic in nature and 60% being irritant. In dilute solutions 5 of 248 subjects exhibited a reaction.
Undiluted propylene glycol tested on the skin of man produced no irritation under open conditions but when applied under occlusive conditions, for 2 weeks, it produced severe erythema, oedema and vesicles, probably due to sweat retention and weak primary irritation. Predictive contact skin sensitisation tests indicate that propylene glycol is an intermediate grade sensitiser with an index of 1% of tested subjects.
Groups of cats fed 5 gm/kg/day of propylene glycol for 14 weeks showed a significant dose-related increase in red blood cell Heinz body formation without any marked signs of haemolytic anaemia. The no-effect-level for cats without formation of Heinz bodies is 100-500 ml/kg. There is no evidence of anaemia or degenerative change. Groups of rats dosed orally with 0.5 or 10 mg/kg/day for 12 weeks had lowered food intake but no adverse effects on body weights. Erythrocytes were more fragile. Heinz bodies were not apparent.

Folk Art Dragonfly Paints

TOXICITY	IRRITATION
Not Available	Not Available

propylene glycol

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 11890 mg/kg[2]	Eye (rabbit): 100 mg - mild
Inhalation (Rat) LC50: >44.9 mg/l4h[1]	Eye (rabbit): 500 mg/24h - mild
Oral (Rat) LD50: 20000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Skin(human):104 mg/3d Intermit Mod
	Skin(human):500 mg/7days mild
	Skin: no adverse effect observed (not irritating)[1]

dipropylene glycol monomethyl ether

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 9500 mg/kg[2]	Eye (human): 8 mg - mild
Oral (Rat) LD50: 5135 mg/kg[2]	Eye (rabbit): 500 mg/24hr - mild
	Eye: no adverse effect observed (not irritating)[1]
	Skin (rabbit): 238 mg - mild
	Skin (rabbit): 500 mg (open)-mild
	Skin: no adverse effect observed (not irritating)[1]

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

TOXICITY	IRRITATION
Oral (Rat) LD50: 500 mg/kg[2]	Not Available

titanium dioxide

TOXICITY	IRRITATION
dermal (hamster) LD50: >=10000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
Inhalation (Rat) LC50: >2.28 mg/l4h[1]	Skin (human): 0.3 mg /3D (int)-mild *
Oral (Rat) LD50: >=2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

polypropylene glycol

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 500 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
Inhalation (Rat) LC50: >2.34 mg/l4h[1]	Skin (rabbit): 500 mg mild
Oral (Rat) LD50: >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

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]

sodium dioctyl sulfosuccinate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 2525 mg/kg[1]	Eye (rabbit): 0.250 mg - mild
Oral (Rat) LD50: >1320 mg/kg[1]	Eye (rabbit): 1% - SEVERE
	Eye: adverse effect observed (irritating)[1]
	Skin (rabbit): 10 mg/24h-moderate
	Skin: adverse effect observed (irritating)[1]

zinc pyrithione

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 100 mg/kg[2]	Eye (rabbit): 1 mg/48h Irritant
Inhalation (Rat) LC50: 0.14 mg/L4h[2]	Eye: adverse effect observed (irritating)[1]
Oral (Mouse) LD50; 160 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]

monoisobutanolamine

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye: adverse effect observed (irreversible damage)[1]

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	Oral (Mouse) LD50; 2150 mg/kg[2]	Skin: adverse effect observed (irritating)[1]

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***

PROPYLENE GLYCOL	The acute oral toxicity of propylene glycol is very low, and large quantities are required to cause perceptible health damage in humans. Serious toxicity generally occurs only at plasma concentrations over 1 g/L, which requires extremely high intake over a relatively short period of time. It would be nearly impossible to reach toxic levels by consuming foods or supplements, which contain at most 1 g/kg of PG. Cases of propylene glycol poisoning are usually related to either inappropriate intravenous administration or accidental ingestion of large quantities by children.The potential for long-term oral toxicity is also low. Because of its low chronic oral toxicity, propylene glycol was classified by the U. S. Food and Drug Administration as "generally recognized as safe" (GRAS) for use as a direct food additive. Prolonged contact with propylene glycol is essentially non-irritating to the skin. Undiluted propylene glycol is minimally irritating to the eye, and can produce slight transient conjunctivitis (the eye recovers after the exposure is removed). Exposure to mists may cause eye irritation, as well as upper respiratory tract irritation. Inhalation of the propylene glycol vapours appears to present no significant hazard in ordinary applications. However, limited human experience indicates that inhalation of propylene glycol mists could be irritating to some individuals It is therefore recommended that propylene glycol not be used in applications where inhalation exposure or human eye contact with the spray mists of these materials is likely, such as fogs for theatrical productions or antifreeze solutions for emergency eye wash stations. Propylene glycol is metabolised in the human body into pyruvic acid (a normal part of the glucose-metabolism process, readily converted to energy), acetic acid (handled by ethanol-metabolism), lactic acid (a normal acid generally abundant during digestion), and propionaldehyde (a potentially hazardous substance). Propylene glycol shows no evidence of being a carcinogen or of being genotoxic. Research has suggested that individuals who cannot tolerate propylene glycol probably experience a special form of irritation, but that they only rarely develop allergic contact dermatitis. Other investigators believe that the incidence of allergic contact dermatitis to propylene glycol may be greater than 2% in patients with eczema. One study strongly suggests a connection between airborne concentrations of propylene glycol in houses and development of asthma and allergic reactions, such as rhinitis or hives in children Another study suggested that the concentrations of PGEs (counted as the sum of propylene glycol and glycol ethers) in indoor air, particularly bedroom air, is linked to increased risk of developing numerous respiratory and immune disorders in children, including asthma, hay fever, eczema, and allergies, with increased risk ranging from 50% to 180%. This concentration has been linked to use of water-based paints and water-based system cleansers. Patients with vulvodynia and interstitial cystitis may be especially sensitive to propylene glycol. Women suffering with yeast infections may also notice that some over the counter creams can cause intense burning. Post menopausal women who require the use of an eostrogen cream may notice that brand name creams made with propylene glycol often create extreme, uncomfortable burning along the vulva and perianal area. Additionally, some electronic cigarette users who inhale propylene glycol vapor may experience dryness of the throat or shortness of breath . As an alternative, some suppliers will put Vegetable Glycerin in the "e-liquid" for those who are allergic (or have bad reactions) to propylene glycol. Adverse responses to intravenous administration of drugs which use PG as an excipient have been seen in a number of people, particularly with large dosages thereof. Responses may include "hypotension, bradycardia... QRS and T abnormalities on the ECG, arrhythmia, cardiac arrest, serum hyperosmolality, lactic acidosis, and haemolysis". A high percentage (12% to 42%) of directly-injected propylene glycol is eliminated/secreted in urine unaltered depending on dosage, with the remainder appearing in its glucuronide-form. The speed of renal filtration decreases as dosage increases, which may be due to propylene glycol's mild anesthetic / CNS-depressant -properties as an alcohol. In one case, intravenous administration of propylene glycol-suspended nitroglycerin to an elderly man may have induced coma and acidosis. Propylene glycol is an approved food additive for dog food under the category of animal feed and is generally recognized as safe for dogs with an LD50 of 9 mL/kg. The LD50 is higher for most laboratory animals (20 mL/kg) Similarly, propylene glycol is an approved food additive for human food as well. The exception is that it is prohibited for use in food for cats due to links to Heinz body anemia.
DIPROPYLENE GLYCOL MONOMETHYL ETHER	for propylene glycol ethers (PGEs): Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM). Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids. Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product. Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body. As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces. As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating None are skin sensitisers. In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested). Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members.

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	One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health. In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity. The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. ***In vitro***, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic ***in vivo***. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.
ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED	* Ashland SDS The material may produce respiratory tract irritation. Symptoms of pulmonary irritation may include coughing, wheezing, laryngitis, shortness of breath, headache, nausea, and a burning sensation. Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence). The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties.
TITANIUM DIOXIDE	* IUCLID 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. For titanium dioxide: Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin. Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica. No data were available on genotoxic effects in titanium dioxide-exposed humans. Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts. Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium. Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light. **Animal carcinogenicity data** Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats. In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative. Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice. In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative. No significant acute toxicological data identified in literature search. The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
POLYPROPYLENE GLYCOL	** Rohm and Haas Paraplex WP-1 MSDS
4-NONYLPHENOL, BRANCHED, ETHOXYLATED	for linear material: Maternal effects, effects on fertility recorded. For nonylphenol and its compounds: Alkylphenols like nonylphenol and bisphenol A have estrogenic effects in the body. They are known as xenoestrogens. Estrogenic substances and other endocrine disruptors are compounds that have hormone-like effects in both wildlife and humans. Xenoestrogens usually function by binding to estrogen receptors and acting competitively against natural estrogens. Nonylphenol has been found to act as an agonist of GPER (G protein-coupled estrogen receptor),. Nonylphenol has been shown to mimic the natural hormone 17beta-estradiol, and it competes with the endogeous hormone for binding with the estrogen receptors ERalpha and ERbeta. Effects in pregnant women.

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	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. Nonylphenol has also been shown to affect cytokine signaling molecule secretions in the human placenta. In vitro cell cultures of human placenta during the first trimester were treated with nonylphenol, which increase the secretion of cytokines including interferon gamma, interleukin 4, and interleukin 10, and reduced the secretion of tumor necrosis factor alpha. This unbalanced cytokine profile at this part of pregnancy has been documented to result in implantation failure, pregnancy loss, and other complications. Effects on metabolism 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 mimic the action of leptin on neuropeptide Y and anorectic POMC neurons, which has an anti-obesity effect by decreasing eating behavior. This was seen when estrogen or estrogen mimics were injected into the ventromedial hypothalamus. On the other hand, nonylphenol has been shown to increase food intake and have obesity enhancing properties by lowering the expression of these anorexigenic neurons in the brain. Additionally, nonylphenol affects the expression of ghrelin: an enzyme produced by the stomach that stimulates appetite. Ghrelin expression is positively regulated by estrogen signaling in the stomach, and it is also important in guiding the differentiation of stem cells into adipocytes (fat cells). Thus, acting as an estrogen mimic, prenatal and perinatal exposure to nonylphenol has been shown to increase appetite and encourage the body to store fat later in life. Finally, long-term exposure to nonylphenol has been shown to affect insulin signaling in the liver of adult male rats. Cancer Nonylphenol exposure has also been associated with breast cancer. It has been shown to promote the proliferation of breast cancer cells, due to its agonistic activity on ERalpha (estrogen receptor alpha) in estrogen-dependent and estrogen-independent breast cancer cells. Some argue that nonylphenol's suggested estrogenic effect coupled with its widespread human exposure could potentially influence hormone-dependent breast cancer disease for 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 exogeneous 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.
SODIUM DIOCTYL SULFOSUCCINATE	Structural changes in blood vessels recorded. for dialkyl sodium sulfosuccinates: The existing data on diethylhexyl sodium sulfosuccinate are thought to be sufficient to support the safety of the entire family of sulfosuccinate diesters of similar alkyl chain length, which are symmetrically substituted, and have similar functions in cosmetic formulations. Numerous studies examining the effect of the oral administration of diethylhexyl sodium sulfosuccinate, both dietary and by gavage, on the reproductive and developmental toxicity in rats were performed; one study was performed in mice. In a developmental study in mice and rats of a test substance containing 0.4% (w/v) diethylhexyl sodium sulfosuccinate, the NOAEL for maternal toxicity and teratogenic effects for both mice and rats was 400 mg/kg bw. In another developmental toxicity study in rats, the parental NOAEL was 400 mg/kg bw for a test substance containing 0.4% (w/v) diethylhexyl sodium sulfosuccinate. In a study in which gravid female Sprague-Dawley rats were fed a diet containing up to 2% diethylhexyl sodium sulfosuccinate, no adverse effects on maternal or fetal parameters were observed in the 1% test group, but in the 2% test group, significant incidences of resorptions and gross abnormalities, primarily exencephaly and, at times, spina bifida,anophthalmia, and associated skeletal defects, were reported. The NOAEL for maternal toxicity and teratogenic effects was 1%. In contrast to oral exposure, these esters are not expected to absorb through the skin to any significant extent, and the reproductive effects observed in test animals orally exposed to diethylhexyl sodium sulfosuccinate are not likely effects of topical application of cosmetics containing these ingredients. Consistent with this view, the Cosmetics Ingredient Review (CIR) Expert Panel:noted that acute dermal toxicity of undiluted diethylhexyl sodium sulfosuccinate was quite low, with a dermal LD50 of >10 g/kg in rabbit. However dialkyl sulfosuccinate salts may enhance the penetration of other ingredients through the skin. Under the exaggerated exposure conditions of the two repeated insult patch tests (RIPTs; continuous occlusive patch testing) presented in an earlier safety assessment of sodium diethylhexyl sulfosuccinate, the ingredient is a cumulative irritant, though not a sensitizer. Diethylhexyl sodium sulfosuccinate was used as a positive control in a Draize ocular irritation study; 10% diethylhexyl sodium sulfosuccinate was severely irritating to rabbit eyes, inducing perforated damages. Metabolism and excretion studies have given mixed results on the primary route of excretion of diethylhexyl sodium sulfosuccinate; it does appear that diethylhexyl sodium sulfosuccinate is metabolized prior to excretion, and most of the dose is excreted within 24 h of dosing. In one oral study in rats, 66% of the radioactivity was excreted in the faeces and only 25-35% in urine, within 24-48 h after dosing. In other rat studies, with oral and i.v. administration, the majority of the radioactivity was excreted in the urine, rather than in the faeces. Studies were also performed in rabbits and dogs, and again conflicting results were obtained. In rabbits, 87% and 69.7% of the radioactivity was excreted in the urine following oral and i.v. dosing, respectively; in dogs, approximately 70% of the radioactivity was excreted in the faeces at 24-48 h after oral and iv. dosing. The limited data available from short-term pharmaceutical studies in test animals exposed to diethylhexyl sodium sulfosuccinate aerosols suggest little potential for respiratory effects. This ingredient is reportedly used at concentrations up to 0.25% in cosmetic products that may be aerosolised. The Panel noted that 95%- 99% of droplets/particles would not be respirable to any appreciable amount. Further more, droplets/particles deposited in the nasopharyngeal or bronchial regions of the respiratory tract present no toxicological concerns based on the chemical properties and biological properties of this ingredient. Coupled with the small actual exposure in the breathing zone and the concentrations at which the ingredients are used, the available information indicates that incidental inhalation would not be a significant route of exposure that might lead to local respiratory or systemic effects. The Panel considered other data available to characterize the potential for the dialkyl sulfosuccinate salts to cause systemic toxicity, irritation, sensitization, reproductive and developmental toxicity, genotoxicity and carcinogenicity. They noted the lack of systemic toxicity in several acute and subchronic oral exposure studies, little or no irritation or sensitization in tests of dermal and ocular exposure, the absence of genotoxicity in Ames tests, and the lack of carcinogenicity in a subchronic oral exposure study. The CIR Expert Panel concluded that eight dialkyl sulfosuccinate salts are safe in the present practices of use and concentration in cosmetics described in this safety assessment when formulated to be non-irritating. Cosmetics Ingredient Review (CIR) Expert Panel: Safety Assessment of Dialkyl Sulfosuccinate Salts as Used in Cosmetics: September 2013 Literature data for other anionic surfactants (e.g. alkyl sulfates, alkane sulfonates and a-olefin sulfonates) demonstrated a similar toxicological and toxicokinetic/metabolic profile as for the sullfosuccinate esters/amides. For these surfactants high oral absorption rates (90%) and low dermal absorption rates (<1%) were observed. For risk characterisation of the registered substance, conservative absorption rates of 90, 2 and 10% were taken into account for oral, dermal and inhalation routes, respectively

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for alkyl sulfates; alkane sulfonates and alpha-olefin sulfonates

Most chemicals of this category are not defined substances, but mixtures of homologues with different alkyl chain lengths. Alpha-olefin sulfonates are mixtures of alkene sulfonate and hydroxyl alkane sulfonates with the sulfonate group in the terminal position and the double bond, or hydroxyl group, located at a position in the vicinity of the sulfonate group.
Common physical and/or biological pathways result in structurally similar breakdown products, and are, together with the surfactant properties, responsible for similar environmental behavior and essentially identical hazard profiles with regard to human health.
Acute toxicity: These substances are well absorbed after ingestion; penetration through the skin is however poor. After absorption, these chemicals are distributed mainly to the liver.

Acute oral LD50 values of alkyl sulfates in rats and/or mice were (in mg/kg):
C10-; 290-580
C10-16-, and C12-; 1000-2000
C12-14, C12-15, C12-16, C12-18 and C16-18-; >2000
C14-18, C16-18-; >5000
The clinical signs observed were non-specific (piloerection, lethargy, decreased motor activity and respiratory rate, diarrhoea). At necropsy the major findings were irritation of the gastrointestinal tract and anemia of inner organs.
Based on limited data, the acute oral LD50 values of alkane sulfonates and alpha-olefin sulfonates of comparable chain lengths are assumed to be in the same range.
The counter ion does not appear to influence the toxicity in a substantial way.

Acute dermal LD50 values of alkyl sulfates in rabbits (mg/ kg):
C12-; 200
C12-13 and C10-16-;>500

Apart from moderate to severe skin irritation, clinical signs included tremor, tonic-clonic convulsions, respiratory failure, and body weight loss in the study with the C12- alkyl sulfate and decreased body weights after administration of the C10-16- alkyl sulfates. No data are available for alkane sulfonates but due to a comparable metabolism and effect concentrations in long-term studies effect concentrations are expected to be in the same range as found for alkyl sulfates.

There are no data available for acute inhalation toxicity of alkyl sulfates, alkane sulfonates or alpha-olefin sulfonates.

In skin irritation tests using rabbits (aqueous solutions, OECD TG 404):
C8-14 and C8-16 (30%), C12-14 (90%), C14-18 (60%)- corrosive
Under occlusive conditions:
C12, and C12-14 (25%), C12-15-, C13-15 and C15-16 (5-7%) - moderate to strong irritants

Comparative studies investigating skin effects like transepidermal water loss, epidermal electrical conductance, skin swelling, extraction of amino acids and proteins or development of erythema in human volunteers consistently showed a maximum of effects with C12-alkyl sulfate, sodium; this salt is routinely used as a positive internal control giving borderline irritant reactions in skin irritation studies performed on humans. As the most irritant alkyl sulfate it can be concluded that in humans 20% is the threshold concentration for irritative effects of alkyl sulfates in general. No data were available with regard to the skin irritation potential of alkane sulfonates. Based on the similar chemical structure they are assumed to exhibit similar skin irritation properties as alkyl sulfates or alpha-olefin sulfonates of comparable chain lengths.

In eye irritation tests, using rabbits, C12-containing alkyl sulfates (>10% concentration) were severely irritating and produced irreversible corneal effects. With increasing alkyl chain length, the irritating potential decreases, and C16-18 alkyl sulfate sodium, at a concentration of 25%, was only a mild irritant.
Concentrated C14-16- alpha-olefin sulfonates were severely irritating, but caused irreversible effects only if applied as undiluted powder. At concentrations below 10% mild to moderate, reversible effects, were found. No data were available for alkane sulfonates

Alkyl sulfates and C14-18 alpha-olefin sulfonates were not skin sensitisers in animal studies. No reliable data were available for alkane sulfonates. Based on the similar chemical structure, no sensitisation is expected.
However anecdotal evidence suggests that sodium lauryl sulfate causes pulmonary sensitisation resulting in hyperactive airway dysfunction and pulmonary allergy accompanied by fatigue, malaise and aching. Significant symptoms of exposure can persist for more than two years and can be activated by a variety of non-specific environmental stimuli such as a exhaust, perfumes and passive smoking.
Absorbed sulfonates are quickly distributed through living systems and are readily excreted. Toxic effects may result from the effects of binding to proteins and the ability of sulfonates to translocate potassium and nitrate (NO_3^-) ions from cellular to interstitial fluids. Airborne sulfonates may be responsible for respiratory allergies and, in some instances, minor dermal allergies. Repeated skin contact with some sulfonated surfactants has produced sensitisation dermatitis in predisposed individuals

Repeat dose toxicity: After repeated oral application of alkyl sulfates with chain lengths between C12 and C18, the liver was the only target organ for systemic toxicity. Adverse effects on this organ included an increase in liver weight, enlargement of liver cells, and elevated levels of liver enzymes. The LOAEL for liver toxicity (parenchymal hypertrophy and an increase in comparative liver weight) was 230 mg/kg/day (in a 13 week study with C16-18 alkyl sulfate, sodium). The lowest NOAEL in rats was 55 mg/kg/day (in a 13 week study with C12-alkyl sulfate, sodium).
C14- and C14-16-alpha-olefin sulfonates produced NOAELs of 100 mg/kg/day (in 6 month- and 2 year studies). A reduction in body weight gain was the only adverse effect identified in these studies.
No data were available with regard to the repeated dose toxicity of alkane sulfonates. Based on the similarity of metabolic pathways between alkane sulfonates, alkyl sulfates and alkyl-olefin sulfonates, the repeated dose toxicity of alkane sulfonates is expected to be similar with NOAEL and LOAEL values in the same range as for alkyl sulfates and alpha-olefin sulfonates, i.e. 100 and 200-250 mg/kg/day, respectively, with the liver as potential target organ.

Genotoxicity: Alkyl sulfates of different chain lengths and with different counter ions were not mutagenic in standard bacterial and mammalian cell systems both in the absence and in the presence of metabolic activation. There was also no indication for a genotoxic potential of alkyl sulfates in various in vivo studies on mice (micronucleus assay, chromosome aberration test, and dominant lethal assay). alpha-Olefin sulfonates were not mutagenic in the Ames test, and did not induce chromosome aberrations in vitro. No genotoxicity data were available for alkane sulfonates. Based on the overall negative results in the genotoxicity assays with alkyl sulfates and alpha-olefin sulfonates, the absence of structural elements indicating mutagenicity, and the overall database on different types of sulfonates, which were all tested negative in mutagenicity assays, a genotoxic potential of alkane sulfonates is not expected.

Carcinogenicity: Alkyl sulfates were not carcinogenic in feeding studies with male and female Wistar rats fed diets with C12-15 alkyl sulfate sodium for two years (corresponding to doses of up to 1125 mg/kg/day).
alpha-Olefin sulfonates were not carcinogenic in mice and rats after dermal application, and in rats after oral exposure.
No carcinogenicity studies were available for the alkane sulfonates.

Reproductive toxicity: No indication for adverse effects on reproductive organs was found in various oral studies with different alkyl sulfates. The NOAEL for male fertility was 1000 mg/kg/day for sodium dodecyl sulfate. In a study using alpha-olefin sulfonates in male and female rats, no adverse effects were identified up to 5000 ppm.

Developmental toxicity: In studies with various alkyl sulfates (C12 up to C16-18- alkyl) in rats, rabbits and mice, effects on litter parameters were restricted to doses that caused significant maternal toxicity (anorexia, weight loss, and death).
The principal effects were higher foetal loss and increased incidences of total litter losses. The incidences of malformations and visceral and skeletal anomalies were unaffected apart from a higher incidence of delayed ossification or skeletal variation in mice at > 500 mg/kg bw/day

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	indicative of a delayed development. The lowest reliable NOAEL for maternal toxicity was about 200 mg/kg/day in rats, while the lowest NOAELs in offspring were 250 mg/kg/day in rats and 300 mg/kg/day for mice and rabbits. For alpha-olefin sulfonates (C14-16-alpha-olefin sulfonate, sodium) the NOAEL was 600 mg/kg/day both for maternal and developmental toxicity. No data were available for the reproductive and developmental toxicity of alkane sulfonates. Based on the available data, the similar toxicokinetic properties and a comparable metabolism of the alkyl sulfates and alkane sulfonates, alkane sulfonates are not considered to be developmental toxicants. Although the database for category members with C<12 is limited, the available data are indicating no risk as the substances have comparable toxicokinetic properties and metabolic pathways. In addition, longer-term studies gave no indication for adverse effects on reproductive organs with different alkyl sulfates
ZINC PYRITHIONE	NOAEL: 11.0 mg/kg/day cynomolgus monkey * [* = Arch Chemical] Acute pulmonary oedema, dyspnea, weight loss or decreased weight gain, recordings from specific areas of the CNS, mydriasis, somnolence, changes in motor activity, recording from peripheral motor nerve, muscle weakness, spastic paralysis, reproductive system tumours, retinal changes, diarrhoea, foetoxicity, specific developmental abnormalities (musculoskeletal system, central nervous system, effects on newborn, foetolethality recorded. for pyrithiones: **Short-term studies:** Zinc pyrithione was orally administered to cynomolgus monkeys daily for 14 or 28 days.In the 14-day study, treatment at 10, 20, 40 or 80 mg/kg bw/day resulted in haemorrhaging of the stomach mucosa and bodyweight loss at the highest tested dose. In the 28-day study, treatment at 0, 5.5, 11 or 22 mg/kg bw, caused a death at the highest dose. Food consumption and bodyweight gain was decreased at the highest dose together with reduced haematocrit, haemoglobin concentration and erythrocyte count. An increased concentration of ketone bodies and decreased pH of the urine was also observed. These changes were either absent or had improved after a 14-day recovery period. In a 90-day study, rats were fed zinc pyrithione in the diet at concentrations of 0, 5, 25 or 125 ppm. Clinical signs first observed during the second week at 125 ppm were a depressed respiratory rate and the onset of progressively restricted movement of the hind limbs which finally resulted in almost complete paralysis. Other changes at 125 ppm were related to severe weight loss and dehydration, resulting from the paralysis. Based on the deaths of nearly all the rats at 125 ppm (from dehydration and/or starvation) and the reduced bodyweight observed at 25 ppm in females, the NOEL for this study was 5 ppm (0.35 mg/kg bw/day for males and 0.39 mg/kg bw/day for females). Daily dermal application of zinc pyrithione to rats at 0, 20, 100 or 1000 mg/kg bw/day for 90 days revealed slight skin irritation, bodyweight loss and reduced food intake at 1000 mg/kg bw/day. For females at 1000 mg/kg bw/day there was an increase in relative kidney weight and some had mineralisation of the kidneys. Increased leucocyte counts and reduced erythrocyte and haematocrit was also observed at the highest dose. Dermal absorption studies in pigs showed that zinc pyrithione is very poorly absorbed through skin (<10% of dose). A maximum of 5% of the applied dose was recovered in the urine and by 48 h the levels in blood, faeces, and urine were essentially at background levels. Whole-body exposure to an aerosol at 0, 0.5, 2.5 or 10 mg/m3 for 6 h/day, 5 days/week over 13 weeks resulted in deaths at 2.5 and 10 mg/m3, reduced bodyweight gain at 10 mg/m3 and reduced creatinine at 10 mg/m3. A dose-related increase in mean absolute lung/mainstream bronchi weight, lung/mainstream bronchi weight relative to body weight and lung/mainstream bronchi weight relative to brain weight was also observed at 2.5 and 10 mg/m3. These weight increases were accompanied by inflammation of interstitial tissue and pulmonary artery hypertrophy. Zinc pyrithione given to monkeys at 0, 0.5, 2 or 8 mg/kg bw/day by stomach tube for 90 days induced some vomiting at 2 and 8 mg/kg bw/day within 1-3 h on the first few treatment days. Appropriate monitoring for adverse changes failed to reveal any other effects. Hence, the NOEL for the study was 8 mg/kg bw/day. **Long-Term Study:** Sodium pyrithione at 0, 0.5, 1.5 or 5 mg/kg bw/day was administered to rats by gavage in a two-year chronic and oncogenicity study. After 12 weeks at 5 mg/kg bw/day, an appreciable reduction in bodyweight gain necessitated the high dose level be reduced to 3.5 mg/kg bw/day. There was reduced bodyweight gain at 3.5 mg/kg bw/day and hind limb muscle wastage at 1.5 and 3.5 mg/kg bw/day. Nerve fibre degeneration of the spinal cord and sciatic nerve was slightly increased at 3.5 mg/kg bw/day. Fibre degeneration in the hind limb skeletal muscle was increased in all rats at 3.5 mg/kg bw/day and to a lesser extent in females at 1.5 mg/kg bw/day. There was an increase in peripheral retinal atrophy in males and females at 3.5 mg/kg bw/day and at 1.5 mg/kg bw/day in females. There was no treatment-related increase in the incidence of tumours. Therefore, under the conditions of this study, the NOEL was 0.5 mg/kg bw/day. **Reproduction and Developmental Studies:** In a 2- generation reproduction study, rats were given sodium pyrithione at 0, 0.5, 1.5 or 4.5 mg/kg bw/day by gavage. Owing to an appreciable reduction in bodyweight gain the highest dose was reduced after 3 weeks to 3.5 mg/kg bw/day for the rest of the study. Rats were maintained for 2 generations, with the first litter used for breeding. In the F0 rats, salivation after dosing was seen in all treated groups, with a dose-related time of onset and severity. At 3.5 mg/kg bw/day a number of females showed hind- limb paralysis in the F0 generation; this was not seen in F1 animals. Body weight gain was statistically significantly decreased in both males and females at 3.5 mg/kg bw/day in the F0 generation, and in females at this dose in the F1 generation. Fertility was decreased at 3.5 mg/kg bw/day in the F0 generation, with the number of rats successfully mating and the number of rats pregnant decreased in comparison to controls. There was no effect on gestational length, the number of pups born or pup bodyweight seen. No effects on fertility were seen in the F1 generation. There was no increase in the incidence of foetal malformations in either generation. On postmortem examination, there was an increase in the incidence of hind- limb muscle atrophy at 3.5 mg/kg bw/day in females in both generations. On histopathological examination, there was an increase in atrophy of skeletal muscles at 3.5 mg/kg bw/day in the F0 generation, and from 1.5 mg/kg bw/day in the F1 generation. Salivation occurred in some F0 rats at 0.5 mg/kg bw/day but none in the F1 generation suggesting that this dose level is a probable NOEL. When pregnant rats had zinc pyrithione topically applied at 0, 2.5, 7.5 or 15 mg/kg bw/day (with or without prevention from ingestion) from gestation days 6 to 15 there was a reduction in bodyweight gain at 7.5 or 15 mg/kg bw/day when ingestion was not prevented. Hind-limb paralysis among dams and reductions in fetal weight were also observed at 15 mg/kg bw/day. These effects were not seen when ingestion was prevented. With oral treatment at the same doses, bodyweight gain was reduced, paralysis occurred and fetal weight was reduced at 7.5 and 15 mg/kg bw/day. There was also an increase in skeletal variations at 15 mg/kg bw/day. **Genotoxicity:** Zinc pyrithione was found to be negative in mutation tests in bacteria and Chinese hamster ovary cells. Similarly, no chromosomal aberration was observed in human lymphocytes incubated ***in vitro*** in the presence of zinc pyrithione or in lymphocytes harvested from monkeys following oral administration in a 28-day toxicity study. A mouse micronucleus assay also yielded negative results. **Human metabolite study** A study of plasma metabolites in human volunteers from a chemical factory producing pyrithiones identified 2-(methylsulfonyl)pyridine as the only metabolite in human serum and proposed that this metabolite could be used as a marker for pyrithione exposure. Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).
MONOISOBUTANOLAMINE	For tris(hydroxymethyl)aminomethane (TRIS AMINO; CAS 77-88-1) and its surrogates 2-amino-2-methyl-1,3-propanediol (AMPD; CAS 115-69-5) and monoisobutanolamine (AMP; CAS 124-68-5) TRIS AMINO and the surrogate chemicals have displayed little if any toxicity to humans during their long history of use as human drugs and/or in personal care products and cosmetics. TRIS AMINO has found use as an IV drug for the management of acidosis in humans for many years and the toxicity of AMPD and AMP have been reviewed by the Cosmetic Ingredient Review Expert Panel which concluded that these materials are safe as used in cosmetic formulations up to 1% **Acute toxicity:** Mammalian toxicity studies have displayed similar results. The oral LD50 value for TRIS AMINO is 5500 mg/kg in the mouse, and its surrogates range from 2150 to greater than 5000 mg/kg in the rat and mouse. TRIS AMINO was non-irritating to eyes when a 40% aqueous solution was applied to the eyes of rabbits (pH 10.4 for 0.1M aqueous solution). In contrast, 95% AMP in water was severely irritating to the eyes, presumably due to the severely alkaline pH of the test solution used (pH 11.3 for 0.1M aqueous solution); however, more neutral cosmetic formulations containing lower concentrations of AMP are only minimally irritating. There is no sensitisation data available for TRIS AMINO; however, based on the following data, TRIS AMINO is not expected to be a sensitiser. Laboratory animal test samples of AMP did not cause allergic skin reactions when tested in guinea pigs following topical or intradermal administration. In patch tests with humans, AMP and cosmetic formulations containing either AMP or AMPD were negative for dermal sensitisation. **Repeated dose toxicity**: Repeated-dose mammalian toxicity studies conducted on TRIS AMINO and the two surrogate chemicals indicate that the compounds are generally well-tolerated at concentrations as high as 500 mg/kg/day via IV infusion for TRIS AMINO and ingestion of up to 3200 ppm in the rodent diet (250-750 mg/kg/day for rats and mice, estimated). A number of human clinical trials of the IV infusion of TRIS AMINO have also been successfully conducted. In all studies, the only target tissue, when observed at all, has been the liver with AMP. Human clinical studies with Keterolac(a major component of which is TRIS AMINO) have suggested that patients with decreased liver

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	function not be given the drug over extended treatment periods based upon changes in several clinical chemistry parameters. Ingestion of relatively high dosages of AMP has caused liver histopathological changes in rats and dogs. The most significant toxicological activity has been a foetotoxic effect of AMP when ingested at relatively high levels by pregnant rats. Subsequent dermal exposure to comparable dosages failed to elicit a developmental effect in rats. Overall, there have been no consistently-noted observations or treatment-related findings among the numerous repeated-dose mammalian toxicity studies that have been conducted over at last 50 years on these compounds that would indicate long-term significant toxicity of either compound at typical human exposure levels. Reflective of these findings is the fact that both TRIS AMINO and AMP display similar patterns of excretion from the body, being primarily eliminated unchanged via the urine over a relatively short period of time. Further, no evidence of either direct reactivity or metabolism to reactive species toward genetic material has been observed. **Genetic toxicity:** Studies conducted on the TRIS AMINO and the surrogate substances in the presence or absence of mammalian metabolic enzymes have all been negative.
PROPYLENE GLYCOL & ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED & POLYPROPYLENE GLYCOL & SODIUM DIOCTYL SULFOSUCCINATE	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 the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
DIPROPYLENE GLYCOL MONOMETHYL ETHER & ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED & TITANIUM DIOXIDE	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.
DIPROPYLENE GLYCOL MONOMETHYL ETHER & POLYPROPYLENE GLYCOL	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
DIPROPYLENE GLYCOL MONOMETHYL ETHER & TITANIUM DIOXIDE & 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.
ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED & POLYPROPYLENE GLYCOL & 4-NONYLPHENOL, BRANCHED, ETHOXYLATED	Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture . On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing. Allergic Contact Dermatitis—Formation, Structural Requirements,and Reactivity of Skin Sensitizers. Ann-Therese Karlberg et al; Chem. Res. Toxicol.2008,21,53-69 Polyethylene glycols (PEGs) have a wide variety of PEG-derived mixtures due to their readily linkable terminal primary hydroxyl groups in combination with many possible compounds and complexes such as ethers, fatty acids, castor oils, amines, propylene glycols, among other derivatives. PEGs and their derivatives are broadly utilized in cosmetic products as surfactants, emulsifiers, cleansing agents, humectants, and skin conditioners. PEGs and PEG derivatives were generally regulated as safe for use in cosmetics, with the conditions that impurities and by-products, such as ethylene oxides and 1,4-dioxane, which are known carcinogenic materials, should be removed before they are mixed in cosmetic formulations. Most PEGs are commonly available commercially as mixtures of different oligomer sizes in broadly- or narrowly-defined molecular weight (MW) ranges. For instance, PEG-10,000 typically designates a mixture of PEG molecules (n = 195 to 265) having an average MW of 10,000. PEG is also known as polyethylene oxide (PEO) or polyoxyethylene (POE), with the three names being chemical synonyms. However, PEGs mainly refer to oligomers and polymers with molecular masses below 20,000 g/mol, while PEOs are polymers with molecular masses above 20,000 g/mol, and POEs are polymers of any molecular mass. Relatively small molecular weight PEGs are produced by the chemical reaction between ethylene oxide and water or ethylene glycol (or other ethylene glycol oligomers), as catalyzed by acidic or basic catalysts. To produce PEO or high-molecular weight PEGs, synthesis is performed by suspension polymerization. It is necessary to hold the growing polymer chain in solution during the course of the poly-condensation process. The reaction is catalyzed by magnesium-, aluminum-, or calcium-organoelement compounds. To prevent coagulation of polymer chains in the solution, chelating additives such as dimethylglyoxime are used Safety Evaluation of Polyethyene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of Toxicology https://doi.org/10.5487/TR.2015.31.2.105
ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED & 4-NONYLPHENOL, BRANCHED, ETHOXYLATED	Human beings have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents, and other cleaning products . Exposure to these chemicals can occur through ingestion, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that volumes well above a reasonable intake level would have to occur to produce any toxic response. Moreover, no fatal case of poisoning with alcohol ethoxylates has ever been reported. Multiple studies investigating the acute toxicity of alcohol ethoxylates have shown that the use of these compounds is of low concern in terms of oral and dermal toxicity . Clinical animal studies indicate these chemicals may produce gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhea, and lethargy. Similarly, slight to severe irritation of the skin or eye was generated when undiluted alcohol ethoxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotoxin, carcinogen, or mutagen (HERA 2007). No information was available on levels at which these effects might occur, though toxicity is thought to be substantially lower than that of nonylphenol ethoxylates. Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture . On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However,their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose allergic contact dermatitis (ACD) to these compounds by patch testing Overall, alcohol alkoxylates (AAs) are not expected to be systemically toxic, although some short chain ethylene glycol ethers, e.g. methyl and ethyl homologues are of concern for a range of adverse health effects. They include skin and eye irritation, liver and kidney damage, bone marrow and central nervous system (CNS) depression, testicular atrophy, developmental toxicity, and immunotoxicity. For higher propyl

Continued...

and butyl homologues, the toxicity involves haemolysis (anaemia) with secondary effects relating to haemosiderin accumulation in the spleen, liver and kidney, and compensatory haematopoiesis in the bone marrow. Systemic toxicity was shown to decrease with increasing alkyl chain lengths and/or alkoxylation degrees (ECETOC, 2005; US EPA, 2010). The chemicals ethylene glycol hexyl ether (with a longer alkyl chain length, CAS No. 112-25-4) and diethylene glycol butyl ether (with a higher ethoxylation degree, CAS No. 112-34-5) have no evidence of systemic effects including haemolysis.
Commercially available AAs are mixtures of homologues of varying carbon chain lengths and it is possible that some of the chemicals with an average alkyl chain length C >=6 may also contain shorter alkyl chains C <6. It is not practical to quantify the proportion of shorter C <6 chain lengths present in such chemicals, or these shorter chain lengths may not be present at all. The available data suggest a lack of systemic toxicity for the AE chemicals with potential short alkyl chain presence (NICNASa); therefore, the toxicity of the chemicals in this assessment is unlikely to be significantly affected by the presence of shorter chain alkyl groups.
Alcohol ethoxylates are according to CESIO (2000) classified as Irritant or Harmful depending on the number of EO-units:
EO < 5 gives Irritant (Xi) with R38 (Irritating to skin) and R41 (Risk of serious damage to eyes)
EO > 5-15 gives Harmful (Xn) with R22 (Harmful if swallowed) - R38/41
EO > 15-20 gives Harmful (Xn) with R22-41
>20 EO is not classified (CESIO 2000)
Oxo-AE, C13 EO10 and C13 EO15, are Irritating (Xi) with R36/38 (Irritating to eyes and skin) .
AE are not included in Annex 1 of the list of dangerous substances of the Council Directive 67/548/EEC

In general, alcohol ethoxylates (AE) are readily absorbed through the skin of guinea pigs and rats and through the gastrointestinal mucosa of rats. AE are quickly eliminated from the body through the urine, faeces, and expired air (CO_2).Orally dosed AE was absorbed rapidly and extensively in rats, and more than 75% of the dose was absorbed. When applied to the skin of humans, the doses were absorbed slowly and incompletely (50% absorbed in 72 hours). Half of the absorbed surfactant was excreted promptly in the urine and smaller amounts of AE appeared in the faeces and expired air (CO_2)). The metabolism of C12 AE yields PEG, carboxylic acids, and CO_2 as metabolites. The LD50 values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.

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

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 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

Continued...

	test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity **Mutagenicity:** Mutagenicity studies have been conducted for several category members. All in vitro and in vivo studies were negative at concentrations up to 5,000 micrograms/plate and 5,000 mg/kg, respectively, indicating that the category members are not genotoxic at the concentrations used in these studies. The uniformly negative outcomes of various mutagenicity studies performed on category members lessen the concern for carcinogenicity. **Reproductive toxicity:** Although mating studies with either the category members or surrogates have not been performed, several of the repeated dose toxicity tests with the surrogates have included examination of reproductive organs. A lower molecular weight glycol ether, ethylene glycol methyl ether (EGME), has been shown to be a testicular toxicant. In addition, results of repeated dose toxicity tests with TGME clearly show testicular toxicity at an oral dose of 4,000 mg/kg/day four times greater that the limit dose of 1,000 mg/kg/day recommended for repeat dose studies. It should be noted that TGME is 350 times less potent for testicular effects than EGME. TGBE is not associated with testicular toxicity, TetraME is not likely to be metabolised by any large extent to 2-MAA (the toxic metabolite of EGME), and a mixture containing predominantly methylated glycol ethers in the C5-C11 range does not produce testicular toxicity (even when administered intravenously at 1,000 mg/kg/day). **Developmental toxicity**: The bulk of the evidence shows that effects on the foetus are not noted in treatments with . 1,000 mg/kg/day during gestation. At 1,250 to 1,650 mg/kg/day TGME (in the rat) and 1,500 mg/kg/day (in the rabbit), the developmental effects observed included skeletal variants and decreased body weight gain.
ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED & 4-NONYLPHENOL, BRANCHED, ETHOXYLATED & SODIUM DIOCTYL SULFOSUCCINATE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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

Folk Art Dragonfly Paints

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

propylene glycol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	19300mg/l	2
EC50	48h	Crustacea	>114.4mg/L	4
LC50	96h	Fish	710mg/L	4
EC50	96h	Algae or other aquatic plants	19000mg/l	2
NOEC(ECx)	336h	Algae or other aquatic plants	<5300mg/l	1

dipropylene glycol monomethyl ether

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>969mg/l	2
EC50	48h	Crustacea	1930mg/l	2
LC50	96h	Fish	>1000mg/l	2
NOEC(ECx)	528h	Crustacea	>=0.5mg/l	2
EC50	96h	Algae or other aquatic plants	>969mg/l	2

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

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	1-10mg/l	Not Available

titanium dioxide

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1008h	Fish	<1.1-9.6	7
EC50	72h	Algae or other aquatic plants	3.75-7.58mg/l	4
EC50	48h	Crustacea	1.9mg/l	2
LC50	96h	Fish	1.85-3.06mg/l	4
NOEC(ECx)	672h	Fish	>=0.004mg/L	2
EC50	96h	Algae or other aquatic plants	179.05mg/l	2

polypropylene glycol

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	>100mg/l	2
EC50	72h	Algae or other aquatic plants	>100mg/l	2
EC50	48h	Crustacea	>100mg/l	2
NOEC(ECx)	504h	Crustacea	>=10mg/l	2

Continued...

	EC50	96h	Algae or other aquatic plants	3000-4000mg/l	2

4-nonylphenol, branched, ethoxylated

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

sodium dioctyl sulfosuccinate

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1008h	Fish	<0.9	7
EC50	72h	Algae or other aquatic plants	38.1-40.8mg/l	4
EC50	48h	Crustacea	6.6mg/l	2
LC50	96h	Fish	12.5mg/l	1
NOEC(ECx)	96h	Fish	0.059mg/l	4

zinc pyrithione

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1440h	Fish	52-180	7
EC50	72h	Algae or other aquatic plants	0.001mg/L	4
EC50	48h	Crustacea	0.002-2.14mg/L	4
LC50	96h	Fish	0.003mg/L	2
NOEC(ECx)	96h	Algae or other aquatic plants	<0.001mg/L	2
EC50	96h	Algae or other aquatic plants	<0.001mg/L	4

monoisobutanolamine

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>103mg/l	2
EC50	48h	Crustacea	193mg/l	1
EC0(ECx)	48h	Crustacea	100mg/l	1
LC50	96h	Fish	100mg/l	1
EC50	96h	Algae or other aquatic plants	>103mg/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.
Propylene glycol is known to exert high levels of biochemical oxygen demand (BOD) during degradation in surface waters. This process can adversely affect aquatic life by consuming oxygen needed by aquatic organisms for survival. Large quantities of dissolved oxygen (DO) in the water column are consumed when microbial populations decompose propylene glycol.
Sufficient dissolved oxygen levels in surface waters are critical for the survival of fish, macro-invertebrates, and other aquatic organisms. If oxygen concentrations drop below a minimum level, organisms emigrate, if able and possible, to areas with higher oxygen levels or eventually die. This effect can drastically reduce the amount of usable aquatic habitat. Reductions in DO levels can reduce or eliminate bottom-feeder populations, create conditions that favour a change in a community's species profile, or alter critical food-web interactions.
log Kow : -1.41- -0.3
Half-life (hr) air : 32
Henry's atm m3 /mol: 1.20E-08
BOD 5: 0.995,2.2%
ThOD : 1.685
BCF : <1
Bioaccumulation : not sig
processes Abiotic: photoxid
For Surfactants: Kow cannot be easily determined due to hydrophilic/hydrophobic properties of the molecules in surfactants. BCF value: 1-350.
Aquatic Fate: Surfactants tend to accumulate at the interface of the air with water and are not extracted into one or the other liquid phases.
Terrestrial Fate: Anionic surfactants are not appreciably sorbed by inorganic solids. Cationic surfactants are strongly sorbed by solids, particularly clays. Significant sorption of anionic and non-ionic surfactants has been observed in activated sludge and organic river sediments. Surfactants have been shown to improve water infiltration into soils with moderate to severe hydrophobic or water-repellent properties.
Ecotoxicity: Some surfactants are known to be toxic to animals, ecosystems and humans, and can increase the diffusion of other environmental contaminants. The acute aquatic toxicity generally is considered to be related to the effects of the surfactant properties on the organism and not to direct chemical toxicity. Surfactants should be considered to be toxic to aquatic species under conditions that allow contact of the chemicals with the organisms. Surfactants are expected to transfer slowly from water into the flesh of fish. During this process, readily biodegradable surfactants are expected to be metabolized rapidly during the process of bioaccumulation. Surfactants are not to be considered to show bioaccumulation potential if they are readily biodegradable.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
propylene glycol	LOW	LOW
dipropylene glycol monomethyl ether	HIGH	HIGH
titanium dioxide	HIGH	HIGH
polypropylene glycol	LOW	LOW
monoisobutanolamine	LOW	LOW

Continued...

Bioaccumulative potential

Ingredient	Bioaccumulation
propylene glycol	LOW (BCF = 1)
dipropylene glycol monomethyl ether	LOW (BCF = 100)
titanium dioxide	LOW (BCF = 10)
polypropylene glycol	LOW (LogKOW = 1.6984)
sodium dioctyl sulfosuccinate	LOW (BCF = 3.78)
zinc pyrithione	LOW (BCF = 240)
monoisobutanolamine	LOW (BCF = 330)

Mobility in soil

Ingredient	Mobility
propylene glycol	HIGH (Log KOC = 1)
dipropylene glycol monomethyl ether	LOW (Log KOC = 10)
titanium dioxide	LOW (Log KOC = 23.74)
polypropylene glycol	LOW (Log KOC = 15.66)
monoisobutanolamine	MEDIUM (Log KOC = 2.196)

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.

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

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

Not Applicable

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

Product name	Group
propylene glycol	Not Available
dipropylene glycol monomethyl ether	Not Available
alcohols C11-14-iso-, C13-rich, ethoxylated	Not Available
titanium dioxide	Not Available
polypropylene glycol	Not Available

Continued...

Product name	Group
4-nonylphenol, branched, ethoxylated	Not Available
sodium dioctyl sulfosuccinate	Not Available
zinc pyrithione	Not Available
monoisobutanolamine	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
propylene glycol	Not Available
dipropylene glycol monomethyl ether	Not Available
alcohols C11-14-iso-, C13-rich, ethoxylated	Not Available
titanium dioxide	Not Available
polypropylene glycol	Not Available
4-nonylphenol, branched, ethoxylated	Not Available
sodium dioctyl sulfosuccinate	Not Available
zinc pyrithione	Not Available
monoisobutanolamine	Not Available

SECTION 15 Regulatory information

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

propylene glycol is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)

dipropylene glycol monomethyl ether is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)

alcohols C11-14-iso-, C13-rich, ethoxylated is found on the following regulatory lists
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

titanium dioxide is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
International Agency fsor Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

polypropylene glycol is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)

4-nonylphenol, branched, ethoxylated is found on the following regulatory lists
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

sodium dioctyl sulfosuccinate is found on the following regulatory lists
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

zinc pyrithione is found on the following regulatory lists
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)

monoisobutanolamine is found on the following regulatory lists
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

Additional Regulatory Information
Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes

Continued...

National Inventory	Status
Canada - NDSL	No (propylene glycol; dipropylene glycol monomethyl ether; alcohols C11-14-iso-, C13-rich, ethoxylated; polypropylene glycol; 4-nonylphenol, branched, ethoxylated; sodium dioctyl sulfosuccinate; zinc pyrithione; monoisobutanolamine)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (alcohols C11-14-iso-, C13-rich, ethoxylated)
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	No (zinc pyrithione)
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	04/10/2024
Initial Date	03/10/2024

SDS Version Summary

Version	Date of Update	Sections Updated
3.1	04/10/2024	Toxicological information - Acute Health (eye), Toxicological information - Acute Health (inhaled), Toxicological information - Acute Health (skin), Toxicological information - Acute Health (swallowed), First Aid measures - Advice to Doctor, Physical and chemical properties - Appearance, Toxicological information - Chronic Health, Hazards identification - Classification, Disposal considerations - Disposal, Exposure controls / personal protection - Engineering Control, Ecological Information - Environmental, Firefighting measures - Fire Fighter (extinguishing media), Firefighting measures - Fire Fighter (fire/explosion hazard), Firefighting measures - Fire Fighter (fire fighting), Firefighting measures - Fire Fighter (fire incompatibility), First Aid measures - First Aid (eye), First Aid measures - First Aid (inhaled), First Aid measures - First Aid (skin), First Aid measures - First Aid (swallowed), Handling and storage - Handling Procedure, Composition / information on ingredients - Ingredients, Stability and reactivity - Instability Condition, Exposure controls / personal protection - Personal Protection (other), Exposure controls / personal protection - Personal Protection (Respirator), Exposure controls / personal protection - Personal Protection (eye), Exposure controls / personal protection - Personal Protection (hands/feet), Accidental release measures - Spills (major), Accidental release measures - Spills (minor), Handling and storage - Storage (storage incompatibility), Handling and storage - Storage (storage requirement), Handling and storage - Storage (suitable container), Transport information - Transport, Identification of the substance / mixture and of the company / undertaking - 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
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration

- AIIC: 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

Continued...

- INSQ: Inventario Nacional de Sustancias Químicas
- NCI: National Chemical Inventory
- FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

end of SDS