Folk Art Multi Surface Paints

Jasco Pty Limited

Chemwatch Hazard Alert Code: 4

Chemwatch: **7912-21**
Version No: **5.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Issue Date: **15/10/2024**
Print Date: **15/10/2024**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	Folk Art Multi Surface 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	Paint. 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 number(s)	**13 11 26 (24/7)**
Other emergency telephone number(s)	**Not Available**

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Label elements

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

Hazard statement(s)

H315	Causes skin irritation.
H318	Causes serious eye damage.
H350	May cause cancer.
H361fd	Suspected of damaging fertility. Suspected of damaging the unborn child.
H401	Toxic to aquatic life.
H412	Harmful to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

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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

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

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
13463-67-7	10-40	C.I. Pigment White 6
57-55-6	1-30	propylene glycol
57455-37-5	0-20	C.I. Pigment Blue 29
51274-00-1	0-10	ferric hydroxide
1333-86-4	<5	C.I. Pigment Black 7
1317-65-3	<5	calcium carbonate
34590-94-8	<5	dipropylene glycol monomethyl ether
127087-87-0	<5	4-nonylphenol, branched, ethoxylated
1332-58-7	<5	kaolin
1309-37-1	<5	ferric oxide
7631-86-9	<5	silica amorphous
1344-28-1.	<5	aluminium oxide
1336-21-6	<1	ammonium hydroxide
68186-36-7	<1	tridecyl alcohol, ethoxylated, phosphated, potassium salt
577-11-7	<1	sodium dioctyl sulfosuccinate
77-99-6	<1	trimethylolpropane
111-76-2	<1	ethylene glycol monobutyl ether
25322-69-4	<1	polypropylene glycol
14808-60-7	<1	silica crystalline - quartz
111-77-3	<1	diethylene glycol monomethyl ether
36968-27-1	<1	C.I. Pigment Red 266
68439-51-0	<1	alcohols C12-14 ethoxylated propoxylated
108-01-0	<1	dimethylethanolamine
9016-45-9	<1	nonylphenol ethoxylates
135-19-3	<1	2-naphthol
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: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs:

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	▸ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▸ Quickly remove all contaminated clothing, including footwear. ▸ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▸ Transport to hospital, or doctor.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Firefighting measures

Extinguishing media

- There is no restriction on the type of extinguisher which may be used.
- Use extinguishing media suitable for surrounding area.

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 breathing apparatus plus protective gloves in the event of a fire. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ Use fire fighting procedures suitable for surrounding area. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire. ▸ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	▸ The material is not readily combustible under normal conditions. ▸ However, it will break down under fire conditions and the organic component may burn. ▸ Not considered to be a significant fire risk. ▸ Heat may cause expansion or decomposition with violent rupture of containers. ▸ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke. Decomposes on heating and produces toxic fumes of: carbon dioxide (CO_2) nitrogen oxides (NOx) phosphorus oxides (POx) sulfur oxides (SOx) silicon dioxide (SiO_2) hydrogen sulfide (H_2S) metal oxides other pyrolysis products typical of burning organic material. When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles. 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	▸ 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	▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by all means available, spillage from entering drains or water courses. ▸ Consider evacuation (or protect in place). ▸ No smoking, naked lights or ignition sources. ▸ Increase ventilation.

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	▸ Stop leak if safe to do so. ▸ Water spray or fog may be used to disperse / absorb vapour. ▸ Contain or absorb spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▸ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

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

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Polyethylene or polypropylene container. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ Avoid reaction with oxidising agents ▸ Avoid strong acids, bases.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	C.I. Pigment White 6	Titanium dioxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
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	propylene glycol	Propane-1,2-diol: particulates only	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	ferric hydroxide	Rouge dust	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	ferric hydroxide	Iron oxide fume (Fe2O3) (as Fe)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	C.I. Pigment Black 7	Carbon black	3 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	calcium carbonate	Calcium carbonate	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	dipropylene glycol monomethyl ether	(2-Methoxymethylethoxy) propanol	50 ppm / 308 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	kaolin	Kaolin	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	ferric oxide	Iron oxide fume (Fe2O3) (as Fe)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	ferric oxide	Rouge dust	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	silica amorphous	Precipitated silica	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Silica gel	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica gel	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Precipitated silica	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Diatomaceous earth (uncalcined)	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Diatomaceous earth (uncalcined)	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Fumed silica (respirable dust)	2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Fume (thermally generated)(respirable dust)	2 mg/m3	Not Available	Not Available	(e) Containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Fumed silica (respirable dust)	2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium oxide	Aluminium oxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	ethylene glycol monobutyl ether	2-Butoxyethanol	20 ppm / 96.9 mg/m3	242 mg/m3 / 50 ppm	Not Available	Not Available
Australia Exposure Standards	silica crystalline - quartz	Quartz (respirable dust)	0.05 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silica crystalline - quartz	Silica - Crystalline: Quartz (respirable dust)	0.05 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	dimethylethanolamine	Dimethylaminoethanol	2 ppm / 7.4 mg/m3	22 mg/m3 / 6 ppm	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
C.I. Pigment White 6	5,000 mg/m3	Not Available
propylene glycol	Not Available	Not Available
C.I. Pigment Blue 29	Not Available	Not Available
ferric hydroxide	2,500 mg/m3	Not Available
C.I. Pigment Black 7	1,750 mg/m3	Not Available
calcium carbonate	Not Available	Not Available
dipropylene glycol monomethyl ether	600 ppm	Not Available
4-nonylphenol, branched, ethoxylated	Not Available	Not Available
kaolin	Not Available	Not Available
ferric oxide	2,500 mg/m3	Not Available
silica amorphous	3,000 mg/m3	Not Available
aluminium oxide	Not Available	Not Available
ammonium hydroxide	Not Available	Not Available
tridecyl alcohol, ethoxylated, phosphated, potassium salt	Not Available	Not Available
sodium dioctyl sulfosuccinate	Not Available	Not Available
trimethylolpropane	Not Available	Not Available
ethylene glycol monobutyl ether	700 ppm	Not Available
polypropylene glycol	Not Available	Not Available
silica crystalline - quartz	25 mg/m3 / 50 mg/m3	Not Available
diethylene glycol monomethyl ether	Not Available	Not Available
C.I. Pigment Red 266	Not Available	Not Available
alcohols C12-14 ethoxylated propoxylated	Not Available	Not Available

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Ingredient	Original IDLH	Revised IDLH
dimethylethanolamine	Not Available	Not Available
nonylphenol ethoxylates	Not Available	Not Available
2-naphthol	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
4-nonylphenol, branched, ethoxylated	E	≤ 0.1 ppm
ammonium hydroxide	E	≤ 0.1 ppm
tridecyl alcohol, ethoxylated, phosphated, potassium salt	E	≤ 0.01 mg/m³
sodium dioctyl sulfosuccinate	E	≤ 0.01 mg/m³
trimethylolpropane	E	≤ 0.01 mg/m³
diethylene glycol monomethyl ether	E	≤ 0.1 ppm
C.I. Pigment Red 266	E	≤ 0.01 mg/m³
alcohols C12-14 ethoxylated propoxylated	E	≤ 0.1 ppm
nonylphenol ethoxylates	E	≤ 0.1 ppm
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:

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 unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure.
- Chemical goggles. Whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted. [AS/NZS 1337.1, EN166 or national equivalent]
- Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection.
- Alternatively a gas mask may replace splash goggles and face shields.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of

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

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

* CPI - Chemwatch Performance Index

Respiratory protection

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

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

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

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

- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.

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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.

- 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

· Respirators may be necessary when engineering and administrative controls do not adequately prevent exposures.
· The decision to use respiratory protection should be based on professional judgment that takes into account toxicity information, exposure measurement data, and frequency and likelihood of the worker's exposure - ensure users are not subject to high thermal loads which may result in heat stress or distress due to personal protective equipment (powered, positive flow, full face apparatus may be an option).
· Published occupational exposure limits, where they exist, will assist in determining the adequacy of the selected respiratory protection. These may be government mandated or vendor recommended.
· Certified respirators will be useful for protecting workers from inhalation of particulates when properly selected and fit tested as part of a complete respiratory protection program.
· Where protection from nuisance levels of dusts are desired, use type N95 (US) or type P1 (EN143) dust masks. Use respirators and components tested and approved under appropriate government standards such as NIOSH (US) or CEN (EU)
· Use approved positive flow mask if significant quantities of dust becomes airborne.
· Try to avoid creating dust conditions.
Where significant concentrations of the material are likely to enter the breathing zone, a Class P3 respirator may be required.
Class P3 particulate filters are used for protection against highly toxic or highly irritant particulates.
Filtration rate: Filters at least 99.95% of airborne particles
Suitable for:
· Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.
· Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.
· Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS
· Highly toxic particles e.g. Organophosphate Insecticides, Radionuclides, Asbestos
Note: P3 Rating can only be achieved when used with a Full Face Respirator or Powered Air-Purifying Respirator (PAPR). If used with any other respirator, it will only provide filtration protection up to a P2 rating.

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

Continued...

Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation. Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination 51cnsde Effects on lungs are significantly enhanced in the presence of respirable particles. Overexposure to respirable dust may produce wheezing, coughing and breathing difficulties leading to or symptomatic of impaired respiratory function.
Ingestion	Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing.
Skin Contact	Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact. Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. 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. 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.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
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. 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. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Overexposure to the breathable dust may cause coughing, wheezing, difficulty in breathing and impaired lung function. Chronic symptoms may include decreased vital lung capacity and chest infections. Repeated exposures in the workplace to high levels of fine-divided dusts may produce a condition known as pneumoconiosis, which is the lodgement of any inhaled dusts in the lung, irrespective of the effect. This is particularly true when a significant number of particles less than 0.5 microns (1/50000 inch) are present. Lung shadows are seen in the X-ray. Symptoms of pneumoconiosis may include a progressive dry cough, shortness of breath on exertion, increased chest expansion, weakness and weight loss. As the disease progresses, the cough produces stringy phlegm, vital capacity decreases further, and shortness of breath becomes more severe. Other signs or symptoms include changed breath sounds, reduced oxygen uptake during exercise, emphysema and rarely, pneumothorax (air in the lung cavity). Removing workers from the possibility of further exposure to dust generally stops the progress of lung abnormalities. When there is high potential for worker exposure, examinations at regular period with emphasis on lung function should be performed. Inhaling dust over an extended number of years may cause pneumoconiosis, which is the accumulation of dusts in the lungs and the subsequent tissue reaction. This may or may not be reversible.

	TOXICITY	IRRITATION
Folk Art Multi Surface Paints	Not Available	Not Available
C.I. Pigment White 6	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): 300ug/3D (intermittent) - Mild
	Oral (Rat) LD50: >=2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
propylene glycol	Dermal (rabbit) LD50: 11890 mg/kg[2]	Eye (Rodent - rabbit): 100mg - Mild
	Inhalation (Rat) LC50: >44.9 mg/l4h[1]	Eye (Rodent - rabbit): 500mg/24H - Mild
	Oral (Rat) LD50: 20000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin (Human - child): 30%/96H(continuous) - Moderate
		Skin (Human - man): 10%/2D
		Skin (Human - woman): 30%/96H - Mild
		Skin (Human): 104mg/3D (intermittent) - Moderate
		Skin (Human): 20%
		Skin (Human): 500mg/7D - Mild
		Skin: no adverse effect observed (not irritating)[1]

Continued...

C.I. Pigment Blue 29	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >10000 mg/kg[2]	Not Available
ferric hydroxide	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >10000 mg/kg[2]	Not Available
C.I. Pigment Black 7	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
calcium carbonate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (Rodent - rabbit): 750ug/24H - Severe
	Inhalation (Rat) LC50: >3 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin (Rodent - rabbit): 500mg/24H - Moderate
		Skin: no adverse effect observed (not irritating)[1]
dipropylene glycol monomethyl ether	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 9500 mg/kg[2]	Eye (Human): 8mg - Mild
	Oral (Rat) LD50: 5135 mg/kg[2]	Eye (Rodent - rabbit): 500mg/24H - Mild
		Eye: no adverse effect observed (not irritating)[1]
		Skin (Rodent - rabbit): 500mg - Mild
		Skin: no adverse effect observed (not irritating)[1]
4-nonylphenol, branched, ethoxylated	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 1310 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
		Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
kaolin	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
ferric oxide	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >5000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
silica amorphous	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (Rodent - rabbit): 25mg/24H - Mild
	Inhalation (Rat) LC50: >0.09<0.84 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >1000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
aluminium oxide	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: >0.888 mg/l4h[1]	Not Available
	Oral (Rat) LD50: >2000 mg/kg[1]	
ammonium hydroxide	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: 2000 ppm4h[2]	Eye (Rodent - rabbit): 1mg/30S - Severe
	Oral (Rat) LD50: 350 mg/kg[2]	Eye (Rodent - rabbit): 250ug - Severe
		Eye (Rodent - rabbit): 44ug - Severe
tridecyl alcohol, ethoxylated, phosphated, potassium salt	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
sodium dioctyl sulfosuccinate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 2525 mg/kg[1]	Eye (Rodent - rabbit): 1% - Severe
	Oral (Rat) LD50: >1320 mg/kg[1]	Eye (Rodent - rabbit): 10%/24H - Severe
		Eye (Rodent - rabbit): 10%/5D - Severe
		Eye (Rodent - rabbit): 250ug - Mild
		Eye: adverse effect observed (irritating)[1]
		Skin (Rodent - rabbit): 10mg/24H - Moderate
		Skin: adverse effect observed (irritating)[1]

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trimethylolpropane	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >500 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: >0.29 mg/l4h[2]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Mouse) LD50; 14000 mg/kg[2]	
ethylene glycol monobutyl ether	**TOXICITY**	**IRRITATION**
	Dermal (Guinea Pig) LD50: 210 mg/kg[2]	Eye (Rodent - rabbit): 100mg/24H - Moderate
	Inhalation (Rat) LC50: 450 ppm4h[2]	Eye: adverse effect observed (irritating)[1]
	Oral (Rat) LD50: 250 mg/kg[2]	Skin (Rodent - rabbit): 500mg - Mild
		Skin: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
polypropylene glycol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 500 mg/kg[2]	Eye (Rodent - rabbit): 500mg - Mild
	Inhalation (Rat) LC50: >2.34 mg/l4h[1]	Eye (Rodent - rabbit): 500mg/24H - Mild
	Oral (Rat) LD50: >2000 mg/kg[1]	Eye (Rodent - rabbit): 500mg/24H - Mild
		Eye (Rodent - rabbit): 500mg/24H - Mild
		Eye: no adverse effect observed (not irritating)[1]
		Skin (Rodent - rabbit): 500mg - Mild
		Skin (Rodent - rabbit): 500mg - Mild
		Skin (Rodent - rabbit): 500mg - Mild
		Skin (Rodent - rabbit): 500mg - Mild
		Skin (Rodent - rabbit): 500mg - Mild
		Skin (Rodent - rabbit): 500mg/24H - Mild
		Skin (Rodent - rabbit): 500mg/24H - Mild
		Skin: no adverse effect observed (not irritating)[1]
silica crystalline - quartz	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 500 mg/kg[2]	Not Available
diethylene glycol monomethyl ether	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 2525 mg/kg[2]	Eye (Rodent - rabbit): 500mg - Moderate
	Oral (Rat) LD50: 4040 mg/kg[2]	Eye (Rodent - rabbit): 500mg/24H - Mild
		Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
C.I. Pigment Red 266	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Not Available
	Inhalation (Rat) LC50: >1.58 mg/L4h[1]	
	Oral (Rat) LD50: >2000 mg/kg[1]	
alcohols C12-14 ethoxylated propoxylated	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 2290 mg/kg[2]	Not Available
	Oral (Rat) LD50: 3530 mg/kg[2]	
dimethylethanolamine	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 1219 mg/kg[1]	Eye (Rodent - rabbit): 5uL - Severe
	Inhalation (Mouse) LC50: 3.25 mg/L4h[2]	Eye: adverse effect observed (irreversible damage)[1]
	Oral (Rat) LD50: 1182.7 mg/kg[1]	Skin (Rodent - rabbit): 445mg - Mild
		Skin: adverse effect observed (corrosive)[1]
		Skin: adverse effect observed (irritating)[1]
nonylphenol ethoxylates	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 2943.2 mg/kg[2]	Eye (Rodent - guinea pig): 20mg - Severe
	Oral (Rat) LD50: 1310 mg/kg[2]	Eye (Rodent - mouse): 20mg - Severe
		Eye (Rodent - rabbit): 100mg - Severe
		Eye (Rodent - rabbit): 15mg - Severe
		Eye (Rodent - rabbit): 20mg - Severe
		Eye (Rodent - rabbit): 5mg - Severe

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	Eye (Rodent - rabbit): 5mg - Severe
	Eye (Rodent - rabbit): 5mg - Severe
	Eye (Rodent - rat): 20mg
	Skin (Human): 15mg/3D (intermittent) - Mild
	Skin (Rodent - rabbit): 500mg - Mild
	Skin (Rodent - rabbit): 500mg - Mild
	Skin (Rodent - rabbit): 500mg - Mild
	Skin (Rodent - rabbit): 500mg - Mild
	Skin (Rodent - rabbit): 500mg - Mild
	Skin (Rodent - rabbit): 500mg - Mild

2-naphthol	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2500 mg/kg[2]	Eye: adverse effect observed (irreversible damage)[1]
	Inhalation (Rat) LC50: 2.2 mg/l4h[2]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: 1300 mg/kg[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***

C.I. PIGMENT WHITE 6	Substance has been investigated as a mutagen, tumorigen and primary irritant. 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.
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.

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	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.
C.I. PIGMENT BLUE 29	NOTE: 90 day (chronic), teratological and mutagenicity tests here all provided negative results. Animal tests have also demonstrated no skin irritation or sensitization. [ICI]
CALCIUM CARBONATE	No evidence of carcinogenic properties. No evidence of mutagenic or teratogenic effects.
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. 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.
4-NONYLPHENOL, BRANCHED, ETHOXYLATED	for linear material: Maternal effects, effects on fertility recorded.
KAOLIN	for bentonite clays:

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	Bentonite (CAS No. 1302-78-9) consists of a group of clays formed by crystallisation of vitreous volcanic ashes that were deposited in water. The expected acute oral toxicity of bentonite in humans is very low (LD50>15 g/kg). However, severe anterior segment inflammation, uveitis and retrocorneal abscess from eye exposure were reported when bentonite had been used as a prophypaste. In a 33 day dietary (2 and 6%) and a 90 day dietary (1, 3 and 5%) studies in chickens, no changes in behaviour, overall state, clinical and biochemical parameters and electrolytic composition of the blood. Repeat dietary administration of bentonite did not affect calcium or phosphorus metabolism. However, larger amounts caused decreased growth, muscle weakness, and death with marked changes in both calcium and phosphorus metabolism. Bentonite did not cause fibrosis after 1 year exposure of 60 mg dust (<5 um) in a rat study. However, in a second rat study, where 5 um particles were intratracheally instilled at 5, 15 and 45 mg/rat, dose-related fibrosis was observed. Bentonite clay dust is believed to be responsible for bronchial asthma in workers at a processing plant in USA. Ingestion of bentonite without adequate liquids may result in intestinal obstruction in humans. Hypokalaemia and microcytic iron-deficiency anaemia may occur in patients after repeat doses of clay. Chronic ingestion has been reported to cause myositis.
SILICA AMORPHOUS	Reports indicate high/prolonged exposures to amorphous silicas induced lung fibrosis in experimental animals; in some experiments these effects were reversible. [PATTYS] For silica amorphous: Derived No Adverse Effects Level (NOAEL) in the range of 1000 mg/kg/d. In humans, synthetic amorphous silica (SAS) is essentially non-toxic by mouth, skin or eyes, and by inhalation. Epidemiology studies show little evidence of adverse health effects due to SAS. Repeated exposure (without personal protection) may cause mechanical irritation of the eye and drying/cracking of the skin. When experimental animals inhale synthetic amorphous silica (SAS) dust, it dissolves in the lung fluid and is rapidly eliminated. If swallowed, the vast majority of SAS is excreted in the faeces and there is little accumulation in the body. Following absorption across the gut, SAS is eliminated via urine without modification in animals and humans. SAS is not expected to be broken down (metabolised) in mammals. After ingestion, there is limited accumulation of SAS in body tissues and rapid elimination occurs. Intestinal absorption has not been calculated, but appears to be insignificant in animals and humans. SASs injected subcutaneously are subjected to rapid dissolution and removal. There is no indication of metabolism of SAS in animals or humans based on chemical structure and available data. In contrast to crystalline silica, SAS is soluble in physiological media and the soluble chemical species that are formed are eliminated via the urinary tract without modification. Both the mammalian and environmental toxicology of SASs are significantly influenced by the physical and chemical properties, particularly those of solubility and particle size. SAS has no acute intrinsic toxicity by inhalation. Adverse effects, including suffocation, that have been reported were caused by the presence of high numbers of respirable particles generated to meet the required test atmosphere. These results are not representative of exposure to commercial SASs and should not be used for human risk assessment. Though repeated exposure of the skin may cause dryness and cracking, SAS is not a skin or eye irritant, and it is not a sensitiser. Repeated-dose and chronic toxicity studies confirm the absence of toxicity when SAS is swallowed or upon skin contact. Long-term inhalation of SAS caused some adverse effects in animals (increases in lung inflammation, cell injury and lung collagen content), all of which subsided after exposure. Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted with SAS in a number of species, at airborne concentrations ranging from 0.5 mg/m3 to 150 mg/m3. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m3. When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m3. The difference in values may be explained by different particle size, and therefore the number of particles administered per unit dose. In general, as particle size decreases so does the NOAEL/LOAEL. Neither inhalation nor oral administration caused neoplasms (tumours). SAS is not mutagenic in vitro. No genotoxicity was detected in in vivo assays. SAS does not impair development of the foetus. Fertility was not specifically studied, but the reproductive organs in long-term studies were not affected. For Synthetic Amorphous Silica (SAS) Repeated dose toxicity Oral (rat), 2 weeks to 6 months, no significant treatment-related adverse effects at doses of up to 8% silica in the diet. Inhalation (rat), 13 weeks, Lowest Observed Effect Level (LOEL) =1.3 mg/m3 based on mild reversible effects in the lungs. Inhalation (rat), 90 days, LOEL = 1 mg/m3 based on reversible effects in the lungs and effects in the nasal cavity. For silane treated synthetic amorphous silica: Repeated dose toxicity: oral (rat), 28-d, diet, no significant treatment-related adverse effects at the doses tested. There is no evidence of cancer or other long-term respiratory health effects (for example, silicosis) in workers employed in the manufacture of SAS. Respiratory symptoms in SAS workers have been shown to correlate with smoking but not with SAS exposure, while serial pulmonary function values and chest radiographs are not adversely affected by long-term exposure to SAS.
TRIDECYL ALCOHOL, ETHOXYLATED, PHOSPHATED, POTASSIUM SALT	for alkyl alcohol alkoxylate phosphate (AAAPD) surfactants (alkyl or alcohol ether phosphates): **Acute toxicity:** This group of surfactants exhibits similar effects to the alcohol ether sulfates (AAASDs) (typically sodium lauryl ether sulfate - SLES - CAS RN 68891-38-3). They are likely to be skin/ eye irritants (R36/38) in their undiluted forms but not acutely toxic. The reported oral LD50 values were higher than 1600 mg/kg for the alkyl ether phosphates family described by CAS RN: 9046-01-9. No effects were found at any concentration tested dermally. Commercial products may contain excess phosphoric acid and may produce serious eye irritation (R41) or may even be classified as corrosive, acidic substances. **Subchronic toxicity:** Data for sulfate derivatives has been identified in the public domain**.** Subchronic 21-day repeat dose dietary studies showed low toxicity of compounds with carbon lengths of C12-15, C12-14 and C13-15 with sodium or ammonium alkyl ethoxylates with POE (polyoxyethylene) n=3. One study indicated that C16-18 POE n=18 had comparable low toxicity. No-observed-adverse-effect levels (NOAELs) range from 120 to 468 mg/kg/day, similar to a NOAEL from a 90-day rat gavage study with NaC12-14 POE n=2(CAS RN 68891-38-3), which was reported to be 225 mg/kg/day. In addition, another 90-day repeat dose dietary study with NaC12-15 POE n=3 (CAS RN 68424-50-0) resulted in low toxicity, with a NOAEL of greater than approximately 50 mg/kg/day (calculated based on dose of 1000 ppm in diet). Effects were usually related to hepatic hypertrophy, increased liver weight, and related increases in haematological endpoints related to liver enzyme induction. SLES was evaluated for effects on the reproduction and prenatal/postnatal development of the rat when administered orally via the drinking water through two successive generations. Based on this study an overall no-observed-adverse-effect level (NOAEL) for systemic effects was 0.1%, which was 86.6 mg/kg/day for the F0 generation, and 149.5 mg/kg/day for the F1 generation. The NOAEL of 86.6 mg/kg/day was selected as the toxicology endpoint for the chronic risk assessment for the sulfate derivatives **Genotoxicity:** Alcohol ether phosphates are unlikely to be genotoxic by analogy with their alcohol ether sulfate equivalents. **Carcinogenicity:** Chronic dietary studies conducted with rats on sulfate derivatives showed no incidence of cancer and no effects at the concentrations tested (lowest dose tested was ca 75 mg/kg/day).] **Reproductive and developmental toxicity:** Studies with sulfate derivatives showed little to no toxicity in dams or pups with the NOEL in a developmental toxicity study in rats with SLES at the limit dose of 1000 mg/kg/day and a reproductive NOAEL of 0.3% in drinking water (equivalent to 300 mg/kg/day), the highest dose tested in a two-generation reproduction study. In studies with phosphate derivatives, the reproductive/ developmental NOAEL for an OECD 422 study with CAS 681340-47-2 was 800 mg/kg/day, the highest dose tested, and for CAS RN 78330-24-2 the NOEL was 200 mg/kg/day. An NOAEL of 200 mg/kg/day was selected as the toxicological endpoint for he chronic risk assessment for phosphate derivatives by the US EPA. Both alcohol ether sulfates and phosphates have been evaluated in acute, subchronic, developmental and reproductive studies capable of detecting effects on endocrine mediated events. The results of these studies did not give any indication of a treatment-related effect on the oestrogen receptor or endocrine system**.** **Metabolic fate:** For compounds of comparable C16 carbon chain, the metabolites of the lower molecular weight ethoxylated (POE n=3) alcohol ether sulfate surfactants are readily absorbed and excreted primarily in the urine whereas the C16 surfactants with increased ethoxylation (POE n=9) are poorly absorbed and excreted primarily in the faeces There was also no evidence of hydrolysis of the sulfate group from C16 POE n= 3 and C16 POE n=9 or of metabolism of the ethoxylate portion of the molecule. With C11 POE n=3 and C12 POE n=3 metabolic studies in rats confirmed that the alkyl chain is extensively metabolised by beta- or omega oxidation leaving the ethoxysulfate, which is excreted directly.

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	By analogy alcohol ether phosphate esters may initially undergo metabolism to generate the corresponding alkyl alcohol alkoxylate and POE (or POE/POP - polyoxypropylene) phosphate glycol; the dephosphoralyted metabolite should be hydrolysed to the POE (or POE/POP) polyalkoxylate glycols and linear branched saturated and unsaturated alkyl alcohol metabolites. The resultant alkyl alcohol metabolites would be oxidised in fatty acid oxidation pathways. The polyalkoxylate glycols may either be conjugated and excreted unchanged or hydrolysed/ oxidised to various degraded metabolites before bring conjugated and excreted **Sensitising potential:** 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.
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 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.

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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 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

ETHYLENE GLYCOL MONOBUTYL ETHER

NOTE: Changes in kidney, liver, spleen and lungs are observed in animals exposed to high concentrations of this substance by all routes. ** ASCC (NZ) SDS
For ethylene glycol monoalkyl ethers and their acetates (EGMAEs):
Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates.
EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers.
Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are

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	similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE ***in vitro*** than those of rats. **Repeat dose toxicity:** The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA ***in vitro*** and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA ***in vitro***. **Mutagenicity:** In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in ***S. typhimurium*** strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. ***In vitro*** cytogenicity and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and in vivo micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic. **Carcinogenicity:** In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity **Reproductive and developmental toxicity**. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes). Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m3 and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m3), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m3), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m3) indicate that the members of the category are not teratogenic. The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m3 (rabbit-EGPE), 100 ppm or 425 mg/m3 (rat-EGPE), 50 ppm or 241 mg/m3 (rat EGBE) and 100 ppm or 483 mg/m3 (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m3 (rat and rabbit-EGHE). Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species. At least one researcher has stated that the reproductive effects were less than that of other monoalkyl ethers of ethylene glycol. Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility. Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions (1). The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of erythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration -dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma. 1: NTP Toxicology Program Technical report Series 484, March 2000.
POLYPROPYLENE GLYCOL	** Rohm and Haas Paraplex WP-1 MSDS
SILICA CRYSTALLINE - QUARTZ	**WARNING:** For inhalation exposure ONLY: This substance has been classified by the IARC as Group 1: **CARCINOGENIC TO HUMANS** The International Agency for Research on Cancer (IARC) has classified occupational exposures to **respirable** (<5 um) crystalline silica as being carcinogenic to humans . This classification is based on what IARC considered sufficient evidence from epidemiological studies of humans for the carcinogenicity of inhaled silica in the forms of quartz and cristobalite. Crystalline silica is also known to cause silicosis, a non-cancerous lung disease. Intermittent exposure produces; focal fibrosis, (pneumoconiosis), cough, dyspnoea, liver tumours. * Millions of particles per cubic foot (based on impinger samples counted by light field techniques). NOTE : the physical nature of quartz in the product determines whether it is likely to present a chronic health problem. To be a hazard the material must enter the breathing zone as respirable particles.
DIETHYLENE GLYCOL MONOMETHYL ETHER	For diethylene glycol monoalkyl ethers and their acetates: This category includes diethylene glycol ethyl ether (DGEE), diethylene glycol propyl ether (DGPE) diethylene glycol butyl ether (DGBE) and diethylene glycol hexyl ether (DGHE) and their acetates. **Acute toxicity:** There are adequate oral, inhalation and/or dermal toxicity studies on the category members. Oral LD50 values in rats for all category members are all > 3000 mg/kg bw, with values generally decreasing with increasing molecular weight. Four to eight hour acute inhalation toxicity studies were conducted for all category members except DGPE in rats at the highest vapour concentrations achievable. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 2000 mg/kg bw (DGHE) to 15000 mg/kg bw (DGEEA). Signs of acute toxicity in rodents are consistent with non-specific CNS depression typical of organic solvents in general. All category members are slightly irritating to skin and slightly to moderately irritating to eyes (with the exception of DGHE, which is highly irritating to eyes). Sensitisation tests with DGEE, DGEEA, DGPE, DGBE and DGBEA in animals and/or humans were negative. **Repeat dose toxicity**: Valid oral studies conducted using DGEE, DGPE, DGBEA, DGHE and the supporting chemical DGBE ranged in duration from 30 days to 2 years. Effects predominantly included kidney and liver toxicity, absolute and/or relative changes in organ weights, and some changes in haematological parameters. All effects were seen at doses greater than 800-1000 mg/kg bw/day from oral or dermal studies; no systemic effects were observed in inhalation studies with less than continuous exposure regimens. **Mutagenicity:** DGEE, DGEEA, DGBE, DGBEA and DGHE generally tested negative for mutagenicity in ***S. typhimurium*** strains TA98, TA100, TA1535, TA1537 and TA1538 and DGBEA tested negative in E. coli WP2uvrA, with and without metabolic activation. ***In vitro*** cytogenicity and sister chromatid exchange assays with DGBE and DGHE in Chinese Hamster Ovary Cells with and without metabolic activation and ***in vivo*** micronucleus or cytogenicity tests with DGEE, DGBE and DGHE in rats and mice were negative, indicating that these diethylene glycol ethers are not likely to be genotoxic. **Reproductive and developmental toxicity:** Reliable reproductive toxicity studies on DGEE, DGBE and DGHE show no effect on fertility at the highest oral doses tested (4,400 mg/kg/day for DGEE in the mouse and 1,000 mg/kg/day for DGBE and DGHE in the rat). The dermal NOAEL for reproductive toxicity in rats administered DGBE also was the highest dose tested (2,000 mg/kg/day). Although decreased sperm motility was noted in F1 mice treated with 4,400 mg/kg/day DGEE in drinking water for 14 weeks, sperm concentrations and morphology, histopathology of the testes and fertility were not affected. Results of the majority of adequate repeated dose toxicity studies in which reproductive organs were examined indicate that DGPE and DGBEA do not cause toxicity to reproductive organs (including the testes). Test material-related testicular toxicity was not noted in the majority of the studies with DGEE or DGEEA. Results of the developmental toxicity studies conducted with DGEE, DGBE and DGHE are almost exclusively negative. In these studies, effects on the foetus are generally not observed (even at concentrations that produced maternal toxicity). Exposure to 102 ppm (560 mg/m3) DGEE by inhalation (maximal achievable vapour concentration) or 1385 mg/kg/day DGEE by the dermal route during gestation did not cause maternal or developmental toxicity in the rat. Maternal toxicity and teratogenesis were not observed in rabbits receiving up to 1000 mg/kg/day DGBE by the dermal route during gestation; however a transient decrease in body weight was observed, which reversed by Day 21 In the mouse, the only concentration of DGEE tested (3500 mg/kg/day by gavage) caused maternal, but no foetal toxicity. Also, whereas oral administration of 2050 mg/kg/day DGBE (gavage) to the mouse and 1000 mg/kg/day DGHE (dietary) caused maternal toxicity, these doses had no effect on the developing foetus

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ALCOHOLS C12-14 ETHOXYLATED PROPOXYLATED	* [Henkel CCINFO 1450373]
DIMETHYLETHANOLAMINE	Dimethylaminoethanol pyroglutamate increased choline and acetylcholine extracellular levels in the brain's prefrontal cortex in vivo in rat experiments. It further improved spatial memory and reduced scopolamine-induced memory deficits [46]. Dimethylaminoethanol cyclohexyl carboxylate fumarate significantly enhanced working memory performance in rats in the radial arm maze According to an electroencephalogram (EEG) analysis, supplements combining vitamins and minerals with compounds containing DMAE in humans for three months showed increased alertness, attention, and overall mood improvement [48]. DMAE also improved sleep quality and was able to induce lucid dreams]. Its administration has been tested in child hyperkinetic syndrome [50] and minimal brain dysfunction syndrome THe daily dosage should be 500–2000 mg in the form of DMAE bitartrate. It is contraindicated during pregnancy, lactation, and in patients with schizophrenia While it is difficult to generalise about the full range of potential health effects posed by exposure to the many different amine compounds, characterised by those used in the manufacture of polyurethane and polyisocyanurate foams, it is agreed that overexposure to the majority of these materials may cause adverse health effects. ▸ Many amine-based compounds can induce histamine liberation, which, in turn, can trigger allergic and other physiological effects, including bronchoconstriction or bronchial asthma and rhinitis. ▸ Systemic symptoms include headache, nausea, faintness, anxiety, a decrease in blood pressure, tachycardia (rapid heartbeat), itching, erythema (reddening of the skin), urticaria (hives), and facial edema (swelling). Systemic effects (those affecting the body) that are related to the pharmacological action of amines are usually transient. Typically, there are four routes of possible or potential exposure: inhalation, skin contact, eye contact, and ingestion. **Inhalation:** Inhalation of vapors may, depending upon the physical and chemical properties of the specific product and the degree and length of exposure, result in moderate to severe irritation of the tissues of the nose and throat and can irritate the lungs. Products with higher vapour pressures have a greater potential for higher airborne concentrations. This increases the probability of worker exposure. Higher concentrations of certain amines can produce severe respiratory irritation, characterised by nasal discharge, coughing, difficulty in breathing, and chest pains. Chronic exposure via inhalation may cause headache, nausea, vomiting, drowsiness, sore throat, bronchopneumonia, and possible lung damage. Also, repeated and/or prolonged exposure to some amines may result in liver disorders, jaundice, and liver enlargement. Some amines have been shown to cause kidney, blood, and central nervous system disorders in laboratory animal studies. While most polyurethane amine catalysts are not sensitisers, some certain individuals may also become sensitized to amines and may experience respiratory distress, including asthma-like attacks, whenever they are subsequently exposed to even very small amounts of vapor. Once sensitised, these individuals must avoid any further exposure to amines. Although chronic or repeated inhalation of vapor concentrations below hazardous or recommended exposure limits should not ordinarily affect healthy individuals, chronic overexposure may lead to permanent pulmonary injury, including a reduction in lung function, breathlessness, chronic bronchitis, and immunologic lung disease. Inhalation hazards are increased when exposure to amine catalysts occurs in situations that produce aerosols, mists, or heated vapors. Such situations include leaks in fitting or transfer lines. Medical conditions generally aggravated by inhalation exposure include asthma, bronchitis, and emphysema. **Skin Contact:** Skin contact with amine catalysts poses a number of concerns. Direct skin contact can cause moderate to severe irritation and injury-i.e., from simple redness and swelling to painful blistering, ulceration, and chemical burns. Repeated or prolonged exposure may also result in severe cumulative dermatitis. Skin contact with some amines may result in allergic sensitisation. Sensitised persons should avoid all contact with amine catalysts. Systemic effects resulting from the absorption of the amines through skin exposure may include headaches, nausea, faintness, anxiety, decrease in blood pressure, reddening of the skin, hives, and facial swelling. These symptoms may be related to the pharmacological action of the amines, and they are usually transient. **Eye Contact:** Amine catalysts are alkaline in nature and their vapours are irritating to the eyes, even at low concentrations. Direct contact with the liquid amine may cause severe irritation and tissue injury, and the "burning" may lead to blindness. (Contact with solid products may result in mechanical irritation, pain, and corneal injury.) Exposed persons may experience excessive tearing, burning, conjunctivitis, and corneal swelling. The corneal swelling may manifest itself in visual disturbances such as blurred or "foggy" vision with a blue tint ("blue haze") and sometimes a halo phenomenon around lights. These symptoms are transient and usually disappear when exposure ceases. Some individuals may experience this effect even when exposed to concentrations below doses that ordinarily cause respiratory irritation. **Ingestion:** The oral toxicity of amine catalysts varies from moderately to very toxic. Some amines can cause severe irritation, ulceration, or burns of the mouth, throat, esophagus,and gastrointestinal tract. Material aspirated (due to vomiting) can damage the bronchial tubes and the lungs. Affected persons also may experience pain in the chest or abdomen, nausea, bleeding of the throat and the gastrointestinal tract, diarrhea, dizziness, drowsiness, thirst, circulatory collapse, coma, and even death. **Polyurethane Amine Catalysts: Guidelines for Safe Handling and Disposal; Technical Bulletin June 2000** **Alliance for Polyurethanes Industry** For dimethylethanolamine (DMAE) and selected salts and esters: **Toxicology:** **Humans**: 10 to 20 mg (0.042-0.084 mmol) of DMAE tartrate administered orally to humans, produced mild mental stimulation. At 20 mg/day (0.084 mmol), there was a gradual increase in muscle tone and perhaps an increased frequency of convulsions in susceptible individuals. Larger doses (not specified) produced insomnia, muscle tenseness, and spontaneous muscle twitches. Doses of DMAE as high as 1200 mg/day (13.46 mmol/day) produced no serious side effects. A single 2500-mg (27.80-mmol) dose taken in a suicide attempt had no adverse effect. A single 2500-mg (27.80-mmol) dose taken in a suicide attempt had no adverse effect. DMAE supplementation is contraindicated during pregnancy and lactation It is also contraindicated for treatment of people with symptoms of schizophrenia and clonic-tonic seizure disorders The principal contraindication to the use of DMAE was grand mal epilepsy. DMAE also antagonizes the depressant effects of barbiturates. A large number of adverse health effects are associated with DMAE. These include cardiovascular, neurological, and/or psychological effects. Specific attribution of adverse effects to DMAE is unlikely, as many of these products also contained Ephedra vulgaris alkaloids and other Ephedra spp. Ephedra alkaloids cause similar cardiovascular and neurological effects reported for DMAE. DMAE, thought to be a precursor for acetylcholine, has been tested for its efficacy in treating a variety of diseases possibly related to deficiencies of acetylcholine, including tardive dyskinesia, Alzheimer's disease, amnesic disorders, age-related cognitive impairment, and Tourette's syndrome, with mixed results . Treatment with DMAE for tardive dyskinesia, a side effect of neuroleptic medications, was associated with serious cholinergic side effects: nasal and oral secretions, dyspnea, and respiratory failure . DMAE was used in the treatment of one patient for a low-frequency action tremor. This treatment was successful for ten years, until side effects of increasing neck pain and orofacial and respiratory dyskinesia occurred. Treatment was discontinued, and it was concluded that the dyskinesia could be attributed to the effects of DMAE. A meta-analysis of randomized controlled trials indicated that DMAE was no more effective than placebo in the treatment of tardive dyskinesia. Rather, there was a significantly increased risk of adverse events associated with the DMAE treatment. DMAE treatment increases in the concentration of choline in both the plasma and the brain of treated rats; the mechanism for this phenomenon was unknown. Since it was known that DMAE inhibits the influx of choline to the brain across the blood brain barrier, it is possible that DMAE also inhibited the efflux of choline from the brain, resulting in an accumulation in the brain. Differential penetration of the blood-brain barrier by several DMAE derivatives has been noted. Radiolabeled DMAE p-chlorophenoxyacetate was found in higher concentrations in the brain than radiolabeled DMAE after intravenous treatment of mice. Higher levels of DMAE were found in the brain after dosing with centrophenoxine than with DMAE, possibly due to improved penetration of the blood-brain barrier by the esterified form of DMAE. Similarly radiolabeled cyprodenate maleate (the cyclohexylpropionic acid ester of DMAE) was more rapidly absorbed and accumulated to a large extent in the brain.

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Choline, or trimethylaminoethanol, may be formed by methylation of DMAE . Choline is an essential nutrient. Although small amounts may be synthesised, choline must be supplemented through the diet to maintain adequate physiological concentrations for optimal health.
Choline is a precursor for the neurotransmitter, acetylcholine. As a possible precursor of choline, DMAE has also been studied as a potential modulator of many biological processes requiring choline; these include the production of structural components of cell membranes (the phospholipids, especially phosphatidylcholine and sphingomyelin), the synthesis of intracellular signalling molecules (diacylglycerol and ceramide) , platelet activating factor and spingophosphorylcholine. Phosphatidylcholine is a required component of very low-density lipoproteins (VLDL) particles, necessary for the transportation of cholesterol and fat from the liver to other sites in the body. Betaine, a metabolite of choline, participates in methyl-group transfer.
In one occupational study in the manufacture of polyurethane foam insulation for refrigerators, adverse effects included disorders of the upper respiratory tract and nervous system, along with significant changes in the immune status of workers exposed to a mixture of DMAE, ethylenediamine, propylene oxide, and 4,4'-methylenediphenyl diisocyanate . A spray painter developed severe respiratory symptoms, which seemed to be related to occupational exposure to a specific type of spray paint containing DMAE. Follow-on skin tests with DMAE (undiluted, and 1:10 and 1:100 dilutions in saline) in three human volunteers produced wheal and flare responses at the high dose. This was interpreted as an irritant response, and not a sign of immunotoxicity . Despite one clear case for occupational asthma form DMAE exposure, it fails to meet the current criteria for classification as a respiratory sensitiser
Neurotoxicity: Using a method to classify the risks associated with occupational exposures to neurotoxic chemicals obtained from four national computer-based registers, DMAE produces a small increase in the risk of damaging the nervous system under normal work conditions.
DMAE (as centrophenoxine, an ester of DMAE)) was tested for its effects on spinal reflexes in mice. 50 mg/kg (0.170 mmol/kg) demonstrated a considerable change in spinal reflexes, specifically in the inhibition of polysynaptic reflexes. Higher doses (400 to 600 mg/kg [1.40 to 2.04 mmol/kg] intraperitoneally) resulted in ataxia, reduced mobility, inhibition, and mortality in some treated mice. Similar doses in rats resulted in limited mobility and an inhibited state.
Intravenous administration of DMAE (175 to 350 mg/kg; 1.95 to 3.90 mmol/kg) resulted in dose-dependant psychoanaleptic effects (as demonstrated by spontaneous running in mice) and an influence on conditioned reflexes in rats.
DMAE appears to exert a central vasomotor stimulant effect. Intracerebroventricular (ICV) administration of DMAE (0.1 to 2.0 mg; 1.0 to 20 umol) resulted in potentiation of the carotid occlusion response (all doses) resulting in an increase in blood pressure in dogs (higher doses). This effect was not abolished by atropine sulfate (ICV).
With meclofenoxate (centrophenoxine hydrochloride) treatment (10 to 40 mg/kg body weight; 0.040 to 0.16 mmol/kg), a significant dose-dependent reduction in both blood pressure (up to 49.7+/-0.39 mmHg reduction) and heart rate (up to 71 +/-4.5% reduction) was observed in the old rats at the 40 mg/kg (0.16 mmol/kg) dose level
Reproductive toxicity: No histopathological changes in the gonads were observed after repeated exposure to DMAE in a 90-day inhalation study in rats
DMAE via inhalation induced maternal toxicity in rats at all tested exposure levels (10, 30, and 100 ppm; 40, 110, and 370 mg/m3; 0.41, 1.20, and 4.10 mmol/m3), as demonstrated by changes in body weight gain in the mid- and high-dose groups and ocular changes in the mid- and low-dose. Sporadic, inconsistent alterations in gestational parameters including significant decreases in viable implants per litter, percentage live foetuses/litter, and litter size in rats exposed to 10 ppm (40 mg/m3; 41 mmol/m3) and a significant decrease in the percentage of male foetuses in rats exposed to 30 ppm (110 mg/m3; 1.20 mmol/m3). Skeletal variations in foetuses included decreased incidences of poorly ossified cervical centrum , bilobed thoracic centrum , bilobed sternebrae ,unossified proximal phalanges of the forelimb, and increased incidences of split cervical centra ,and bilobed thoracic centrum . However, a consistent pattern was lacking, resulting in a NOAEL for embryofoetal toxicity and teratogenicity of 100 ppm (370 mg/m3; 4.10 mmol/m3) or greater. A NOAEL for maternal toxicity was estimated at 10 ppm (40 mg/m3; 0.41 mmol/m3).
A five-generation study was conducted; each generation of rats or only the first and fifth generations were exposed in utero to centrophenoxine on gestation days 11 to 14 (during embryogenesis), Treating Wistar dams with meclofenoxate prenatally resulted in significant increases in weight of the offspring. The increase in embryo weights did not continue into postnatal life. Continuous treatment through several generations increased fertility and an overall increase in the number of offspring
Carcinogenicity: There was no statistically significant increase, or morphological difference, in the incidence of neoplasms in any organ in female C3H/HeN mice given drinking water with 10 mM (900 ug/mL) DMAE for 105 weeks, or in female C3H/HeJ(+) mice given 15 mM (1300 ug/mL) DMAE for 123 weeks . No changes in the structure, appearance, or microscopic morphology of various organs were observed. Treatment with DMAE did not affect survival, initial body weight gain, or mature body weight of either strain of mouse
Di- and triaminoethanols, which are structurally related to DMAE and are found in cutting fluids, pesticides, and cosmetics, can give rise to N-nitrosodiethanolamine (NDELA) via nitrosation resulting from reaction with nitrite or nitrous oxide. The authors also noted that NDELA has been shown to be a potent carcinogen, producing mainly hepatocellular carcinomas in rats and epithelial neoplasms of the nasal cavity and trachea in hamsters.
Genotoxicity: Salmonella typhimurium assay. Tester strains TA98, TA100, TA1535, TA1537, and TA1538 were all tested, both in the presence and absence of a metabolic activation system. DMAE, ranging from 0.37 to 995 umol (0.033 to 89.5 mg)/plate failed to demonstrate any mutagenic response.
DMAE also failed to induce any sex-linked recessive lethal mutations in the Drosophila melanogaster (7200 or 8100 ppm; 80.10 or 90.10 mmol/L).
The genotoxicity of DMAE was investigated in several mammalian systems, both in vitro and in vivo. In vitro assays included sister chromatid exchange and hypoxanthine-guanine phosphoribosyl transferase forward gene mutation test (HGPT), both in Chinese hamster ovary cells. All of the in vitro assays failed to demonstrate genotoxicity within the dose ranges..
Immunotoxicity: DMAE was unable to covalently derivatise protein in an in vitro assay. It is thought that the ability to covalently derivatise protein enables some low-molecular-weight chemicals (LMWC) to induce allergic antibody-mediated responses that may cause asthma in people occupationally exposed to LMWC. The ability of DMAE to act as a skin sensitiser was tested in the murine local lymph node assay at 0, 3, 10, and 30% w/v (0, 33, 110, and 330 mmol/L). The test resulted in test:control ratios of 0, 1.93, 2.13, and 14.50 respectively. Typically, ratios greater than 3 are
indicative of potential sensitisers; therefore, based on this test, DMAE was classified as a potential skin sensitizer. Human experiences with DMAE under normal handling precautions have not supported this result. Similarly, DMAE, evaluated in the guinea pig maximisation procedure, was without any clear evidence of skin sensitization
Metabolism: DMAE is absorbed (either from the small intestine after oral dosing or from the bloodstream after injections), and rapidly transported to the liver where much of it is metabolised. DMAE is metabolised through the phospholipid cycle to produce phosphoryldimethylethanolamine and glycerophosphatidylcholine Pigs and rats dosed with cyprodenate maleate, the cyclohexylpropionic acid ester of DMAE, was found to be was well absorbed from the digestive tract and distributed to tissues and organs. Similarly, centrophenoxine (an ester of DMAE) was well absorbed after oral administration. After transport to the liver, a portion of centrophenoxine is converted to its constituent moieties, DMAE and p-chlorophenoxyacetic acid (PCPA), while the unmetabolised form was transported throughout the body by the circulatory system
In humans, 33% of an injected 1 g (10 mmol) dose of DMAE was excreted unchanged. It was suggested that the remaining dose may have been demethylated to ethanolamine and entered into normal metabolic pathways.

main concern with pharmaceutical drugs and dietary supplements are adverse effects. Long-term safety evidence is typically unavailable for many nootropic compounds. Racetams, piracetam and other compounds that are structurally related to piracetam, have few serious adverse effects and low toxicity, but there is little evidence that they enhance cognition in people having no cognitive impairments.
Some nootropics can increase adrenaline levels in the body, producing effects similar to drinking large amounts of caffeine. Some drugs increase the number of certain chemicals (neurotransmitters), such as dopamine, that are released in parts of the brain associated with addiction. Research into how drugs work to stimulate the mind is still inconclusive, but early research suggests that drugs may act on different systems in the body simultaneously. One explanation is that it increases blood flow to the brain, allowing it to use more oxygen.
Research into nootropics is still limited, so there are many uncertainties about the side effects the drugs may cause with continued use.
Nootropics help mask fatigue, procrastination, or boredom, but they don’t make people smarter, and their effects last as long as the drug remains in the body. Some of these drugs are addictive and have various side effects. It can be especially harmful to young people, as their brains continue to develop until their mid-twenties.
In the United States, dietary supplements may be marketed if the manufacturer can show that the supplement is generally recognized as safe, and if the manufacturer does not make any claims about using the supplement to treat or prevent any disease or condition; supplements that contain drugs or advertise health claims are illegal under US law.

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	Nootropics are a heterogeneous group of drugs that affect the metabolism of neuronal cells in the central nervous system. They mainly improve cognitive function, especially in cases where there is damage or degeneration. Most of these substances do not have an immediate effect after a single administration and must be used for some length of time before there is a measurable improvement. They are used in acute, subacute, and chronic conditions of memory, consciousness, and learning disorders and as a supportive treatment in patients with Alzheimer's disease, schizophrenia, hyperkinetic disorder, or senile dementia. Nootropics are usually very well tolerated. Side effects are rare and typically mild, but some complications can occur. For example, people with cardiovascular disease should not use guarana. This is probably due to the relatively high caffeine content nootropics users should consider their state of health and mood before deciding to try a certain compound; however, if the recommended dosage is followed, no serious complications should occur. Because of their potential for improving memory and thinking and their easy availability, nootropics have particularly attracted the attention of college students, who call them "smart drugs". Because of the incomplete clinical evidence on their effectiveness, safety, and social consequences in the case of long-term use, especially with synthetic variants of these drugs, they cannot be recommended to healthy individuals who do not suffer from any cognitive dysfunction. There have not been sufficient experimental studies and results to support prophylactic use, even though the use of herbal supplements with nootropic effects has shown little risk of side effects and contraindications have been minimal. In any case, to be safe, none of these substances should be used during pregnancy or breastfeeding. Future research regarding nootropics should focus on experiments with more diverse human groups, whether in terms of age, health, gender, or weight. It should also mainly focus on young, healthy people, mostly university students, who use these substances a lot and obtain them, especially on the black market. Furthermore, already advanced methods based on neuroimaging assessment should be used more in experiments and studies to confirm or refute the potential beneficial effects.
NONYLPHENOL ETHOXYLATES	Oral (rat) TDLo: 150 mg/kg/3D-I Skin (rabbit): 500 mg mild
2-NAPHTHOL	For 2-naphthol In workers exposed to 2-naphthol an increased incidence of dermatitis, conjunctivitis, and rhinitis have been reported in poorly documented studies. In addition, changes in kidney function, and an increased incidence in chronic hepatitis and impairment of the nervous system were reported from workers who were also exposed to a variety of other chemicals **Acute toxicity:** 2-Naphthol can be absorbed through the skin. Rapid conjugation with glucuronide and sulphate in the liver and renal excretion of the unchanged and conjugated forms seems to be the principal mechanism of elimination. The acute oral LD50 in rats was determined as 1320 mg/kg bw in a study following OECD TG 401. Clinical signs included reduced activity, accelerated breathing, closure of eyes, nasal discharge and diarrhoea, and at exposure levels near to or exceeding the LD50 also tumbling, reduced reflexes and seizures. The inhalation 4-hour-LC50 in rats was determined as 2200 mg/m3 (aerosol; OECD TG 403). Clinical signs included irregular breathing, reduced activity, impaired motility and reflexes, nasal discharge, corneal opacity and diarrhea. 2-Naphthol was not irritating to the skin of rabbits in a test performed according to OECD TG 404, but caused serious damage to the eyes of rabbits in a study in accordance with OECD TG 405 (corneal vascularization/opacity). 2-Naphthol is a skin sensitiser, based on results from a guinea pig maximization test [OECD TG 406]. An increased incidence of contact dermatitis in exposed workers is reported in an old and poorly documented study. **Repeat dose toxicity:** After repeated administration to rats by the oral route for 28 days, there were indications of a possible effect on the adrenals in both sexes at dose levels of 50 mg/kg bw/day and above (increased relative and absolute adrenal weights). At 150 mg/kg bw/day, an increase in serum creatinine and changes in serum electrolytes were found in males, indicating an effect on the kidneys. Poorly documented studies in dogs and rats involving repeated administration by the subcutaneous and inhalation route showed effects on the liver and kidneys. Concentration dependent disturbances in blood clotting and functional impairment of the liver and kidney with accompanying histopathological effects occurred at 10.1 and 1.35 mg/m3. **Genotoxicity**: 2-Naphthol was not mutagenic in several Ames tests both in the absence and in the presence of metabolic activation, even at cytotoxic concentrations. Inconsistent results have been observed in bacterial DNA repair tests, but it did not induce unscheduled DNA synthesis in rat hepatocytes in a test performed according to current standards. 2-Naphthol was not tested for its potential to induce chromosomal aberrations ***in vitro***. In an ***in vivo*** micronucleus assay with 2-naphthol, no evidence of genotoxicity was found. These data show that 2-naphthol is not mutagenic ***in vivo***. **Carcinogenicity:** There are no adequate data available for the evaluation of the carcinogenic potential of 2-naphthol. **Reproductive toxicity:** 2-Naphthol was tested for its reproductive toxicity in a one-generation study according to OECD TG 415. The administration of the test substance had no adverse effect on the reproductive abilities of the parental generation. **Developmental toxicity:** No teratogenic effects were observed (NOEL for male reproductive toxicity: 160 mg/kg bw/day (highest tested dose); NOELs for female reproductive toxicity and for toxicity to the offspring: 40 mg/kg bw/day each). (160 mg/kg bw/day may suppress nursing; reduced body weights and reduced viability was seen in the offspring at 160 mg/kg bw/day). The LOEL for systemic toxicity in males was 10 mg/kg bw/day (salivation), the NOEL for systemic toxicity in females was 10 mg/kg bw/day (nasal discharge, reduced food consumption, decreased locomotor activity and salivation at 40 mg/kg bw/day).
C.I. PIGMENT WHITE 6 & SILICA AMORPHOUS	The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
PROPYLENE GLYCOL & CALCIUM CARBONATE & SODIUM DIOCTYL SULFOSUCCINATE & POLYPROPYLENE GLYCOL	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.
FERRIC HYDROXIDE & C.I. PIGMENT BLACK 7 & KAOLIN & ALUMINIUM OXIDE & TRIDECYL ALCOHOL, ETHOXYLATED, PHOSPHATED, POTASSIUM SALT & C.I. PIGMENT RED 266 & ALCOHOLS C12-14 ETHOXYLATED PROPOXYLATED	No significant acute toxicological data identified in literature search.
CALCIUM CARBONATE & DIPROPYLENE GLYCOL MONOMETHYL ETHER & FERRIC OXIDE & AMMONIUM HYDROXIDE & DIMETHYLETHANOLAMINE & NONYLPHENOL ETHOXYLATES	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.
CALCIUM CARBONATE & 4-NONYLPHENOL, BRANCHED, ETHOXYLATED & AMMONIUM HYDROXIDE & SODIUM DIOCTYL SULFOSUCCINATE & ETHYLENE GLYCOL	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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MONOBUTYL ETHER & DIMETHYLETHANOLAMINE	
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 & 4-NONYLPHENOL, BRANCHED, ETHOXYLATED & ETHYLENE GLYCOL MONOBUTYL ETHER & DIMETHYLETHANOLAMINE & 2-NAPHTHOL	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.
4-NONYLPHENOL, BRANCHED, ETHOXYLATED & NONYLPHENOL ETHOXYLATES	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. 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.
4-NONYLPHENOL, BRANCHED, ETHOXYLATED & TRIDECYL ALCOHOL, ETHOXYLATED, PHOSPHATED, POTASSIUM SALT & POLYPROPYLENE GLYCOL & ALCOHOLS C12-14 ETHOXYLATED PROPOXYLATED & NONYLPHENOL ETHOXYLATES	Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture . On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing. 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.

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	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
4-NONYLPHENOL, BRANCHED, ETHOXYLATED & ALCOHOLS C12-14 ETHOXYLATED PROPOXYLATED & NONYLPHENOL ETHOXYLATES	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 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.

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	Metabolism: The main metabolic pathway for metabolism of ethylene glycol monoalkyl ethers (EGME, EGEE, and EGBE) is oxidation via alcohol and aldehyde dehydrogenases (ALD/ADH) that leads to the formation of an alkoxy acids. Alkoxy acids are the only toxicologically significant metabolites of glycol ethers that have been detected ***in vivo.*** The principal metabolite of TGME is believed to be 2-[2-(2-methoxyethoxy)ethoxy] acetic acid . Although ethylene glycol, a known kidney toxicant, has been identified as an impurity or a minor metabolite of glycol ethers in animal studies it does not appear to contribute to the toxicity of glycol ethers. The metabolites of category members are not likely to be metabolized to any large extent to toxic molecules such as ethylene glycol or the mono alkoxy acids because metabolic breakdown of the ether linkages also has to occur **Acute toxicity:** Category members generally display low acute toxicity by the oral, inhalation and dermal routes of exposure. Signs of toxicity in animals receiving lethal oral doses of TGBE included loss of righting reflex and flaccid muscle tone, coma, and heavy breathing. Animals administered lethal oral doses of TGEE exhibited lethargy, ataxia, blood in the urogenital area and piloerection before death. **Irritation:** The data indicate that the glycol ethers may cause mild to moderate skin irritation. TGEE and TGBE are highly irritating to the eyes. Other category members show low eye irritation. **Repeat dose toxicity**: Results of these studies suggest that repeated exposure to moderate to high doses of the glycol ethers in this category is required to produce systemic toxicity In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME. Testicular effects included spermatid giant cells, focal tubular hypospermatogenesis, and increased cytoplasmic vacuolisation . Due to a high incidence of similar spontaneous changes in normal New Zealand White rabbits , the testicular effects were considered not to be related to treatment . Thus, the NOAELs for TGME, TGEE and TGBE were established at 1000 mg/kg/day. Findings from this report were considered unremarkable. A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day . In this study, significantly-increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500 mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or haemolysed blood in the stomach These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity **Mutagenicity:** Mutagenicity studies have been conducted for several category members. All in vitro and in vivo studies were negative at concentrations up to 5,000 micrograms/plate and 5,000 mg/kg, respectively, indicating that the category members are not genotoxic at the concentrations used in these studies. The uniformly negative outcomes of various mutagenicity studies performed on category members lessen the concern for carcinogenicity. **Reproductive toxicity:** Although mating studies with either the category members or surrogates have not been performed, several of the repeated dose toxicity tests with the surrogates have included examination of reproductive organs. A lower molecular weight glycol ether, ethylene glycol methyl ether (EGME), has been shown to be a testicular toxicant. In addition, results of repeated dose toxicity tests with TGME clearly show testicular toxicity at an oral dose of 4,000 mg/kg/day four times greater that the limit dose of 1,000 mg/kg/day recommended for repeat dose studies. It should be noted that TGME is 350 times less potent for testicular effects than EGME. TGBE is not associated with testicular toxicity, TetraME is not likely to be metabolised by any large extent to 2-MAA (the toxic metabolite of EGME), and a mixture containing predominantly methylated glycol ethers in the C5-C11 range does not produce testicular toxicity (even when administered intravenously at 1,000 mg/kg/day). **Developmental toxicity**: The bulk of the evidence shows that effects on the foetus are not noted in treatments with . 1,000 mg/kg/day during gestation. At 1,250 to 1,650 mg/kg/day TGME (in the rat) and 1,500 mg/kg/day (in the rabbit), the developmental effects observed included skeletal variants and decreased body weight gain.
DIETHYLENE GLYCOL MONOMETHYL ETHER & 2-NAPHTHOL	The material may produce moderate eye irritation leading to 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 Multi Surface Paints	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available

C.I. Pigment White 6	**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

propylene glycol	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	19300mg/l	2

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	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

C.I. Pigment Blue 29

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	000mg/l	Not Available

ferric hydroxide

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	18mg/l	2
EC50	48h	Crustacea	>100mg/l	2
NOEC(ECx)	504h	Fish	0.52mg/l	2
LC50	96h	Fish	0.05mg/l	2
LC50	96h	Fish	0.05mg/l	2
NOEC(ECx)	504h	Fish	0.52mg/l	2
EC50	72h	Algae or other aquatic plants	18mg/l	2
EC50	48h	Crustacea	>100mg/l	2

C.I. Pigment Black 7

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>0.2mg/l	2
EC50	48h	Crustacea	33.076-41.968mg/l	4
LC50	96h	Fish	>100mg/l	2
NOEC(ECx)	24h	Crustacea	3200mg/l	1

calcium carbonate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>14mg/l	2
LC50	96h	Fish	>165200mg/L	4
NOEC(ECx)	1h	Fish	4-320mg/l	4

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

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

kaolin

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

ferric oxide

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	18mg/l	2
EC50	48h	Crustacea	>100mg/l	2
NOEC(ECx)	504h	Fish	0.52mg/l	2
LC50	96h	Fish	0.05mg/l	2

silica amorphous

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	14.1mg/l	2
EC50	48h	Crustacea	>86mg/l	2
LC50	96h	Fish	1033.016mg/l	2
EC50	96h	Algae or other aquatic plants	217.576mg/l	2
EC0(ECx)	24h	Crustacea	>=10000mg/l	1

aluminium oxide

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.017mg/L	2
EC50	48h	Crustacea	0.736mg/L	2
LC50	96h	Fish	0.078-0.108mg/l	2

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	NOEC(ECx)	72h	Algae or other aquatic plants	>100mg/l	1
	EC50	96h	Algae or other aquatic plants	0.005mg/L	2

ammonium hydroxide

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	33.3mg/L	4
EC50(ECx)	96h	Crustacea	0.83mg/L	5

tridecyl alcohol, ethoxylated, phosphated, potassium salt

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

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

trimethylolpropane

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1008h	Fish	0.4-2.6	7
EC50	72h	Algae or other aquatic plants	>1000mg/l	2
EC50	48h	Crustacea	10330-16360mg/L	4
LC50	96h	Fish	>100mg/l	2
EC0(ECx)	48h	Crustacea	>=102mg/l	1

ethylene glycol monobutyl ether

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	623mg/l	2
EC10(ECx)	48h	Crustacea	7.2mg/l	2
EC50	48h	Crustacea	164mg/l	2
LC50	96h	Fish	1250mg/l	2
EC50	96h	Algae or other aquatic plants	720mg/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
EC50	96h	Algae or other aquatic plants	3000-4000mg/l	2

silica crystalline - quartz

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

diethylene glycol monomethyl ether

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>500mg/l	1
EC0(ECx)	48h	Crustacea	500mg/l	1
EC50	48h	Crustacea	>500mg/l	1
LC50	96h	Fish	>969.6mg/L	4
EC50	96h	Algae or other aquatic plants	>1000mg/l	2

C.I. Pigment Red 266

Endpoint	Test Duration (hr)	Species	Value	Source
ErC50	72h	Algae or other aquatic plants	17mg/l	2
EC50	72h	Algae or other aquatic plants	>1mg/l	2
EC50	48h	Crustacea	>100mg/l	2
LC50	96h	Fish	>100mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	1mg/l	2

alcohols C12-14 ethoxylated propoxylated

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

dimethylethanolamine

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	35mg/l	1
EC0(ECx)	48h	Crustacea	62.5mg/l	1
EC50	48h	Crustacea	98.77mg/l	1

Continued...

	LC50	96h	Fish	88-131mg/l	1

nonylphenol ethoxylates

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1008h	Fish	<0.2	7
EC50	48h	Crustacea	12.2mg/L	4
LC50	96h	Fish	1-1.8mg/L	4
EC50	96h	Algae or other aquatic plants	12mg/l	4
NOEC(ECx)	2400h	Fish	0.035mg/L	4

2-naphthol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	5.8mg/l	2
EC50	48h	Crustacea	0.85mg/l	1
LC50	96h	Fish	2.43-3.9mg/L	4
NOEC(ECx)	648h	Fish	0.001mg/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***

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

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
C.I. Pigment White 6	HIGH	HIGH
propylene glycol	LOW	LOW
dipropylene glycol monomethyl ether	HIGH	HIGH
silica amorphous	LOW	LOW
trimethylolpropane	LOW	LOW
ethylene glycol monobutyl ether	LOW (Half-life = 56 days)	LOW (Half-life = 1.37 days)
polypropylene glycol	LOW	LOW
diethylene glycol monomethyl ether	LOW	LOW
dimethylethanolamine	LOW	LOW
2-naphthol	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
C.I. Pigment White 6	LOW (BCF = 10)
propylene glycol	LOW (BCF = 1)
dipropylene glycol monomethyl ether	LOW (BCF = 100)
silica amorphous	LOW (LogKOW = 0.5294)
sodium dioctyl sulfosuccinate	LOW (BCF = 3.78)
trimethylolpropane	LOW (BCF = 16.2)
ethylene glycol monobutyl ether	LOW (BCF = 2.51)
polypropylene glycol	LOW (LogKOW = 1.6984)
diethylene glycol monomethyl ether	LOW (BCF = 0.18)
dimethylethanolamine	LOW (LogKOW = -0.9351)
nonylphenol ethoxylates	LOW (BCF = 1.4)
2-naphthol	LOW (LogKOW = 2.7)

Mobility in soil

Ingredient	Mobility
C.I. Pigment White 6	LOW (Log KOC = 23.74)
propylene glycol	HIGH (Log KOC = 1)
dipropylene glycol monomethyl ether	LOW (Log KOC = 10)
silica amorphous	LOW (Log KOC = 23.74)
trimethylolpropane	HIGH (Log KOC = 1)
ethylene glycol monobutyl ether	HIGH (Log KOC = 1)
polypropylene glycol	LOW (Log KOC = 15.66)
diethylene glycol monomethyl ether	HIGH (Log KOC = 1)

Continued...

Ingredient	Mobility
dimethylethanolamine	HIGH (Log KOC = 1.602)
2-naphthol	LOW (Log KOC = 2976)

SECTION 13 Disposal considerations

Waste treatment methods

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

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
C.I. Pigment White 6	Not Available
propylene glycol	Not Available
C.I. Pigment Blue 29	Not Available
ferric hydroxide	Not Available
C.I. Pigment Black 7	Not Available
calcium carbonate	Not Available
dipropylene glycol monomethyl ether	Not Available
4-nonylphenol, branched, ethoxylated	Not Available
kaolin	Not Available
ferric oxide	Not Available
silica amorphous	Not Available
aluminium oxide	Not Available
ammonium hydroxide	Not Available
tridecyl alcohol, ethoxylated, phosphated, potassium salt	Not Available
sodium dioctyl sulfosuccinate	Not Available
trimethylolpropane	Not Available
ethylene glycol monobutyl ether	Not Available
polypropylene glycol	Not Available
silica crystalline - quartz	Not Available
diethylene glycol monomethyl ether	Not Available
C.I. Pigment Red 266	Not Available
alcohols C12-14 ethoxylated propoxylated	Not Available
dimethylethanolamine	Not Available
nonylphenol ethoxylates	Not Available
2-naphthol	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
C.I. Pigment White 6	Not Available

Continued...

Product name	Ship Type
propylene glycol	Not Available
C.I. Pigment Blue 29	Not Available
ferric hydroxide	Not Available
C.I. Pigment Black 7	Not Available
calcium carbonate	Not Available
dipropylene glycol monomethyl ether	Not Available
4-nonylphenol, branched, ethoxylated	Not Available
kaolin	Not Available
ferric oxide	Not Available
silica amorphous	Not Available
aluminium oxide	Not Available
ammonium hydroxide	Not Available
tridecyl alcohol, ethoxylated, phosphated, potassium salt	Not Available
sodium dioctyl sulfosuccinate	Not Available
trimethylolpropane	Not Available
ethylene glycol monobutyl ether	Not Available
polypropylene glycol	Not Available
silica crystalline - quartz	Not Available
diethylene glycol monomethyl ether	Not Available
C.I. Pigment Red 266	Not Available
alcohols C12-14 ethoxylated propoxylated	Not Available
dimethylethanolamine	Not Available
nonylphenol ethoxylates	Not Available
2-naphthol	Not Available

SECTION 15 Regulatory information

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

C.I. Pigment White 6 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)

propylene glycol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

C.I. Pigment Blue 29 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

ferric hydroxide is found on the following regulatory lists

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)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

C.I. Pigment Black 7 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
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)

calcium carbonate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

dipropylene glycol monomethyl ether is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

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

Continued...

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

kaolin is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

ferric oxide is found on the following regulatory lists

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 6
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

silica amorphous is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring
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 - Not Classified as Carcinogenic
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

aluminium oxide is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

ammonium hydroxide 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 6
Australian Inventory of Industrial Chemicals (AIIC)

tridecyl alcohol, ethoxylated, phosphated, potassium salt is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

sodium dioctyl sulfosuccinate is found on the following regulatory lists

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

trimethylolpropane is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

ethylene glycol monobutyl ether 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 6
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

polypropylene glycol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

silica crystalline - quartz is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring
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 1: Carcinogenic to humans
International Agency fsor Research on Cancer (IARC) - Agents Classified by the IARC Monographs

diethylene glycol monomethyl ether 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 10 / Appendix C
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

C.I. Pigment Red 266 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

alcohols C12-14 ethoxylated propoxylated is found on the following regulatory lists

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

dimethylethanolamine 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 4
Australian Inventory of Industrial Chemicals (AIIC)

nonylphenol ethoxylates is found on the following regulatory lists

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

Continued...

Chemical Footprint Project - Chemicals of High Concern List

2-naphthol 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
Canada - NDSL	No (C.I. Pigment White 6; propylene glycol; C.I. Pigment Blue 29; C.I. Pigment Black 7; dipropylene glycol monomethyl ether; 4-nonylphenol, branched, ethoxylated; kaolin; ferric oxide; aluminium oxide; ammonium hydroxide; tridecyl alcohol, ethoxylated, phosphated, potassium salt; sodium dioctyl sulfosuccinate; trimethylolpropane; ethylene glycol monobutyl ether; polypropylene glycol; silica crystalline - quartz; diethylene glycol monomethyl ether; C.I. Pigment Red 266; alcohols C12-14 ethoxylated propoxylated; dimethylethanolamine; nonylphenol ethoxylates; 2-naphthol)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (tridecyl alcohol, ethoxylated, phosphated, potassium salt; alcohols C12-14 ethoxylated propoxylated)
Japan - ENCS	No (kaolin; tridecyl alcohol, ethoxylated, phosphated, potassium salt; C.I. Pigment Red 266)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	No (tridecyl alcohol, ethoxylated, phosphated, potassium salt; C.I. Pigment Red 266; alcohols C12-14 ethoxylated propoxylated)
Vietnam - NCI	Yes
Russia - FBEPH	No (tridecyl alcohol, ethoxylated, phosphated, potassium salt)
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	15/10/2024
Initial Date	26/09/2024

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	26/09/2024	Name
5.1	15/10/2024	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, Toxicological information - Chronic Health, Hazards identification - Classification, Disposal considerations - Disposal, Exposure controls / personal protection - Engineering Control, Ecological Information - Environmental, Exposure controls / personal protection - Exposure Standard, 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 (skin), First Aid measures - First Aid (swallowed), Handling and storage - Handling Procedure, Composition / information on ingredients - Ingredients, 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), Handling and storage - Storage (storage incompatibility), Handling and storage - Storage (storage requirement), Toxicological information - Toxicity and Irritation (Other), 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

Continued...

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

end of SDS