

# Folk Art Metallic Paints

## Jasco Pty Limited

Chemwatch: 7912-20

Version No: 3.1

Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Chemwatch Hazard Alert Code: 4

Issue Date: 08/10/2024

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

#### Product Identifier

Product name	Folk Art Metallic 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.
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#### 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	<a href="http://www.jasco.com.au">www.jasco.com.au</a>
Email	quickinfo@jasco.com.au

#### Emergency telephone number

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

### SECTION 2 Hazards identification

#### Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification <sup>[1]</sup>	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Carcinogenicity Category 1A, Specific Target Organ Toxicity - Repeated Exposure Category 1, 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)	
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Signal word	Danger
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#### Hazard statement(s)

H315	Causes skin irritation.
H318	Causes serious eye damage.
H335	May cause respiratory irritation.
H350	May cause cancer.
H372	Causes damage to organs through prolonged or repeated exposure.
H412	Harmful to aquatic life with long lasting effects.

#### Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P270	Do not eat, drink or smoke when using this product.
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.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
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.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

**Precautionary statement(s) Disposal**

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

See section below for composition of Mixtures

**Mixtures**

CAS No	%[weight]	Name
12001-26-2	35-85	<a href="#">mica</a>
13463-67-7	10-40	<a href="#">C.I. Pigment White 6</a>
1309-37-1	1-30	<a href="#">ferric oxide</a>
7429-90-5	1-10	<a href="#">aluminium</a>
57-55-6	1-10	<a href="#">propylene glycol</a>
1317-65-3	1-10	<a href="#">calcium carbonate</a>
78330-21-9	1-10	<a href="#">alcohols C11-14-iso-, C13-rich, ethoxylated</a>
51274-00-1	<5	<a href="#">C.I. Pigment Yellow 42</a>
1333-86-4	<5	<a href="#">C.I. Pigment Black 7</a>
127087-87-0	<1	<a href="#">4-nonylphenol, branched, ethoxylated</a>
1332-58-7	<1	<a href="#">kaolin</a>
95-38-5	<1	<a href="#">2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol</a>
4724-48-5	<1	<a href="#">octylphosphonic acid</a>
25322-69-4	<1	<a href="#">polypropylene glycol</a>
124-68-5	<1	<a href="#">monoisobutanolamine</a>
18282-10-5	<1	<a href="#">stannic oxide</a>
13463-41-7	<1	<a href="#">zinc pyrithione</a>
111-46-6	<1	<a href="#">diethylene glycol</a>
577-11-7	<1	<a href="#">sodium dioctyl sulfosuccinate</a>
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**

<b>Eye Contact</b>	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> <li>▶ Immediately hold eyelids apart and flush the eye continuously with running water.</li> <li>▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.</li> <li>▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.</li> <li>▶ Transport to hospital or doctor without delay.</li> <li>▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> </ul>
<b>Skin Contact</b>	<p>If skin or hair contact occurs:</p> <ul style="list-style-type: none"> <li>▶ Immediately flush body and clothes with large amounts of water, using safety shower if available.</li> <li>▶ Quickly remove all contaminated clothing, including footwear.</li> </ul>

Continued...

	<ul style="list-style-type: none"> <li>▶ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre.</li> <li>▶ Transport to hospital, or doctor.</li> </ul>
Inhalation	<ul style="list-style-type: none"> <li>▶ If fumes or combustion products are inhaled remove from contaminated area.</li> <li>▶ Lay patient down. Keep warm and rested.</li> <li>▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.</li> <li>▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.</li> <li>▶ Transport to hospital, or doctor, without delay.</li> </ul>
Ingestion	<ul style="list-style-type: none"> <li>▶ <b>If swallowed do NOT induce vomiting.</b></li> <li>▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.</li> <li>▶ Observe the patient carefully.</li> <li>▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.</li> <li>▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.</li> <li>▶ Seek medical advice.</li> </ul>

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

Treat symptomatically.

**SECTION 5 Firefighting measures****Extinguishing media**

Metal dust fires need to be smothered with sand, inert dry powders.

**DO NOT USE WATER, CO2 or FOAM.**

- ▶ Use DRY sand, graphite powder, dry sodium chloride based extinguishers, G-1 or Met L-X to smother fire.
- ▶ Confining or smothering material is preferable to applying water as chemical reaction may produce flammable and explosive hydrogen gas.
- ▶ Chemical reaction with CO2 may produce flammable and explosive methane.
- ▶ If impossible to extinguish, withdraw, protect surroundings and allow fire to burn itself out.
- ▶ **DO NOT** use halogenated fire extinguishing agents.

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

<b>Fire Incompatibility</b>	▶ Reacts with acids producing flammable / explosive hydrogen (H2) gas
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**Advice for firefighters**

<b>Fire Fighting</b>	<ul style="list-style-type: none"> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ Wear breathing apparatus plus protective gloves in the event of a fire.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water courses.</li> <li>▶ Use fire fighting procedures suitable for surrounding area.</li> <li>▶ <b>DO NOT</b> approach containers suspected to be hot.</li> <li>▶ Cool fire exposed containers with water spray from a protected location.</li> <li>▶ If safe to do so, remove containers from path of fire.</li> <li>▶ Equipment should be thoroughly decontaminated after use.</li> </ul>
<b>Fire/Explosion Hazard</b>	<ul style="list-style-type: none"> <li>▶ The material is not readily combustible under normal conditions.</li> <li>▶ However, it will break down under fire conditions and the organic component may burn.</li> <li>▶ Not considered to be a significant fire risk.</li> <li>▶ Heat may cause expansion or decomposition with violent rupture of containers.</li> <li>▶ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO).</li> <li>▶ May emit acrid smoke.</li> </ul> <p>Decomposes on heating and produces toxic fumes of:</p> <ul style="list-style-type: none"> <li>carbon dioxide (CO2)</li> <li>nitrogen oxides (NOx)</li> <li>phosphorus oxides (POx)</li> <li>sulfur oxides (SOx)</li> <li>silicon dioxide (SiO2)</li> <li>metal oxides</li> <li>other pyrolysis products typical of burning organic material.</li> </ul> <p>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.</p> <p>May emit poisonous fumes. May emit corrosive fumes.</p>
<b>HAZCHEM</b>	Not Applicable

**SECTION 6 Accidental release measures****Personal precautions, protective equipment and emergency procedures**

See section 8

**Environmental precautions**

See section 12

**Methods and material for containment and cleaning up**

<b>Minor Spills</b>	<ul style="list-style-type: none"> <li>▶ Clean up all spills immediately.</li> <li>▶ Avoid breathing vapours and contact with skin and eyes.</li> <li>▶ Control personal contact with the substance, by using protective equipment.</li> <li>▶ Contain and absorb spill with sand, earth, inert material or vermiculite.</li> <li>▶ Wipe up.</li> <li>▶ Place in a suitable, labelled container for waste disposal.</li> </ul>
<b>Major Spills</b>	<ul style="list-style-type: none"> <li>▶ Clear area of personnel and move upwind.</li> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ Wear full body protective clothing with breathing apparatus.</li> <li>▶ Prevent, by all means available, spillage from entering drains or water courses.</li> <li>▶ Consider evacuation (or protect in place).</li> </ul>

- ▶ No smoking, naked lights or ignition sources.
- ▶ Increase ventilation.
- ▶ 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

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

### Conditions for safe storage, including any incompatibilities

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

## SECTION 8 Exposure controls / personal protection

### Control parameters

#### Occupational Exposure Limits (OEL)

#### INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	mica	Mica	2.5 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
Australia Exposure Standards	C.I. Pigment White 6	Titanium dioxide	10 mg/m <sup>3</sup>	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	ferric oxide	Rouge dust	10 mg/m <sup>3</sup>	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 (Fe <sub>2</sub> O <sub>3</sub> ) (as Fe)	5 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium	Aluminium (metal dust)	10 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium	Aluminium, pyro powders (as Al)	5 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium	Aluminium (welding fumes) (as Al)	5 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
Australia Exposure Standards	propylene glycol	Propane-1,2-diol total: (vapour & particulates)	150 ppm / 474 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
Australia Exposure Standards	propylene glycol	Propane-1,2-diol: particulates only	10 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
Australia Exposure Standards	calcium carbonate	Calcium carbonate	10 mg/m <sup>3</sup>	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	C.I. Pigment Black 7	Carbon black	3 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
Australia Exposure Standards	kaolin	Kaolin	10 mg/m <sup>3</sup>	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	stannic oxide	Tin oxide & inorganic compounds, except SnH <sub>4</sub> (as Sn)	2 mg/m <sup>3</sup>	Not Available	Not Available	Not Available

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	diethylene glycol	2,2'-Oxybis[ethanol]	23 ppm / 100 mg/m3	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
mica	1,500 mg/m3	Not Available
C.I. Pigment White 6	5,000 mg/m3	Not Available
ferric oxide	2,500 mg/m3	Not Available
aluminium	Not Available	Not Available
propylene glycol	Not Available	Not Available
calcium carbonate	Not Available	Not Available
alcohols C11-14-iso-, C13-rich, ethoxylated	Not Available	Not Available
C.I. Pigment Yellow 42	Not Available	Not Available
C.I. Pigment Black 7	1,750 mg/m3	Not Available
4-nonylphenol, branched, ethoxylated	Not Available	Not Available
kaolin	Not Available	Not Available
2-(8-heptadeceny)-4,5-dihydro-1H-imidazole-1-ethanol	Not Available	Not Available
octylphosphonic acid	Not Available	Not Available
polypropylene glycol	Not Available	Not Available
monoisobutanolamine	Not Available	Not Available
stannic oxide	100 mg/m3	Not Available
zinc pyrithione	Not Available	Not Available
diethylene glycol	Not Available	Not Available
sodium dioctyl sulfosuccinate	Not Available	Not Available


**Occupational Exposure Banding**

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
alcohols C11-14-iso-, C13-rich, ethoxylated	E	≤ 0.1 ppm
C.I. Pigment Yellow 42	E	≤ 0.01 mg/m <sup>3</sup>
4-nonylphenol, branched, ethoxylated	E	≤ 0.1 ppm
2-(8-heptadeceny)-4,5-dihydro-1H-imidazole-1-ethanol	E	≤ 0.1 ppm
octylphosphonic acid	E	≤ 0.01 mg/m <sup>3</sup>
monoisobutanolamine	E	≤ 0.01 mg/m <sup>3</sup>
zinc pyrithione	E	≤ 0.01 mg/m <sup>3</sup>
sodium dioctyl sulfosuccinate	E	≤ 0.01 mg/m <sup>3</sup>

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

**MATERIAL DATA****Exposure controls**

Appropriate engineering controls					
	<p>Metal dusts must be collected at the source of generation as they are potentially explosive.</p> <ul style="list-style-type: none"> <li>▶ Avoid ignition sources.</li> <li>▶ Good housekeeping practices must be maintained.</li> <li>▶ Dust accumulation on the floor, ledges and beams can present a risk of ignition, flame propagation and secondary explosions.</li> <li>▶ Do not use compressed air to remove settled materials from floors, beams or equipment</li> <li>▶ Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation.</li> <li>▶ Use non-sparking handling equipment, tools and natural bristle brushes. Cover and reseal partially empty containers. Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations.</li> <li>▶ Do not allow chips, fines or dusts to contact water, particularly in enclosed areas.</li> <li>▶ Metal spraying and blasting should, where possible, be conducted in separate rooms. This minimises the risk of supplying oxygen, in the form of metal oxides, to potentially reactive finely divided metals such as aluminium, zinc, magnesium or titanium.</li> <li>▶ Work-shops designed for metal spraying should possess smooth walls and a minimum of obstructions, such as ledges, on which dust accumulation is possible.</li> <li>▶ Wet scrubbers are preferable to dry dust collectors.</li> <li>▶ Bag or filter-type collectors should be sited outside the workrooms and be fitted with explosion relief doors.</li> <li>▶ Cyclones should be protected against entry of moisture as reactive metal dusts are capable of spontaneous combustion in humid or partially wetted states.</li> <li>▶ Local exhaust systems must be designed to provide a minimum capture velocity at the fume source, away from the worker, of 0.5 metre/sec.</li> <li>▶ Local ventilation and vacuum systems must be designed to handle explosive dusts. Dry vacuum and electrostatic precipitators must not be used, unless specifically approved for use with flammable/ explosive dusts.</li> </ul> <p>Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p> <table border="1"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>welding, brazing fumes (released at relatively low velocity into moderately still air)</td> <td>0.5-1.0 m/s (100-200 f/min.)</td> </tr> </tbody> </table>	Type of Contaminant:	Air Speed:	welding, brazing fumes (released at relatively low velocity into moderately still air)	0.5-1.0 m/s (100-200 f/min.)
Type of Contaminant:	Air Speed:				
welding, brazing fumes (released at relatively low velocity into moderately still air)	0.5-1.0 m/s (100-200 f/min.)				

	<p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="384 237 1145 389"> <thead> <tr> <th data-bbox="384 237 863 264">Lower end of the range</th> <th data-bbox="863 237 1145 264">Upper end of the range</th> </tr> </thead> <tbody> <tr> <td data-bbox="384 264 863 293">1: Room air currents minimal or favourable to capture</td> <td data-bbox="863 264 1145 293">1: Disturbing room air currents</td> </tr> <tr> <td data-bbox="384 293 863 322">2: Contaminants of low toxicity or of nuisance value only.</td> <td data-bbox="863 293 1145 322">2: Contaminants of high toxicity</td> </tr> <tr> <td data-bbox="384 322 863 351">3: Intermittent, low production.</td> <td data-bbox="863 322 1145 351">3: High production, heavy use</td> </tr> <tr> <td data-bbox="384 351 863 389">4: Large hood or large air mass in motion</td> <td data-bbox="863 351 1145 389">4: Small hood-local control only</td> </tr> </tbody> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	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
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<p><b>Individual protection measures, such as personal protective equipment</b></p>											
<p><b>Eye and face protection</b></p>	<ul style="list-style-type: none"> <li>▶ 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.</li> <li>▶ 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]</li> <li>▶ Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection.</li> <li>▶ Alternatively a gas mask may replace splash goggles and face shields.</li> <li>▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].</li> </ul>										
<p><b>Skin protection</b></p>	<p>See Hand protection below</p>										
<p><b>Hands/feet protection</b></p>	<ul style="list-style-type: none"> <li>▶ Elbow length PVC gloves</li> </ul> <p><b>NOTE:</b></p> <ul style="list-style-type: none"> <li>▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.</li> <li>▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.</li> </ul> <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> <li>· frequency and duration of contact,</li> <li>· chemical resistance of glove material,</li> <li>· glove thickness and</li> <li>· dexterity</li> </ul> <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> <li>· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> <li>· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> <li>· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.</li> <li>· Contaminated gloves should be replaced.</li> </ul> <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> <li>· Excellent when breakthrough time &gt; 480 min</li> <li>· Good when breakthrough time &gt; 20 min</li> <li>· Fair when breakthrough time &lt; 20 min</li> <li>· Poor when glove material degrades</li> </ul> <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> <li>· Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.</li> <li>· Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential</li> </ul> <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <ul style="list-style-type: none"> <li>▶ Butyl rubber gloves</li> <li>· Nitrile rubber gloves (Note: Nitric acid penetrates nitrile gloves in a few minutes.)</li> </ul>										
<p><b>Body protection</b></p>	<p>See Other protection below</p>										
<p><b>Other protection</b></p>	<ul style="list-style-type: none"> <li>▶ Overalls.</li> <li>▶ P.V.C apron.</li> <li>▶ Barrier cream.</li> <li>▶ Skin cleansing cream.</li> <li>▶ Eye wash unit.</li> </ul>										

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

Glove selection is based on a modified presentation of the: **"Forsberg Clothing Performance Index"**.

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

Folk Art Metallic Paints

Material	CPI
BUTYL	C
NATURAL RUBBER	C
NEOPRENE	C
NITRILE	C
PE/EVAL/PE	C
PVA	C
VITON	C

\* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

**Ansell Glove Selection**

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

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

**Respiratory protection**

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

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

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

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

^ - Full-face

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

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

- 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/m <sup>3</sup> )	Not Available	Enclosed Space Ignition Deflagration Density (g/m <sup>3</sup> )	Not Available

## SECTION 10 Stability and reactivity

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

## SECTION 11 Toxicological information

### Information on toxicological effects

Inhaled	<p>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.</p> <p>Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>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</p> <p>Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.</p> <p>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.</p>
Ingestion	<p>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.</p> <p>Accidental ingestion of the material may be damaging to the health of the individual.</p>
Skin Contact	<p>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.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p> <p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p>
Eye	<p>When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.</p>
Chronic	<p>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.</p> <p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>Long term exposure to the dusts of titanium and several of its compounds produces chronic lung disease (fibrosis) in animals. Radiological evidence exists amongst titanium dioxide workers suggesting chronic lung changes which resemble a slight form of silicosis. Workers chronically exposed to titanium or titanium dioxide dusts show a high incidence of chronic bronchitis (endobronchitis and peribronchitis).</p>



Early stages of this disease are characterised by impaired pulmonary respiration and ventilatory capacity and by reduced blood alkalinity. Cardiac changes characteristic of pulmonary disease (with hypertrophy of the right auricle) have also been observed amongst workers. Titanium employed in implants has provoked immune responses which occur locally as metallosis and systemically as raised serum levels of activated T-lymphocytes. Some concern has been expressed about the potential for generating bone-resorbing mediators associated with titanium wear-debris.

The largest of the cohort studies was among white male production workers in the titanium dioxide industry in six European countries. The study indicated a slightly increased risk for lung cancer compared with the general population. However, there was no evidence of an exposure-response relationship within the cohort. No increase in the mortality rates for kidney cancer was found when the cohort was compared with the general population, but there was a suggestion of an exposure-response relationship in internal analyses. The other cohort studies, both of which were conducted in the USA, did not report an increased risk for lung cancer or cancer at any other site; no results for kidney cancer were reported, presumably because there were few cases.

One population-based case-control study conducted in Montreal did not indicate an increased risk for lung or kidney cancer.

In summary, the studies do not suggest an association between occupational exposure to titanium dioxide as it occurred in recent decades in western Europe and North America and risk for cancer.

All the studies had methodological limitations; misclassification of exposure could not be ruled out. None of the studies was designed to assess the impact of particle size (fine or ultrafine) or the potential effect of the coating compounds on the risk for lung cancer.

An increased incidence of lung adenomas in rats of both sexes and of cystic keratinising lesions, diagnosed as squamous cell carcinomas in female rats, was seen in animals subject to high doses of inhaled titanium dioxide. Intratracheal delivery of titanium dioxide in combination with benz[a]pyrene produced an increase in benign and malignant tumours of the larynx, trachea and lungs in hamsters.

Squamous cell carcinomas developed after exposure to 250 mg/m<sup>3</sup> for 6 hours/day, 5 days/week for 2 years in rats; the type of carcinoma that developed was considered to be a unique experimentally induced tumour and to be of questionable relevance for extrapolation of the results to humans. Given the extremely high level of dust in the lungs, the carcinomas were postulated to be the result of saturation of the normal pulmonary clearance mechanisms. At 50 mg/m<sup>3</sup>, massive accumulations of dust-laden macrophages, foamy dust cells and free particles were considered indicative of such overload.

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.

Folk Art Metallic Paints	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
mica	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
C.I. Pigment White 6	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (hamster) LD50: >=10000 mg/kg <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Inhalation (Rat) LC50: >2.28 mg/l4h <sup>[1]</sup>	Skin (rabbit) Draize 0.3mg/3hrInt Mild
ferric oxide	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral (Rat) LD50: >5000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
aluminium	<b>TOXICITY</b>	<b>IRRITATION</b>
	Inhalation (Rat) LC50: >2.3 mg/l4h <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (Rat) LD50: >2000 mg/kg <sup>[1]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
propylene glycol	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 11890 mg/kg <sup>[2]</sup>	Eye (rabbit): 100 mg - mild
	Inhalation (Rat) LC50: >44.9 mg/l4h <sup>[1]</sup>	Eye (rabbit): 500 mg/24h - mild
	Oral (Rat) LD50: 20000 mg/kg <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
		Skin(human):104 mg/3d Intermit Mod
calcium carbonate	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye (rabbit): 0.75 mg/24h - SEVERE
	Inhalation (Rat) LC50: >3 mg/l4h <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (Rat) LD50: >2000 mg/kg <sup>[1]</sup>	Skin (rabbit): 500 mg/24h-moderate
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
alcohols C11-14-iso-, C13-rich, ethoxylated	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral (Rat) LD50: 500 mg/kg <sup>[2]</sup>	Not Available

C.I. Pigment Yellow 42	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral (Rat) LD50: >5000 mg/kg <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
C.I. Pigment Black 7	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >2000 mg/kg <sup>[1]</sup> Oral (Rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
4-nonylphenol, branched, ethoxylated	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral (Rat) LD50: 1310 mg/kg <sup>[2]</sup>	Eye (rabbit): SEVERE Eye: adverse effect observed (irritating) <sup>[1]</sup> Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin (rabbit): Mild Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
kaolin	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
2-(8-heptadeceny)-4,5-dihydro-1H-imidazole-1-ethanol	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral (Rat) LD50: 870 mg/kg <sup>[2]</sup>	Eye (rabbit): Severe ** [Manufacturer] Eye: adverse effect observed (irreversible damage) <sup>[1]</sup> Skin (rabbit): Severe * Skin: adverse effect observed (corrosive) <sup>[1]</sup>
octylphosphonic acid	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup> Oral (Rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup> Skin: adverse effect observed (corrosive) <sup>[1]</sup>
polypropylene glycol	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 500 mg/kg <sup>[2]</sup> Inhalation (Rat) LC50: >2.34 mg/L4h <sup>[1]</sup> Oral (Rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin (rabbit): 500 mg mild Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
monoisobutanolamine	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >2000 mg/kg <sup>[1]</sup> Oral (Mouse) LD50: 2150 mg/kg <sup>[2]</sup>	Eye: adverse effect observed (irreversible damage) <sup>[1]</sup> Skin: adverse effect observed (irritating) <sup>[1]</sup>
stannic oxide	<b>TOXICITY</b>	<b>IRRITATION</b>
	Inhalation (Rat) LC50: >2.04 mg/L4h <sup>[1]</sup> Oral (Rat) LD50: >20000 mg/kg <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
zinc pyrithione	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 100 mg/kg <sup>[2]</sup> Inhalation (Rat) LC50: 0.14 mg/L4h <sup>[2]</sup> Oral (Mouse) LD50: 160 mg/kg <sup>[2]</sup>	Eye (rabbit): 1 mg/48h Irritant Eye: adverse effect observed (irritating) <sup>[1]</sup> Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
diethylene glycol	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 11890 mg/kg <sup>[2]</sup> Inhalation (Rat) LC50: >4.6 mg/L4h <sup>[1]</sup> Oral (Rat) LD50: 12565 mg/kg <sup>[2]</sup>	Eye (rabbit) 50 mg mild Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin (human): 112 mg/3d-I mild Skin (rabbit): 500 mg mild Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
sodium dioctyl sulfosuccinate	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 2525 mg/kg <sup>[1]</sup> Oral (Rat) LD50: >1320 mg/kg <sup>[1]</sup>	Eye (rabbit): 0.250 mg - mild Eye (rabbit): 1% - SEVERE Eye: adverse effect observed (irritating) <sup>[1]</sup> Skin (rabbit): 10 mg/24h-moderate Skin: adverse effect observed (irritating) <sup>[1]</sup>

**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.

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 estrogen 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.
<b>CALCIUM CARBONATE</b>	No evidence of carcinogenic properties. No evidence of mutagenic or teratogenic effects.
<b>ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED</b>	* Ashland SDS
<b>4-NONYLPHENOL, BRANCHED, ETHOXYLATED</b>	<p>for linear material: Maternal effects, effects on fertility recorded.</p> <p>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 endogenous hormone for binding with the estrogen receptors ERalpha and ERbeta.</p> <p>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.</p> <p>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.</p> <p>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 to mimic the action of leptin on neuropeptide Y and anorectic POMC neurons, which has an anti-obesity effect by decreasing eating behavior. This was seen when estrogen or estrogen mimics were injected into the ventromedial hypothalamus. On the other hand, nonylphenol has been shown to increase food intake and have obesity enhancing properties by lowering the expression of these anorexigenic neurons in the brain. Additionally, nonylphenol affects the expression of ghrelin: an enzyme produced by the stomach that stimulates appetite. Ghrelin expression is positively regulated by estrogen signaling in the stomach, and it is also important in guiding the differentiation of stem cells into adipocytes (fat cells). Thus, acting as an estrogen mimic, prenatal and perinatal exposure to nonylphenol has been shown to increase appetite and encourage the body to store fat later in life. Finally, long-term exposure to nonylphenol has been shown to affect insulin signaling in the liver of adult male rats.</p> <p>Cancer 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.</p> <p>Nonylphenol was not mutagenic to Salmonella typhimurium, TA100, TA1535, TA98, TA1537 and Escherichia coli WP2 uvrA, with or without an exogenous metabolic activation system.</p> <p>Nonylphenol induced neither structural chromosomal aberrations nor polyploidy in CHL/IU cells, in the absence or presence of an exogenous metabolic activation system.</p>
<b>KAOLIN</b>	<p>for bentonite clays: 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&gt;15 g/kg). However, severe anterior segment inflammation, uveitis and retrocorneal abscess from eye exposure were reported when bentonite had been used as a prophylactic.</p> <p>In a 33 day dietary (2 and 6%) and a 90 day dietary (1, 3 and 5%) studies in chickens, 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.</p> <p>Bentonite did not cause fibrosis after 1 year exposure of 60 mg dust (&lt;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.</p> <p>Ingestion of bentonite without adequate liquids may result in intestinal obstruction in humans.</p> <p>Hypokalaemia and microcytic iron-deficiency anaemia may occur in patients after repeat doses of clay. Chronic ingestion has been reported to cause myositis.</p>
<b>2-(8-HEPTADECENYL)-4,5-DIHYDRO-1H-IMIDAZOLE-1-ETHANOL</b>	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p> <p>For imidazoline surfactants (amidoamine/ imidazoline - AAI) All substances within the AAI group show the same reactive groups, show similar composition of amide, imidazoline, and some dimer structures of both, with the length of original EA amines used for production as biggest difference. Inherent reactivity and toxicity is not expected to differ much between these substances.</p> <p>All in vivo skin irritation/corrosion studies performed on AAI substances all indicate them to be corrosive following 4 hour exposure. There do not seem to be big differences in response with the variation on EA length used for the production of the AAI.</p> <p>The available data available for AAI substances indicate that for AAI based on shorter polyethylenamines (EA), higher toxicity is observed compared to AAI based on longer EA. The forming of imidazoline itself does not seem to play a significant role. For cross-reading in general Fatty acid reaction product with diethylenetriamine (AAI-DETA) therefore represents the worst case. In series of 28-day and combined repeated dose/reproduction screening toxicity studies (OECD 422) AAI-DETA has shown the highest level of toxicity</p>

Acute oral exposure of tall oil + triethylenepentamine (TEPA) show limited acute toxicity, with a LD50 above 2000 mg/kg bw. Hence no classification is required.

Acute dermal testing with corrosive materials is not justified. As a consequence no classification can be made for acute dermal toxicity.

Effects will be characterised by local tissue damage. Systemic uptake via skin is likely to be very limited. The low acute oral toxicity indicate a low systemic toxicity.

For dermal exposure no good overall NOAEL can be established as effects are rather characterized by local corrosive effects that are related to duration, quantity and concentration, than by systemic toxicity due to dermal uptake. The mode of action for AAI follows from its structure, consisting of an apolar fatty acid chain and a polar end of a primary amine from the polyethyleneamine. The structure can disrupt the cytoplasmic membrane, leading to lyses of the cell content and consequently the death of the cell.

The AAI are protonated under environmental conditions which causes them to strongly adsorb to organic matter. This leads to a low dermal absorption.

No classification for acute dermal toxicity is therefore indicated.

Also for acute inhalation toxicity information for classification is lacking, and is testing not justified. Due to very low vapour pressure is the likelihood of exposure low.

AAI do not contain containing aliphatic, alicyclic and aromatic hydrocarbons and have a relatively high viscosity and so do not indicate an immediate concern for aspiration hazard.

Various studies with different AAI indicate that these substances can cause dermal sensitisation.

All substances within the AAI group show the same reactive groups, show similar composition of amide, imidazoline, and some dimer structures of both, with the length of original EA amines used for production as biggest difference. Inherent reactivity and toxicity is not expected to differ much between these substances, aspects which determine sensitization.

The actual risk of sensitisation is probably low, as AAI are corrosive to skin and consequently exposure will be low due to necessary protective measures to limit dermal exposure.

The likelihood for exposure via inhalation and thus experience respiratory irritation or becoming sensitised to AAI, is very low considering the high boiling point (> 300 deg C) and very low vapour pressure (0.00017 mPa at 25 deg C for diethylenetriamine ( DETA) based AAI). In case of high exposure by inhalation, local effects will be more prominent than possible systemic effects considering the low systemic toxicity seen in acute oral toxicity testing

However, some calculations can be made for systemic effects following short-term inhalation exposure by extrapolating information from an OECD 422 study on "tall oil reaction products with tetraethylenepentamine showing a NOAEL of 300 mg/kg/day. This would certainly be protective for levels of acute inhalation expected to lead to similar systemic exposure levels.

The corrected 8 hr inhalation NOAEC for workers is NOAEL (300 mg/kg) \* 1.76 mg/m<sup>3</sup> = 529 mg/m<sup>3</sup> (assuming no difference in absorption following oral and inhalation exposure). Assessment factors further applied: No interspecies factor is needed due to allometric scaling applied in calculation of corrected NOAEC. Further combined inter-/intra-species for workers AF = 3 (ECETOC concept). As this involves acute exposures, no extrapolation for duration is needed.

This results in a DNEL of 529/3 = 176 mg/m<sup>3</sup>. A short term/acute exposure at this level can be assumed not to lead to systemic toxicity.

Repeat dose toxicity:

A combined repeated dose/reproduction screening toxicity study according to OECD 422 with Fatty acid reaction products with tetraethylene-pentamine resulted to a NOAEL of 300 mg/kg bw/day, the highest dose tested. Also available data from the group of Amidoamine/Imidazoline (AAI) substances, including 90-day studies in rat and dogs on a similar substance, indicate very low toxicity. Consequently, serious toxicity is not observed at levels requiring consideration classification for STOTS-RE

Genotoxicity:

Tall oil, reaction products with tetraethylenepentamine is not mutagenic in the Salmonella typhimurium reverse mutation assay (based on test with Fatty acids C16-18, C18 unsaturated reaction products with tetraethylenepentamine), is not clastogenic in human lymphocytes, and not mutagenic in the TK mutation test with L5178Y mouse lymphoma cells.

It can therefore be concluded that tall oil, reaction products with tetraethylenepentamine not genotoxic.

Toxicity to reproduction:

The database of relevant studies available for the group of amidoamine/ imidazolines (AAI) include various OECD 422 studies and an OECD 414 study, that all show no concerns regarding reproduction or developmental toxicity. Also all already available data from the group of AAI substances, including a 90-day study in dogs on a similar substance, indicate low toxicity and no adverse effects on reproductive organs.

REACH Dossier

The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

For amphoteric imidazoline derivatives:

Generally these amphoteric surfactants do not seem to be irritant to the skin and only to a small extent irritating to the eye . Some variation in test results have been reported.

Cocoamphodipropionate was found to be non-irritating as a concentration of 7.5-70% , whereas cocoamphopropionate was slightly irritating to rabbit skin at a concentration of 15-16%. Cocoamphodiacetate was non-irritating to slightly irritating at a concentration of 10-12% .

A Draize test has shown that cocoamphodipropionate was practically non-irritating to the eye at a concentration of 7.5%, whereas cocoamphopropionate was non-irritating to slightly irritating at 5% and 16%. Cocoamphodiacetate was moderately to severely irritating to the eye at a concentration of 10-12%. Cocoamphoacetate was slightly to severely irritating at 16 to 50% .

**Sensitisation:** Cocoamphoacetate and cocoamphopropionate were non-irritating and non-sensitising in a repeated insult patch test (non-occlusive) involving 141 subjects. The concentration of the surfactants was 10% in distilled water. During induction, each chemical was applied to the back three times per week for three weeks. The challenge phase was initiated 10 to 15 days after application of the final induction patch. Cocoamphoacetate and cocoamphopropionate did not induce sensitization in any of the subjects . Cocoamphoacetate was non-sensitising in guinea pigs when tested in the Magnusson-Kligman maximization test. The tested concentrations for induction and challenge were 25, 50 and 100%

**Mutagenicity:** Cocoamphodiacetate, cocoamphopropionate, and cocoamphodipropionate were non-mutagenic, when evaluated in the Ames Salmonella/microsome assay using different strains of *Salmonella typhimurium* .

No tests on reproductive toxicity and carcinogenicity were available.

**Classification** The amphoteric surfactants described in this category are not included in Annex 1 of list of dangerous substances of Council Directive 67/548/EEC.

#### POLYPROPYLENE GLYCOL

\*\* Rohm and Haas Paraplex WP-1 MSDS

The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

#### MONOISOBUTANOLAMINE

For tris(hydroxymethyl)aminomethane (TRIS AMINO; CAS 77-88-1) and its surrogates 2-amino-2-methyl-1,3-propanediol (AMPD; CAS 115-69-5) and monoisobutanolamine (AMP; CAS 124-68-5)

TRIS AMINO and the surrogate chemicals have displayed little if any toxicity to humans during their long history of use as human drugs and/or in personal care products and cosmetics. TRIS AMINO has found use as an IV drug for the management of acidosis in humans for many years and the toxicity of AMPD and AMP have been reviewed by the Cosmetic Ingredient Review Expert Panel which concluded that these materials are safe as used in cosmetic formulations up to 1%

**Acute toxicity:** Mammalian toxicity studies have displayed similar results. The oral LD50 value for TRIS AMINO is 5500 mg/kg in the mouse, and its surrogates range from 2150 to greater than 5000 mg/kg in the rat and mouse. TRIS AMINO was non-irritating to eyes when a 40% aqueous solution was applied to the eyes of rabbits (pH 10.4 for 0.1M aqueous solution). In contrast, 95% AMP in water was severely irritating to the eyes, presumably due to the severely alkaline pH of the test solution used (pH 11.3 for 0.1M aqueous solution); however, more neutral cosmetic formulations containing lower concentrations of AMP are only minimally irritating. There is no sensitisation data available for TRIS AMINO; however, based on the following data, TRIS AMINO is not expected to be a sensitizer. Laboratory animal test samples of AMP did not cause allergic skin reactions when tested in guinea pigs following topical or intradermal administration. In patch tests with humans, AMP and cosmetic formulations containing either AMP or AMPD were negative for dermal sensitisation.

**Repeated dose toxicity:** Repeated-dose mammalian toxicity studies conducted on TRIS AMINO and the two surrogate chemicals indicate that the compounds are generally well-tolerated at concentrations as high as 500 mg/kg/day via IV infusion for TRIS AMINO and ingestion of up to 3200 ppm in the rodent diet (250-750 mg/kg/day for rats and mice, estimated). A number of human clinical trials of the IV infusion of TRIS AMINO have also been successfully conducted. In all studies, the only target tissue, when observed at all, has been the liver with AMP.

Continued...

	<p>Human clinical studies with Keterolac(a major component of which is TRIS AMINO) have suggested that patients with decreased liver function not be given the drug over extended treatment periods based upon changes in several clinical chemistry parameters. Ingestion of relatively high dosages of AMP has caused liver histopathological changes in rats and dogs. The most significant toxicological activity has been a foetotoxic effect of AMP when ingested at relatively high levels by pregnant rats. Subsequent dermal exposure to comparable dosages failed to elicit a developmental effect in rats. Overall, there have been no consistently-noted observations or treatment-related findings among the numerous repeated-dose mammalian toxicity studies that have been conducted over at last 50 years on these compounds that would indicate long-term significant toxicity of either compound at typical human exposure levels. Reflective of these findings is the fact that both TRIS AMINO and AMP display similar patterns of excretion from the body, being primarily eliminated unchanged via the urine over a relatively short period of time. Further, no evidence of either direct reactivity or metabolism to reactive species toward genetic material has been observed. <b>Genetic toxicity:</b> Studies conducted on the TRIS AMINO and the surrogate substances in the presence or absence of mammalian metabolic enzymes have all been negative.</p>
<p style="text-align: center;"><b>ZINC PYRITHIONE</b></p>	<p>NOAEL: 11.0 mg/kg/day cynomolgus monkey * [* = Arch Chemical] Acute pulmonary oedema, dyspnea, weight loss or decreased weight gain, recordings from specific areas of the CNS, mydriasis, somnolence, changes in motor activity, recording from peripheral motor nerve, muscle weakness, spastic paralysis, reproductive system tumours, retinal changes, diarrhoea, foetotoxicity, specific developmental abnormalities (musculoskeletal system, central nervous system, effects on newborn, foetolethality recorded).</p> <p>for pyrithiones:</p> <p><b>Short-term studies:</b> Zinc pyrithione was orally administered to cynomolgus monkeys daily for 14 or 28 days. In the 14-day study, treatment at 10, 20, 40 or 80 mg/kg bw/day resulted in haemorrhaging of the stomach mucosa and bodyweight loss at the highest tested dose. In the 28-day study, treatment at 0, 5.5, 11 or 22 mg/kg bw, caused a death at the highest dose. Food consumption and bodyweight gain was decreased at the highest dose together with reduced haematocrit, haemoglobin concentration and erythrocyte count. An increased concentration of ketone bodies and decreased pH of the urine was also observed. These changes were either absent or had improved after a 14-day recovery period.</p> <p>In a 90-day study, rats were fed zinc pyrithione in the diet at concentrations of 0, 5, 25 or 125 ppm. Clinical signs first observed during the second week at 125 ppm were a depressed respiratory rate and the onset of progressively restricted movement of the hind limbs which finally resulted in almost complete paralysis. Other changes at 125 ppm were related to severe weight loss and dehydration, resulting from the paralysis. Based on the deaths of nearly all the rats at 125 ppm (from dehydration and/or starvation) and the reduced bodyweight observed at 25 ppm in females, the NOEL for this study was 5 ppm (0.35 mg/kg bw/day for males and 0.39 mg/kg bw/day for females). Daily dermal application of zinc pyrithione to rats at 0, 20, 100 or 1000 mg/kg bw/day for 90 days revealed slight skin irritation, bodyweight loss and reduced food intake at 1000 mg/kg bw/day. For females at 1000 mg/kg bw/day there was an increase in relative kidney weight and some had mineralisation of the kidneys. Increased leucocyte counts and reduced erythrocyte and haematocrit was also observed at the highest dose. Dermal absorption studies in pigs showed that zinc pyrithione is very poorly absorbed through skin (&lt;10% of dose). A maximum of 5% of the applied dose was recovered in the urine and by 48 h the levels in blood, faeces, and urine were essentially at background levels.</p> <p>Whole-body exposure to an aerosol at 0, 0.5, 2.5 or 10 mg/m<sup>3</sup> for 6 h/day, 5 days/week over 13 weeks resulted in deaths at 2.5 and 10 mg/m<sup>3</sup>, reduced bodyweight gain at 10 mg/m<sup>3</sup> and reduced creatinine at 10 mg/m<sup>3</sup>. A dose-related increase in mean absolute lung/mainstream bronchi weight, lung/mainstream bronchi weight relative to body weight and lung/mainstream bronchi weight relative to brain weight was also observed at 2.5 and 10 mg/m<sup>3</sup>. These weight increases were accompanied by inflammation of interstitial tissue and pulmonary artery hypertrophy.</p> <p>Zinc pyrithione given to monkeys at 0, 0.5, 2 or 8 mg/kg bw/day by stomach tube for 90 days induced some vomiting at 2 and 8 mg/kg bw/day within 1-3 h on the first few treatment days. Appropriate monitoring for adverse changes failed to reveal any other effects. Hence, the NOEL for the study was 8 mg/kg bw/day.</p> <p><b>Long-Term Study:</b> Sodium pyrithione at 0, 0.5, 1.5 or 5 mg/kg bw/day was administered to rats by gavage in a two-year chronic and oncogenicity study. After 12 weeks at 5 mg/kg bw/day, an appreciable reduction in bodyweight gain necessitated the high dose level be reduced to 3.5 mg/kg bw/day. There was reduced bodyweight gain at 3.5 mg/kg bw/day and hind limb muscle wastage at 1.5 and 3.5 mg/kg bw/day. Nerve fibre degeneration of the spinal cord and sciatic nerve was slightly increased at 3.5 mg/kg bw/day. Fibre degeneration in the hind limb skeletal muscle was increased in all rats at 3.5 mg/kg bw/day and to a lesser extent in females at 1.5 mg/kg bw/day. There was an increase in peripheral retinal atrophy in males and females at 3.5 mg/kg bw/day and at 1.5 mg/kg bw/day in females. There was no treatment-related increase in the incidence of tumours. Therefore, under the conditions of this study, the NOEL was 0.5 mg/kg bw/day.</p> <p><b>Reproduction and Developmental Studies:</b> In a 2-generation reproduction study, rats were given sodium pyrithione at 0, 0.5, 1.5 or 4.5 mg/kg bw/day by gavage. Owing to an appreciable reduction in bodyweight gain the highest dose was reduced after 3 weeks to 3.5 mg/kg bw/day for the rest of the study. Rats were maintained for 2 generations, with the first litter used for breeding. In the F0 rats, salivation after dosing was seen in all treated groups, with a dose-related time of onset and severity. At 3.5 mg/kg bw/day a number of females showed hind-limb paralysis in the F0 generation; this was not seen in F1 animals. Body weight gain was statistically significantly decreased in both males and females at 3.5 mg/kg bw/day in the F0 generation, and in females at this dose in the F1 generation. Fertility was decreased at 3.5 mg/kg bw/day in the F0 generation, with the number of rats successfully mating and the number of rats pregnant decreased in comparison to controls. There was no effect on gestational length, the number of pups born or pup bodyweight seen. No effects on fertility were seen in the F1 generation. There was no increase in the incidence of foetal malformations in either generation. On postmortem examination, there was an increase in the incidence of hind-limb muscle atrophy at 3.5 mg/kg bw/day in females in both generations. On histopathological examination, there was an increase in atrophy of skeletal muscles at 3.5 mg/kg bw/day in the F0 generation, and from 1.5 mg/kg bw/day in the F1 generation. Salivation occurred in some F0 rats at 0.5 mg/kg bw/day but none in the F1 generation suggesting that this dose level is a probable NOEL.</p> <p>When pregnant rats had zinc pyrithione topically applied at 0, 2.5, 7.5 or 15 mg/kg bw/day (with or without prevention from ingestion) from gestation days 6 to 15 there was a reduction in bodyweight gain at 7.5 or 15 mg/kg bw/day when ingestion was not prevented. Hind-limb paralysis among dams and reductions in fetal weight were also observed at 15 mg/kg bw/day.</p> <p>These effects were not seen when ingestion was prevented. With oral treatment at the same doses, bodyweight gain was reduced, paralysis occurred and fetal weight was reduced at 7.5 and 15 mg/kg bw/day. There was also an increase in skeletal variations at 15 mg/kg bw/day.</p> <p><b>Genotoxicity:</b> Zinc pyrithione was found to be negative in mutation tests in bacteria and Chinese hamster ovary cells. Similarly, no chromosomal aberration was observed in human lymphocytes incubated <i>in vitro</i> in the presence of zinc pyrithione or in lymphocytes harvested from monkeys following oral administration in a 28-day toxicity study. A mouse micronucleus assay also yielded negative results.</p> <p><b>Human metabolite study</b></p> <p>A study of plasma metabolites in human volunteers from a chemical factory producing pyrithiones identified 2-(methylsulfonyl)pyridine as the only metabolite in human serum and proposed that this metabolite could be used as a marker for pyrithione exposure.</p> <p>Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).</p>
<p style="text-align: center;"><b>DIETHYLENE GLYCOL</b></p>	<p>Diglycolic acid is formed following the oxidation of accidentally ingested diethylene glycol in the body and can lead to severe complications with fatal outcome.</p>
<p style="text-align: center;"><b>SODIUM DIOCTYL SULFOSUCCINATE</b></p>	<p>Structural changes in blood vessels recorded.</p> <p>for dialkyl sodium sulfosuccinates:</p> <p>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.</p> <p>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%.</p> <p>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.</p>

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 alpha-olefin sulfonates) demonstrated a similar toxicological and toxicokinetic/metabolic profile as for the sulfosuccinate 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.

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 sensitizers 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 an 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<sub>3</sub><sup>-</sup>) 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

MICA & FERRIC OXIDE & CALCIUM CARBONATE & ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED & C.I. PIGMENT YELLOW 42 & 2-(8-HEPTADECENYL)-4,5-DIHYDRO-1H-IMIDAZOLE-1-ETHANOL & OCTYLPHOSPHONIC ACID

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.

MICA & ALUMINIUM & C.I. PIGMENT YELLOW 42 & C.I. PIGMENT BLACK 7 & KAOLIN & 2-(8-HEPTADECENYL)-4,5-DIHYDRO-1H-IMIDAZOLE-1-ETHANOL & OCTYLPHOSPHONIC ACID

No significant acute toxicological data identified in literature search.

C.I. PIGMENT WHITE 6 & C.I. PIGMENT YELLOW 42

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 & ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED & 2-(8-HEPTADECENYL)-4,5-DIHYDRO-1H-IMIDAZOLE-1-ETHANOL & POLYPROPYLENE GLYCOL & SODIUM DIOCTYL SULFOSUCCINATE

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

CALCIUM CARBONATE & ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED & 4-NONYLPHENOL, BRANCHED, ETHOXYLATED & SODIUM DIOCTYL SULFOSUCCINATE

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

ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED & 4-NONYLPHENOL, BRANCHED, ETHOXYLATED & POLYPROPYLENE GLYCOL

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture.

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



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

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

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

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

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

Safety Evaluation of Polyethylene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of Toxicology

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**ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED & 4-NONYLPHENOL, BRANCHED, ETHOXYLATED**

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

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

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

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

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

Overall, alcohol alkoxyates (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  $C \geq 6$  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<sub>2</sub>). Orally dosed AE was absorbed rapidly and extensively in rats, and more than 75% of the dose was absorbed. When applied to the skin of humans, the doses were absorbed slowly and incompletely (50% absorbed in 72 hours). Half of the absorbed surfactant was excreted promptly in the urine and smaller amounts of AE appeared in the faeces and expired air (CO<sub>2</sub>). The metabolism of C12 AE yields PEG, carboxylic acids, and CO<sub>2</sub> as metabolites. The LD<sub>50</sub> values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.

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

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

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

For high boiling ethylene glycol ethers (typically triethylene- and tetraethylene glycol ethers):

**Skin absorption:** Available skin absorption data for triethylene glycol ether (TGBE), triethylene glycol methyl ether (TGME), and triethylene glycol ethylene ether (TGEE) suggest that the rate of absorption in skin of these three glycol ethers is 22 to 34 micrograms/cm<sup>2</sup>/hr, with the methyl ether having the highest permeation constant and the butyl ether having the lowest. The rates of absorption of TGBE, TGEE and TGME are at least 100-fold less than EGME, EGEE, and EGBE, their ethylene glycol monoalkyl ether counterparts, which have absorption rates that range from 214 to 2890 micrograms/cm<sup>2</sup>/hr. Therefore, an increase in either the chain length of the alkyl substituent or the number of ethylene glycol moieties appears to lead to a decreased rate of percutaneous absorption. However, since the ratio of the change in values of the ethylene glycol to the diethylene glycol series is larger than that

of the diethylene glycol to triethylene glycol series, the effect of the length of the chain and number of ethylene glycol moieties on absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetraethylene glycol methyl ether (TetraME) and tetraethylene glycol butyl ether (TetraBE) are expected to be less permeable to skin than TGME and TGBE, the differences in permeation between these molecules may only be slight.

**Metabolism:** The main metabolic pathway for metabolism of ethylene glycol monoalkyl ethers (EGME, EGEE, and EGBE) is oxidation via alcohol and aldehyde dehydrogenases (ALD/ADH) that leads to the formation of an alkoxy acids. Alkoxy acids are the only toxicologically significant metabolites of glycol ethers that have been detected *in vivo*. The principal metabolite of TGME is believed to be 2-[2-(2-methoxyethoxy)ethoxy] acetic acid. Although ethylene glycol, a known kidney toxicant, has been identified as an impurity or a minor metabolite of glycol ethers in animal studies it does not appear to contribute to the toxicity of glycol ethers.

The metabolites of category members are not likely to be metabolized to any large extent to toxic molecules such as ethylene glycol or the mono alkoxy acids because metabolic breakdown of the ether linkages also has to occur

**Acute toxicity:** Category members generally display low acute toxicity by the oral, inhalation and dermal routes of exposure. Signs of toxicity in animals receiving lethal oral doses of TGBE included loss of righting reflex and flaccid muscle tone, coma, and heavy breathing. Animals administered lethal oral doses of TGEE exhibited lethargy, ataxia, blood in the urogenital area and piloerection before death.

**Irritation:** The data indicate that the glycol ethers may cause mild to moderate skin irritation. TGEE and TGBE are highly irritating to the eyes. Other category members show low eye irritation.

**Repeat dose toxicity:** Results of these studies suggest that repeated exposure to moderate to high doses of the glycol ethers in this category is required to produce systemic toxicity

In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME.

Testicular effects included spermatid giant cells, focal tubular hypospermatogenesis, and increased cytoplasmic vacuolisation. Due to a high incidence of similar spontaneous changes

in normal New Zealand White rabbits, the testicular effects were considered not to be related to treatment. Thus, the NOAELs for TGME, TGEE and TGBE were established at 1000 mg/kg/day. Findings from this report were considered unremarkable.

A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day. In this study, significantly-increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500 mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or

haemolysed blood in the stomach. These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats

In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity

**Mutagenicity:** Mutagenicity studies have been conducted for several category members. All *in vitro* and *in vivo* studies were negative at concentrations up to 5,000 micrograms/plate and 5,000 mg/kg, respectively, indicating that the category members are not genotoxic at the concentrations used in these studies. The uniformly negative outcomes of various mutagenicity studies performed on category members lessen the concern for carcinogenicity.

**Reproductive toxicity:** Although mating studies with either the category members or surrogates have not been performed, several of the repeated dose toxicity tests with the surrogates have included examination of reproductive organs. A lower molecular weight glycol ether, ethylene glycol methyl ether (EGME), has been shown to be a testicular toxicant. In addition, results of repeated dose toxicity tests with TGME clearly show testicular toxicity at an oral dose of 4,000 mg/kg/day four times greater than the limit dose of 1,000 mg/kg/day recommended for repeat dose studies. It should be noted that TGME is 350 times less potent for testicular effects than EGME. 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.

The material may produce respiratory tract irritation. Symptoms of pulmonary irritation may include coughing, wheezing, laryngitis, shortness of breath, headache, nausea, and a burning sensation.

Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence).

The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties.

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

ALCOHOLS C11-14-ISO-, C13-RICH, ETHOXYLATED & 2-(8-HEPTADECENYL)-4,5-DIHYDRO-1H-IMIDAZOLE-1-ETHANOL			
4-NONYLPHENOL, BRANCHED, ETHOXYLATED & DIETHYLENE GLYCOL			
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

## Toxicity

Folk Art Metallic Paints	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
mica	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
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
	aluminium	Endpoint	Test Duration (hr)	Species	Value
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
EC50		96h	Algae or other aquatic plants	0.005mg/L	2
NOEC(ECx)		72h	Algae or other aquatic plants	>100mg/l	1
propylene glycol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	19300mg/l	2
	EC50	48h	Crustacea	>114.4mg/L	4
	LC50	96h	Fish	710mg/L	4
	NOEC(ECx)	336h	Algae or other aquatic plants	<5300mg/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
alcohols C11-14-iso-, C13-rich, ethoxylated	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	1-10mg/l	Not Available
	NOEC(ECx)	1h	Fish	4-320mg/l	4
C.I. Pigment Yellow 42	Endpoint	Test Duration (hr)	Species	Value	Source
	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
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
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
4-nonylphenol, branched, ethoxylated	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	>10mg/l	2
	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	

Continued...

	EC50	96h	Algae or other aquatic plants	12mg/l	2
kaolin	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	Not Available	Not Available	Not Available	Not Available	Not Available
2-(8-heptadeceny)-4,5-dihydro-1H-imidazole-1-ethanol	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	72h	Algae or other aquatic plants	0.017mg/l	2
	EC50	48h	Crustacea	0.163mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	0.011mg/l	2
octylphosphonic acid	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	72h	Algae or other aquatic plants	23mg/l	2
	EC50	48h	Crustacea	3.2mg/l	2
	LC50	96h	Fish	>40mg/l	2
polypropylene glycol	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	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
monoisobutanolamine	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	72h	Algae or other aquatic plants	>103mg/l	2
	EC50	48h	Crustacea	193mg/l	1
	EC0(ECx)	48h	Crustacea	100mg/l	1
	LC50	96h	Fish	100mg/l	1
stannic oxide	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	EC50	48h	Crustacea	>51.1mg/l	4
	LC50	96h	Fish	>5.08mg/l	4
	NOEC(ECx)	96h	Algae or other aquatic plants	1mg/l	4
zinc pyrithione	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	BCF	1440h	Fish	52-180	7
	EC50	72h	Algae or other aquatic plants	0.001mg/L	4
	EC50	48h	Crustacea	0.002-2.14mg/L	4
	LC50	96h	Fish	0.003mg/L	2
	NOEC(ECx)	96h	Algae or other aquatic plants	<0.001mg/L	2
diethylene glycol	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	72h	Algae or other aquatic plants	>6500<13000mg/l	2
	NOEC(ECx)	192h	Algae or other aquatic plants	800mg/l	1
	EC50	48h	Crustacea	>100mg/l	2
	LC50	96h	Fish	>100mg/l	4
sodium dioctyl sulfosuccinate	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	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
<b>Legend:</b>	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data				

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

Continued...

**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
2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol	LOW	LOW
octylphosphonic acid	HIGH	HIGH
polypropylene glycol	LOW	LOW
monoisobutanolamine	LOW	LOW
diethylene glycol	LOW	LOW

#### Bioaccumulative potential

Ingredient	Bioaccumulation
C.I. Pigment White 6	LOW (BCF = 10)
propylene glycol	LOW (BCF = 1)
2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol	LOW (LogKOW = 7.5137)
octylphosphonic acid	LOW (LogKOW = 2.7401)
polypropylene glycol	LOW (LogKOW = 1.6984)
monoisobutanolamine	LOW (BCF = 330)
zinc pyrrithione	LOW (BCF = 240)
diethylene glycol	LOW (BCF = 180)
sodium dioctyl sulfosuccinate	LOW (BCF = 3.78)

#### Mobility in soil

Ingredient	Mobility
C.I. Pigment White 6	LOW (Log KOC = 23.74)
propylene glycol	HIGH (Log KOC = 1)
2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol	LOW (Log KOC = 206300)
octylphosphonic acid	LOW (Log KOC = 76.24)
polypropylene glycol	LOW (Log KOC = 15.66)
monoisobutanolamine	MEDIUM (Log KOC = 2.196)
diethylene glycol	HIGH (Log KOC = 1)

### SECTION 13 Disposal considerations

#### Waste treatment methods

<b>Product / Packaging disposal</b>	<ul style="list-style-type: none"> <li>▶ <b>DO NOT</b> allow wash water from cleaning or process equipment to enter drains.</li> <li>▶ It may be necessary to collect all wash water for treatment before disposal.</li> <li>▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.</li> <li>▶ Where in doubt contact the responsible authority.</li> <li>▶ Recycle wherever possible.</li> <li>▶ 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.</li> <li>▶ 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).</li> <li>▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.</li> </ul>
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### SECTION 14 Transport information

#### Labels Required

<b>Marine Pollutant</b>	NO
<b>HAZCHEM</b>	Not Applicable

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

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

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

#### 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
mica	Not Available

Product name	Group
C.I. Pigment White 6	Not Available
ferric oxide	Not Available
aluminium	Not Available
propylene glycol	Not Available
calcium carbonate	Not Available
alcohols C11-14-iso-, C13-rich, ethoxylated	Not Available
C.I. Pigment Yellow 42	Not Available
C.I. Pigment Black 7	Not Available
4-nonylphenol, branched, ethoxylated	Not Available
kaolin	Not Available
2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol	Not Available
octylphosphonic acid	Not Available
polypropylene glycol	Not Available
monoisobutanolamine	Not Available
stannic oxide	Not Available
zinc pyrithione	Not Available
diethylene glycol	Not Available
sodium dioctyl sulfosuccinate	Not Available

#### 14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
mica	Not Available
C.I. Pigment White 6	Not Available
ferric oxide	Not Available
aluminium	Not Available
propylene glycol	Not Available
calcium carbonate	Not Available
alcohols C11-14-iso-, C13-rich, ethoxylated	Not Available
C.I. Pigment Yellow 42	Not Available
C.I. Pigment Black 7	Not Available
4-nonylphenol, branched, ethoxylated	Not Available
kaolin	Not Available
2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol	Not Available
octylphosphonic acid	Not Available
polypropylene glycol	Not Available
monoisobutanolamine	Not Available
stannic oxide	Not Available
zinc pyrithione	Not Available
diethylene glycol	Not Available
sodium dioctyl sulfosuccinate	Not Available

## SECTION 15 Regulatory information

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

#### mica 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)

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

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)

**aluminium is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

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)

**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)

**alcohols C11-14-iso-, C13-rich, ethoxylated is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

**C.I. Pigment Yellow 42 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 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 for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

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

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

**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)

**2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

**octylphosphonic acid is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

**polypropylene glycol is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

**monoisobutanolamine is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

**stannic oxide 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)

**zinc pyrithione is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

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

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

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

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

Australian Inventory of Industrial Chemicals (AIIC)

**diethylene glycol 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 5

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

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)

**Additional Regulatory Information**

Not Applicable

**National Inventory Status**

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (octylphosphonic acid)
Canada - NDSL	No (mica; C.I. Pigment White 6; ferric oxide; aluminium; propylene glycol; alcohols C11-14-iso-, C13-rich, ethoxylated; C.I. Pigment Yellow 42; C.I. Pigment Black 7; 4-nonylphenol, branched, ethoxylated; kaolin; 2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol; polypropylene glycol; monoisobutanolamine; stannic oxide; zinc pyrithione; diethylene glycol; sodium dioctyl sulfosuccinate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (alcohols C11-14-iso-, C13-rich, ethoxylated)
Japan - ENCS	No (mica; aluminium; C.I. Pigment Yellow 42; kaolin)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	No (mica)
Taiwan - TCSI	Yes
Mexico - INSQ	No (octylphosphonic acid)
Vietnam - NCI	Yes
Russia - FBEPH	No (C.I. Pigment Yellow 42; octylphosphonic acid; zinc pyrithione)
<b>Legend:</b>	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

## SECTION 16 Other information

<b>Revision Date</b>	08/10/2024
<b>Initial Date</b>	26/09/2024

## SDS Version Summary

Version	Date of Update	Sections Updated
2.1	26/09/2024	Toxicological information - Acute Health (eye), Toxicological information - Acute Health (inhaled), Toxicological information - Acute Health (skin), Toxicological information - Acute Health (swallowed), First Aid measures - Advice to Doctor, Physical and chemical properties - Appearance, Toxicological information - Chronic Health, Hazards identification - Classification, Disposal considerations - Disposal, Exposure controls / personal protection - Engineering Control, Ecological Information - Environmental, 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 (inhaled), First Aid measures - First Aid (skin), First Aid measures - First Aid (swallowed), Handling and storage - Handling Procedure, Stability and reactivity - Instability Condition, Exposure controls / personal protection - Personal Protection (other), Exposure controls / personal protection - Personal Protection (Respirator), Exposure controls / personal protection - Personal Protection (eye), Exposure controls / personal protection - Personal Protection (hands/feet), Accidental release measures - Spills (major), Accidental release measures - Spills (minor), Handling and storage - Storage (storage incompatibility), Handling and storage - Storage (storage requirement), Handling and storage - Storage (suitable container), Toxicological information - Toxicity and Irritation (Other), Transport information - Transport, Identification of the substance / mixture and of the company / undertaking - Use
3.1	08/10/2024	Toxicological information - Acute Health (eye), Toxicological information - Acute Health (inhaled), Toxicological information - Acute Health (skin), Toxicological information - Acute Health (swallowed), First Aid measures - Advice to Doctor, Physical and chemical properties - Appearance, Toxicological information - Chronic Health, Hazards identification - Classification, Disposal considerations - Disposal, Exposure controls / personal protection - Engineering Control, Ecological Information - Environmental, Firefighting measures - Fire Fighter (extinguishing media), Firefighting measures - Fire Fighter (fire/explosion hazard), Firefighting measures - Fire Fighter (fire fighting), Firefighting measures - Fire Fighter (fire incompatibility), First Aid measures - First Aid (eye), First Aid measures - First Aid (inhaled), First Aid measures - First Aid (skin), First Aid measures - First Aid (swallowed), Handling and storage - Handling Procedure, Composition / information on ingredients - Ingredients, Stability and reactivity - Instability Condition, Exposure controls / personal protection - Personal Protection (other), Exposure controls / personal protection - Personal Protection (Respirator), Exposure controls / personal protection - Personal Protection (eye), Exposure controls / personal protection - Personal Protection (hands/feet), Accidental release measures - Spills (major), Accidental release measures - Spills (minor), Handling and storage - Storage (storage incompatibility), Handling and storage - Storage (storage requirement), Handling and storage - Storage (suitable container), Transport information - Transport, Name

## Other information

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

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

## Definitions and abbreviations

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



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

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