

Tremflex 25

Tremco CPG Australia Pty Ltd

Chemwatch Hazard Alert Code: 3

Chemwatch: 7995-27

Version No: 2.1

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

Initial Date: 10/11/2025

Revision Date: 10/11/2025

Print Date: 24/02/2026

L.GHS.AUS.EN.E

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

Product Identifier

Product name	Tremflex 25
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	Chemicals for construction or industry. Use according to manufacturer's directions.
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Details of the manufacturer or importer of the safety data sheet

Registered company name	Tremco CPG Australia Pty Ltd
Address	12/4 Southridge Street Eastern Creek NSW 2766 Australia
Telephone	1800 318 038
Fax	02 9638 2955
Website	tremco.com.au
Email	orders@tremco.com.au

Emergency telephone number

Association / Organisation	CHEMTREC AUSTRALIA (SYDNEY)
Emergency telephone number(s)	+612 9037 2994 (Aus)
Other emergency telephone number(s)	+1 703-741-5500 (Worldwide)

SECTION 2 Hazards identification

Classification of the substance or mixture

HAZARDOUS CHEMICAL. NON-DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

Chemwatch Hazard Ratings

	Min	Max	
Flammability	0		
Toxicity	1		
Body Contact	3		
Reactivity	1		
Chronic	3		

0 = Minimum
1 = Low
2 = Moderate
3 = High
4 = Extreme

Poisons Schedule	Not Applicable
Classification ^[1]	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Sensitisation (Respiratory) Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Germ Cell Mutagenicity Category 2, Carcinogenicity Category 2, Reproductive Toxicity Category 1B, Hazardous to the Aquatic Environment Acute Hazard Category 2
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.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H335	May cause respiratory irritation.
H341	Suspected of causing genetic defects.
H351	Suspected of causing cancer.
H360FD	May damage fertility. May damage the unborn child.
H401	Toxic to aquatic life.

Precautionary statement(s) Prevention

P202	Do not handle until all safety precautions have been read and understood.
P261	Avoid breathing mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P284	[In case of inadequate ventilation] wear respiratory protection.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
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.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
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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No further product hazard information.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
9009-54-5	44.5	<u>polyurethane polymer</u>
471-34-1	28	<u>calcium carbonate</u>
117-81-7	22	<u>di-sec-octyl phthalate</u>
13463-67-7	5	<u>titanium dioxide</u>
2530-83-8	0.5	<u>gamma-glycidoxypropyltrimethoxysilane</u>

Legend: 1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available

SECTION 4 First aid measures

Description of first aid measures

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

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

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Firefighting measures**Extinguishing media**

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

Special hazards arising from the substrate or mixture

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

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<p>Combustible. Will burn if ignited. Combustion products include:</p> <ul style="list-style-type: none"> ▶ carbon monoxide (CO) ▶ carbon dioxide (CO₂) <p>hydrogen chloride phosgene nitrogen oxides (NO_x) phosphorus oxides (PO_x) silicon dioxide (SiO₂) metal oxides</p> <ul style="list-style-type: none"> ▶ other pyrolysis products typical of burning organic material. <p>May emit poisonous fumes. May emit corrosive fumes.</p>
HAZCHEM	Not Applicable

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

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid contact with skin and eyes. ▶ Wear impervious gloves and safety goggles. ▶ Trowel up/scrape up. ▶ Place spilled material in clean, dry, sealed container. ▶ Flush spill area with water.
Major Spills	<ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Neutralise/decontaminate residue (see Section 13 for specific agent). ▶ Collect solid residues and seal in labelled drums for disposal. ▶ 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.

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SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ Avoid skin contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ DO NOT allow material to come in direct contact with human skin or eyes. ▶ DO NOT allow material to come in contact with exposed food or food contact surfaces. ▶ Suitable PPE must be worn at all times. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	<ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ No smoking, naked lights or ignition sources. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Metal can or drum ▶ Packaging as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Avoid strong acids, bases. ▶ Avoid reaction with oxidising agents

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	calcium carbonate	Calcium carbonate	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Workplace exposure limits for airborne contaminants (WEL list) (From 1 December 2026) - Appendix A - Workplace Exposure Limits	calcium carbonate	Calcium carbonate	10 mg/m3	Not Available	Not Available	a Containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	di-sec-octyl phthalate	Di-sec-octyl phthalate	5 mg/m3	10 mg/m3	Not Available	Not Available
Australia Workplace exposure limits for airborne contaminants (WEL list) (From 1 December 2026) - Appendix A - Workplace Exposure Limits	di-sec-octyl phthalate	Di-sec-octyl phthalate	2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	titanium dioxide	Titanium dioxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Workplace exposure limits for airborne contaminants (WEL list) (From 1 December 2026) - Appendix A - Workplace Exposure Limits	titanium dioxide	Titanium dioxide	10 mg/m3	Not Available	Not Available	a Containing no asbestos and < 1% crystalline silica.

MATERIAL DATA

Exposure controls

Appropriate engineering controls	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. An approved self contained breathing apparatus (SCBA) may be required in some situations.</p> <p>Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p>
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	Type of Contaminant:	Air Speed:
	solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)
Within each range the appropriate value depends on:		
	Lower end of the range	Upper end of the range
	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
	3: Intermittent, low production.	3: High production, heavy use
	4: Large hood or large air mass in motion	4: Small hood-local control only
Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.		
Individual protection measures, such as personal protective equipment		
Eye and face protection	<ul style="list-style-type: none"> Safety glasses with side shields. Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59]. 	
Skin protection	See Hand protection below	
Hands/feet protection	<ul style="list-style-type: none"> Wear chemical protective gloves, e.g. PVC. Wear safety footwear or safety gumboots, e.g. Rubber 	
Body protection	See Other protection below	
Other protection	<ul style="list-style-type: none"> Overalls. P.V.C apron. Barrier cream. Skin cleansing cream. Eye wash unit. 	

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

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

Tremflex 25

Material	CPI
BUTYL	A
VITON	A
NITRILE	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® 37-185
AlphaTec® 38-612
AlphaTec® 58-008
AlphaTec® 58-530B
AlphaTec® 58-530W
AlphaTec® 58-735

Respiratory protection

Type A-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	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-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.

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AlphaTec® 79-700
AlphaTec® Solvex® 37-675
DermaShield™ 73-711
MICROFLEX® 73-847

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

- 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.
- Class P2 particulate filters are used for protection against mechanically and thermally generated particulates or both.
- P2 is a respiratory filter rating under various international standards, Filters at least 94% 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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Black, grey or white paste with faint odour; does not mix with water.		
Physical state	Non Slump Paste	Relative density (Water = 1)	~1.3 @20C
Odour	Slight	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Applicable	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)	~80	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Heat of Combustion (kJ/g)	Not Available	Ignition Distance (cm)	Not Available
Flame Height (cm)	Not Available	Flame Duration (s)	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	Enclosed Space Ignition Deflagration Density (g/m3)	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

a) Acute Toxicity	Based on available data, the classification criteria are not met.
b) Skin Irritation/Corrosion	There is sufficient evidence to classify this material as skin corrosive or irritating.

c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	There is sufficient evidence to classify this material as sensitising to skin or the respiratory system
e) Mutagenicity	There is sufficient evidence to classify this material as mutagenic
f) Carcinogenicity	There is sufficient evidence to classify this material as carcinogenic
g) Reproductivity	There is sufficient evidence to classify this material as toxic to reproductivity
h) STOT - Single Exposure	There is sufficient evidence to classify this material as toxic to specific organs through single exposure
i) STOT - Repeated Exposure	Based on available data, the classification criteria are not met.
j) Aspiration Hazard	Based on available data, the classification criteria are not met.
Inhaled	<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>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p>
Ingestion	<p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>Phthalates (aromatic dicarboxylic acid esters), in general, exhibit low toxicity, partly because of poor absorption but mainly as a result of rapid metabolism in which the esters are saponified to phthalic acid (which is rapidly excreted) and the parent alcohol (which is subsequently metabolised). The pathology of these compounds seems to be related to the released alcohol and its biological effects. The rate of absorption of ingested phthalate esters is influenced by the content of dietary fat. Ingested phthalate esters may to a lesser degree be absorbed as the monoester derivatives or in the case of di(2-ethylhexyl)phthalate, as the diester. Cumulative toxicity of the phthalates has been observed on repeated administration. Both di-n-octyl phthalate and di(2-ethylhexyl)phthalate were found to have 22-28 times greater toxicity (based on LD50s) following repeated administration to animals. The liver has been shown to be the target organ affected by the phthalates. In general phthalates have induced liver enlargement; this increase in liver weight has been attributed to rapid cell division (hyperplasia) along with the detachment of cells (hypertrophy). The increase in liver weight caused by phthalates has been found to reverse to normal or even below normal levels on prolonged exposure.</p> <p>Exposure to phthalates, in general, has been found to be associated with a reduction in circulating cholesterol and serum triglyceride levels which accounted for a reduction in liver steroidogenesis. The phthalates also effect carbohydrate metabolism in the liver producing depleted glycogen electron transport inhibitors following interaction with mitochondria. Testicular atrophy produced in rats during feeding studies depends on the length and structure of the alcohol; in general the lower molecular weight esters produce the more severe effects. The toxicity of phthalic acid isomers decreases in the order o-phthalic acid, isophthalic acid and terephthalic acid. Phthalic acid is not metabolised but is excreted, unchanged, in the urine and faeces. Terephthalic acid appears to potentiate the biological effects of substances such as antibiotics, thiamine and sulfonamides.</p>
Skin Contact	<p>The material may accentuate any pre-existing dermatitis condition</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>The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either</p> <ul style="list-style-type: none"> ▶ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or ▶ produces significant, but moderate, 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. <p>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	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	<p>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. Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population.</p> <p>Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.</p> <p>Harmful: 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>There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: - clear evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects.</p> <p>There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of:</p> <ul style="list-style-type: none"> - clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. <p>Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.</p> <p>The various phthalates have different uses, chemical structures and toxicity profiles. It is therefore difficult to generalise about the safety of all phthalates as a group. The main health concern associated with some phthalates is that animal studies have shown that high regular doses can affect the reproductive system in developing young, particularly males. While there is no significant risk to the general population, young children may experience higher exposures than the general population if they chew or suck on phthalate-containing toys, or if they ingest phthalates over a long period from other products containing high levels of phthalates.</p> <p>In animal tests, phthalates have been shown to "feminise" male animals, increasing the likelihood of small or undeveloped testes, undescended testicles, and low sperm counts. A 2005 study also linked higher foetal exposure to phthalates through the mother's blood with</p>

increased risk of developmental abnormalities in male infants. Higher phthalate levels are also associated with lower testosterone production and reduced sperm count in men.

One study suggested that high levels of phthalates may be connected to the current obesity epidemic in children. It was found that obese children show greater exposure to phthalates than non-obese children. It was reported that the obesity risk increases according to the level of the chemical found in the children's bloodstream. In a national cross-section of U.S. men, concentrations of several prevalent phthalate metabolites showed statistically significant correlations with abnormal obesity and insulin resistance. A further study found that people with elevated phthalate levels had roughly twice the risk of developing diabetes compared with those with lower levels. This study also found that phthalates were associated with disrupted insulin production.

Much of the current research on effects of phthalate exposure has been focused towards children and men's health, however, women may be at higher risk for potential adverse health effects of phthalates due to increased cosmetic use. According to in vivo and observational studies there is an association between phthalate exposure and endocrine disruption leading to development of breast cancer. This finding may be associated with the demethylation of the oestrogen receptor complex in breast cancer cells.

A Russian study describes exposure by workers to mixed phthalates (and other plasticisers) - pain, numbness and spasms in the upper and lower extremities were related to duration of exposures. Symptoms usually developed after the sixth or seventh year of work. Neurological studies revealed the development of polyneuritis in about 30% of the workers involved in this study. About 30% of the workforce showed depression of the vestibular receptors. Because the study described mixed exposures it is difficult to determine what, if any, unique role was played by the phthalates. Increased incidences of anovulatory reproductive cycles and low oestrogen concentrations were reported among Russian women working with phthalate plasticisers; the abnormal cycles were associated with spontaneous abortion. The specific phthalates implicated, dose levels and other data were not reported. It has been alleged that the phthalates mimic or interfere with sex packaging) and are used as ingredients in paints, inks and adhesives. Their potential for entering the human body is marked. They have been added to a list of chemicals (including alkyl phenolics, polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs) and dioxins) which are implicated in reducing sperm counts and fertility in males a phenomenon which has apparently arisen since the mid 1960s.

Phthalates are generally considered to be in a class of endocrine disruptors known as "xenoestrogens," for their ability to mimic the effect of oestrogen on the body.

Although the human foetus is "bathed" in naturally occurring oestrogens during pregnancy it is suggested that it has developed a protective mechanism against natural oestrogens but is not safe from synthetic variants. These tend to accumulate in body fats which sets them apart from the natural product. During early pregnancy, fats are broken down and may flood the body with concentrated pollutants

Human phthalate exposure during pregnancy results in decreased anogenital distance among baby boys. Boys born to mothers with the highest levels of phthalates were 7 times more likely to have a shortened anogenital distance.

While anogenital distance is routinely used as a measure of foetal exposure to endocrine disruptors in animals, this parameter is rarely assessed in humans, and its significance is unknown

One study also found that female animals exposed to higher levels of phthalates experienced increased risk of miscarriage, a common symptom of excessive estrogen levels in human women, and stillbirth. Prematurity may also be linked to phthalate exposure.

Another study found a link between exposure to phthalates and increased rates of childhood obesity.

In adult human men, phthalates have been linked to greater waist circumference and higher insulin resistance, a common precursor to type 2 (adult onset) diabetes. They have been linked to thyroid irregularities, asthma, and skin allergies in both sexes. Though the exact mechanism is unclear, studies have linked higher rates of respiratory infections and other symptoms in children living in houses with vinyl floors. One possible explanation is inhalation of dust tainted by phthalates, which are used in cosmetics such as nail polishes and hand creams precisely because of their ability to bind to human tissues.

Animal studies have shown increased risks of certain birth defects (including the genital abnormalities and, in rats, extra ribs) and low birth rates in rats whose mothers were fed higher levels of phthalates.

These effects on foetal development are of particular concern because young women of childbearing age often have higher than average phthalate levels in the body thanks to their use of cosmetics, many of which contain phthalates.

The EU has applied limitations to the use of several phthalates in general food contact applications (packaging and closures) and medical device applications. The USA has introduced regulation of phthalate esters as components of children's toys and childcare articles for children under the age of 12 that could be 'placed in the mouth'.

Endocrine disruptors such as phthalates can be added to the effects of other endocrine disruptors, so even very small amounts can interact with other chemicals to have cumulative, adverse "cocktail effects"

Large amounts of specific phthalates fed to rodents have been shown to damage their liver and testes, and initial rodent studies also indicated hepatocarcinogenicity. Later studies on primates showed that the mechanism is specific to rodents - humans are resistant to the effect

Studies conducted on mice exposed to phthalates in utero did not result in metabolic disorder in adults. However, "At least one phthalate, monoethylhexyl phthalate (MEHP) has been found to interact with all three peroxisome proliferator-activated receptors (PPARs) PPARs are members of the nuclear receptor superfamily involved in lipid and carbohydrate metabolism.

Prenatal exposure to phthalates may affect children's mental, motor and behavioral development during the preschool year.

A 2009 study found that prenatal phthalate exposure was related to low birth weight in infants. Low birth weight is the leading cause of death in children under 5 years of age and increases the risk of cardiovascular and metabolic disease in adulthood. Another study found that women who deliver prematurely have, on average, up to three times the phthalate level in their urine compared to women who carry to term. Several findings point to a statistically significant correlation between urine phthalate concentrations in children and symptoms of attention deficit hyperactivity disorder (ADHD)

Pure calcium carbonate does not produce pneumoconiosis probably being eliminated from the lungs slowly by solution.

As mined, unsterilised particulates can carry bacteria into the air passages and lungs, producing infection and bronchitis.

2-Ethylhexanoic acid (2-EHA) its esters and its salts are of concern to human health because of their potential to induce carcinogenicity, liver toxicity and developmental/reproductive toxicity. 2-EHA is of low acute oral and dermal toxicity, is a mild skin irritant and a severe eye irritant. It is not mutagenic in Ames test, but is capable of inducing chromosome aberration and sister chromatid exchanges in vitro, liver toxicity and liver tumours after repeated dose treatment. In addition, 2-EHA has been associated with reproductive and developmental toxicity in experimental animals.

2-EHA is quickly resorbed orally, dermally and following inhalation and almost fully excreted mainly in urine. As in the case of fatty acids, degradation mainly takes place by means of peroxisomal beta-oxidation.

2-EHA has been shown to be a liver and developmental toxicant in animal studies at high doses; in developmental toxicity studies, it was postulated that the maternal liver toxicity began a cascade of effects that included metallothionein (MT) induction, zinc accumulation in the liver due to MT binding, and a resulting zinc deficiency in the developing embryo; the zinc deficiency causes the developmental toxicity; a reproductive/developmental toxicity study was also performed with up to 1% dietary di-2-ethylhexyl terephthalate (DEHT; a 2-ethylhexanoic acid precursor); no reproductive or developmental effects were observed, suggesting that the process of metabolic conversion of DEHT to 2-ethylhexanol and subsequent hydrolysis to 2-ethylhexanoic acid results in a time course of 2-ethylhexanoic acid appearance such that allows clearance before sufficient levels can arise to produce acute liver toxicity.

The closely related substance, 2-ethylhexanol is metabolised to form 2-ethylhexanoic acid and so presents a similar toxicity profile..

2-Ethylhexanoic acid (CAS No. 149-57-5) is classified as hazardous, as a Category 3 reproductive toxin, with the risk phrase "Possible risk of harm to the unborn child (Xn; R63). These effects were noted in the absence of signs of maternal toxicity. The lowest observed adverse effect level (LOAEL) for developmental toxicity was reported to be 100 mg/kg bw/day. Effects on fertility were also reported, with evidence being sufficient to warrant classification as potentially toxic to fertility.

2-Ethylhexanol (CAS No. 104-76-7) was reported to cause developmental toxicity, but not teratogenicity, in rats following treatment via the oral route (NICNAS a). These effects were noted in the absence of signs of marked maternal toxicity. The no observed adverse effect level (NOAEL) for developmental toxicity was reported to be 130 mg/kg bw/day.

Various studies on reproduction toxicity have produced indications of an embryotoxic effect of 2-EHA. After oral administration, NOAEL values for maternal toxicity and foetotoxic effects of 2-EHA were determined in rabbits at 25 and >250 mg/kg body weight/day and in rats at 250 and 100 mg/kg body weight/day. The foetotoxic findings in rats were based on a reduced skeleton ossification at the next higher dose (250 mg/kg body weight/day). No teratogenic effects were observed in this study. In comparison with the structural isomer valproic acid, a known human teratogen, 2-EHA does have similar reprotoxic effects at maternal toxic doses in animal experiments but a far lower potency. Following sub-chronic oral administration of 2-EHA, critical effects like liver changes (higher relative liver weight, histological changes in hepatocytes) were observed in rats and mice and histological renal tubule results were observed in mice. Furthermore, statistically significant, higher cholesterol values were found in all treated male rats (61, 303 and 917 mg/kg body weight/day) and in male and female mice in the middle and high dose groups (885-3139 mg/kg body weight/day). In rats the maximum dose with no adverse effect (NOAEL) was 61 mg/kg body weight/day. In bacterial test systems, mutagenicity studies produced negative findings. In test systems with mammalian cells,

by contrast, the findings were weakly positive. Cytogenetic and SCE studies involving CHO cells were positive, one SCE test in human lymphocytes was questionably positive and one experiment concerning tritium-thymidine incorporation into the DNA of mouse lymphocytes was negative. Furthermore, An unpublished micronucleus study on the bone marrow of CD-1 mice was conducted in compliance with OECD Guideline 474. No significant increase in the micronuclei was observed at doses of 400, 800 or 1,600 mg/kg body weight (Inveresk Research International Ltd, 1994). Furthermore, *in vitro* and *in vivo* genotoxicity data (micronucleus test, dominant lethal test) are available for 2-ethylhexanol which is rapidly and quantitatively converted into 2-EHA in metabolism studies. This data do not indicate any genotoxic potential which means that such an effect of 2-EHA is not likely either. As 2-EHA can induce both DNA synthesis and inhibition of intercellular communication in hepatic cells, a tumour-promoting potential in rodents, comparable to that of other peroxisome proliferators, cannot be ruled out. The carcinogenic effect of peroxisome proliferators in rodents (e.g. of di(2-ethylhexyl)phthalate, DEHP) is not deemed to be relevant for humans.

Calcium/zinc and barium/zinc salts of 2-EHA are used as thermo-stabilisers for PVC, together with co-stabilisers like polyols or epoxy compounds, in order to capture the hydrochloride cleaved during the thermal loading of PVC; in addition various salts are used in other food and beverage containers as plasticisers. The migration of 2-EHA from the sealing compounds in the metal lids. has been demonstrated in food contamination. The potential for human exposure to 2-EHA therefore is significant.

Chemicals containing epoxy groups are of concern for cancer effects, though the concern is lower for epoxy groups with di-substituted carbons (US EPA 1994)

The epoxide group is an alkylating agent and thus may produce damage to nucleotides found within the cell; such damage is potentially tumourigenic. Alkylating agents may damage the stem cell which acts as the precursor to components of the blood. Loss of the stem cell may result in pancytopenia (a reduction in the number of red and white blood cells and platelets) with a latency period corresponding to the lifetime of the individual blood cells. Granulocytopenia (a reduction in granular leukocytes) develops within days and thrombocytopenia (a disorder involving platelets), within 1-2 weeks, whilst loss of erythrocytes (red blood cells) needs months to become clinically manifest. Aplastic anaemia develops due to complete destruction of the stem cells.

Chemicals containing epoxy functional groups are of concern for reproductive effects, though the concern for epoxy groups with di-substituted carbons is lower than that for singly substituted epoxy groups (US EPA, 1994).

High blood concentrations of calcium ion may give rise to vasodilation and depress cardiac function leading to hypotension and syncope.

Calcium ions enhance the effects of digitalis on the heart and may precipitate digitalis intoxication. Calcium salts also reduce the absorption of tetracyclines

In neonates calcification of soft-tissue has been observed following therapeutic administration.

Some studies show that large quantities of calcium intake can cause hypercalcaemia, which can in turn lead to renal failure. Renal failure can occur within hours or days or, alternatively, settles gradually, evolving over several years until it reaches terminal stages. Similarly, acute renal failure can also develop into chronic forms of the disease.

Hypercalcaemia conditions can be associated with normal or reduced calcium serum levels, as the body tends to maintain a balanced metabolism of the mineral, known as the compensation phase. When there is a slight increase in the concentration of ions in the blood, calcium excretion markedly increases, while intestinal absorption decreases. After kidney damage has set in, a loss of calcium may occur, thereby decreasing the serum concentration.

Serum protein levels may decrease as a result of proteinuria in cases of renal complications. Proteinuria is an indicator of kidney disease and represents an independent risk factor for the progression of such a condition. Increased serum creatinine levels may represent an important parameter, given that kidney diseases are associated with increased serum creatinine levels. When renal pathology occurs, a progressive loss of glomerular filtration begins, resulting in increased plasma creatinine concentrations. During the course of kidney failure, discrete, but constant, increments in plasma creatinine levels occur.

Renal disease with albuminuria may also be the cause of hypoalbuminemia in patients with liver disease. In cases of established liver damage, increased calcium urinary excretion may occur. Therefore, a similar increase may cause the decline in serum calcium levels in the current study.

For di-sec-octyl phthalate:

Oral studies of 90-days to 2-years in rat, 1-year in guinea pig and up to 1-year in dog have shown a no-effect level of about 60 mg/kg/day. Higher doses produced growth retardation and increased weights of livers and kidneys.

Rats and mice fed on diets containing 6000-12000 (rats) and 3000-6000 (mice) mg/kg body weight for 103 weeks showed an increased incidence of hepatocellular carcinomas in female rats and male and female mice, and an increased incidence of either hepatocellular carcinomas or neoplastic nodules in male rats. About 35% of the hepatocellular carcinomas in mice had metastasised to the lungs.

The substance can cause testicular damage in rats (dietary and gavage studies) with a no-effect level in 0.3% to 0.5% in the diet. Inhalation or dermal exposures did not produce testicular effects. When the substance was fed to pregnant rats (5 ml/kg) it produced slight effects on embryonic and foetal development with skeletal abnormalities more common.

Tremflex 25	TOXICITY	IRRITATION
	Not Available	Not Available
polyurethane polymer	TOXICITY	IRRITATION
	Not Available	Not Available
calcium carbonate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (Rodent - rabbit): 750ug/24H - Severe
	Inhalation (Rat) LC50: >3 mg/14h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Skin (Rodent - rabbit): 500mg/24H - Moderate
		Skin: no adverse effect observed (not irritating) ^[1]
di-sec-octyl phthalate	TOXICITY	IRRITATION
	Dermal (Guinea Pig) LD50: 10000 mg/kg ^[2]	Eye (Rodent - rabbit): 500mg - Mild
	Inhalation (Rat) LC50: >10.62 mg/14h ^[2]	Eye (Rodent - rabbit): 500mg/24H - Mild
	Oral (Mouse) LD50; 1500 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]
		Skin (Rodent - rabbit): 500mg/24H - Mild
		Skin: adverse effect observed (irritating) ^[1]
titanium dioxide	TOXICITY	IRRITATION
	dermal (hamster) LD50: >=10000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (Rat) LC50: >2.28 mg/14h ^[1]	Skin (Human): 300ug/3D (intermittent) - Mild
	Oral (Rat) LD50: >=2000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]

gamma-glycidioxypropyltrimethoxysilane	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 4247.9 mg/kg ^[2]	Eye (Rodent - rabbit): 100mg - Mild
	Inhalation (Rat) LC50: >5.3 mg/L4h ^[2]	Eye: adverse effect observed (irreversible damage) ^[1]
	Oral (Rat) LD50: 7010 mg/kg ^[2]	Skin (Rodent - rabbit): 500mg - Mild
		Skin: no adverse effect observed (not irritating) ^[1]

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

POLYURETHANE POLYMER	Data for polyurethane foam. Inhalation (human)TCLo: 12 mg/m3/11W-C No data available [RTECS] 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.
CALCIUM CARBONATE	No evidence of carcinogenic properties. No evidence of mutagenic or teratogenic effects. The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
DI-SEC-OCTYL PHTHALATE	Oral (rat) NOAEL: 28.9-36.1 mg/kg/day Gastrointestinal changes, respiratory system changes, somnolence, haemorrhage, necrotic changes in GI tract, lowered blood pressure, liver, endocrine tumours, foetotoxicity, paternal effects, maternal effects, specific developmental abnormalities (hepatobiliary system, musculoskeletal system, cardiovascular system, urogenital system, central nervous system, eye/ear), foetolethality recorded. Di-sec-octyl phthalate (DEHP) is not acutely toxic in small laboratory animals via the oral route. The oral LD50 reported for mice is 26.3 g/kg; for rats, it is 33.8 g/kg. No skin irritation or sensitisation potential has been demonstrated in either animals or humans, and the lethal dermal dose in rabbits is about 25 ml/kg. Deaths in rats and chronic diffuse inflammation of the lung in mice exposed to DEHP at unspecified levels have been reported. Long-term dietary toxicity studies in rats, guinea pigs, and dogs have established a no-effect dose level of about 60 mg/kg/day, and no carcinogenic or histologic abnormalities were observed at this level. Higher doses were associated with growth retardation and increased liver and kidney weights but not histologic abnormalities. Metabolic studies have demonstrated that laboratory animals do not appreciably metabolise DEHP. Teratogenicity studies in pregnant rats indicated that fertility is unaffected at doses of 0.1, 0.2, or 0.33 percent of the acute intraperitoneal LD50 dose for rats, although slight effects on embryonic and foetal development were observed in these animals; skeletal deformities were the most common teratogenic effects observed. Mutagenic effects were observed at intravenous doses of one-third, one-half, and two-thirds of the acute LD50; these effects are consistent with DEHP's ability to produce dominant lethal mutations. A study of workers exposed to a mixture of the vapors of diethyl phthalate, dibutyl phthalate, and di-2-ethylhexyl phthalate reported that exposures to 1 to 6 ppm caused no peripheral polyneuritis. However, Russian investigators examined male and female workers exposed to between 1.7 and 66 mg/m3 of various combinations of airborne phthalates (including butyl phthalate, higher aryl phthalates, dioctyl phthalate and others) and noted complaints of pain, numbness, and spasms in the upper and lower extremities after six to seven years of exposure. Polyneuritis was observed in 32 percent of the workers studied, and 78 percent of these workers showed depression of vestibular receptors. The material may produce peroxisome proliferation. Peroxisomes are single, membrane limited, cytoplasmic organelles that are found in the cells of animals, plants, fungi and protozoa. Peroxisome proliferators include certain hypolipidaemic drugs, phthalate ester plasticisers, industrial solvents, herbicides, food flavours, leukotriene D4 antagonists and hormones. Numerous studies in rats and mice have demonstrated the hepatocarcinogenic effects of peroxisome proliferators, and these compounds have been unequivocally established as carcinogens. However it is generally conceded that compounds inducing proliferation in rats and mice have little, if any, effect on human liver except at very high doses or extreme conditions of exposure. The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. Transitional Phthalate Esters: produced from alcohols with straight-chain carbon backbones of C4 to C6. This subcategory also includes a phthalate produced from benzyl alcohol as one ester group with the second ester composed of an alkyl group with a C5 carbon backbone and butyrate group. Phthalate esters containing >10% C4 to C6 molecules were conservatively included in this subcategory. Branched C7 and C8 isomers (di-iso-heptyl, di-iso-octyl and diethylhexyl phthalates) in contrast to linear dihexyl and dioctyl phthalate are members of this family. Transitional phthalates have varied uses, but are largely used as plasticisers for PVC. Physicochemical properties also vary in that the lower molecular weight transitional phthalates are more water-soluble than higher molecular weight transitional phthalates, but none would be characterised as highly water soluble. Transitional phthalates have lower water solubility than the low molecular weight phthalates and except for butylbenzyl phthalate (BBP), existing data suggest they do not exhibit acute or chronic aquatic toxicity. What distinguishes some of the transitional phthalates from others is their greater mammalian toxicity potential, particularly with regard to reproductive and developmental effects, compared to either the low or high molecular weight phthalate subcategories Acute Toxicity. The available data on phthalates spanning the carbon range from C4 to C6 indicate that phthalate esters in the transitional subcategory are minimally toxic by acute oral and dermal administration. The oral LD50 value for BBP exceeds 2 g/kg, and for materials with higher molecular weights, the LD50 values exceed the maximum amounts which can be administered to the animals in a manner consistent with the principles of responsible animal use. One member of this subcategory, diethylhexyl phthalate (DEHP), has been tested for acute inhalation toxicity. It did not cause an effect at the highest concentration tested. Further, considering the low volatility of these substances, inhalation exposure at toxicologically significant levels is not anticipated. Repeated Dose Toxicity. Several substances in the C4 to C6 range, including BBP, have been tested for repeated dose toxicity in studies ranging from 3 weeks to 2 years. The principal effects found in these studies were those associated with peroxisome proliferation including liver enlargement and induction of peroxisomal enzymes. As shown in a comparative study of liver effects, the strongest inducers of peroxisome proliferation are diisononyl phthalate (DINP) and di-iso-decyl phthalate (DIDP) with substances of shorter chain length (e.g., BBP) showing much less pronounced effects. Thus it is reasonable to conclude that other members of this subcategory would show effects similar to BBP and less pronounced than DINP or DIDP. It should also be noted that the relevance of these findings to human health is, at best, questionable. It has been shown that these effects are mediated through the peroxisome proliferation-activated receptor alpha (PPARα) and that levels of PPARα are much higher in rodents than they are in humans. Thus one would expect humans to be substantially less responsive than rodents to peroxisome proliferating agents. Empirical evidence that this is true is provided by studies in primates in which repeated administration of DINP had no effects on liver, kidney or testicular parameters. Several of the substances in the transitional phthalate esters subcategory, however, have been shown to produce testicular atrophy when given to juvenile rats at high levels. Testicular atrophy has been associated with BBP and other substances with C4 to C6 linear carbon chains. However, molecules with fewer than 4 or more than 6 carbons did not produce testicular atrophy in these studies. Although the relevance of these data are uncertain, as the testes is not a target organ for diethylhexyl phthalate (DEHP) in primates, these data do provide one of the distinguishing toxicological characteristics of this subcategory and are one of the underlying reasons supporting the differentiation of phthalate esters on the basis of length of the linear region of the carbon chain. Genetic Toxicity (Salmonella). A number of the substances in this subcategory including the reference substance BBP has been assessed in the Salmonella and mouse lymphoma assays. All of these substances were inactive in these assays.

Tremflex 25

Chromosomal Aberrations. BBP and dihexyl phthalate (DHP) were inactive in micronucleus assays in mice. DEHP was inactive in a cytogenetics assay in rat bone marrow. Diisoheptyl phthalate was inactive in CHO cells, in vitro.

Reproductive toxicity: A series of studies assessed the structure-activity relationship of the effects of phthalate esters on fertility using a continuous breeding protocol. The test substances included in these studies were diethyl-, dipropyl-, dibutyl-, dipentyl-, d-n-hexyl-, di-2(ethylhexyl)-, and di-n-octyl phthalates. The most profound effects were on fertility (i.e., number of females delivering/ number mated) and number of live births. The substance showing the greatest activity was DEHP which produced effects at dietary levels of 0.1 % with a no effect level of 0.01 %. The next most active compounds were di-n-hexyl- and di-n-pentyl phthalate which showed effects in the range of 0.3 to 0.5 %; no effect levels were not experimentally defined. Dipropyl phthalate had an effect on live birth index at 2.5 % but produced no effects at 1.25 %. Diethyl phthalate and di-n-octyl phthalate were inactive at the highest levels tested, 2.5 % and 5.0 %, respectively. These data demonstrated that molecules with linear alkyl chains of 4 to 6 carbons profoundly affect fertility in rodents, with DEHP being the most active. Molecules with longer or shorter side chains are essentially inactive in these assays. These data were also a basis for the separation of phthalates into three categories based on length of side chain.

In addition to these data there are reproductive toxicity studies on BBP and DEHP.

A 2-generation reproductive study was conducted in rats in which BBP was administered via the diet. Parental effects were limited to changes in body weight, weight gain, and increased absolute and relative liver weights. In the F1 parents, treatment with BBP affected mating and fertility indices and sperm number and motility. The F1 male offspring exhibited shortened anogenital distance, delayed acquisition of puberty and retention of nipples and areolae as well as reproductive effects. The NOAEL of the study was reported to be 3750 mg/ kg for reproductive effects. However, for male F1 and F2 offspring, the NOEL for reproductive effects was reported to be 50 mg/ kg based on reductions in anogenital distance. These studies along with previous data provide a good basis to assess the reproductive effects of C4 to C6 phthalate esters. Although several substances (diheptyl, heptyl nonyl, heptyl undecyl) have ester side chain constituents that predominantly fall in the high molecular weight subcategory, these substances are conservatively assumed to exhibit reproductive effects similar to other transitional phthalates.

Developmental toxicity: There have been extensive studies of the developmental toxicity of BBP and DEHP. These substances produce structural malformations and also affect male reproductive development. No effect levels are in the range of 50 to 300 mg/ kg bw/ day. There is also an unpublished developmental toxicity study of di-isoheptyl phthalate (DIHP). The results of these studies are broadly consistent with the structure-activity relationships previously described, i.e., that phthalate esters with linear carbon chains of C4 to C6 carbons produce much more profound effects than either shorter or longer molecules.

Phthalate esters with >10% C4 to C6 isomers were conservatively placed in the transitional subcategory. This conclusion is supported by developmental test data on "711P" (which showed structural malformations in rats at 1000 mg/ kg/ day with a NOAEL of 200 mg/ kg/ day. "711P" is an equal composition mixture of six phthalate esters consisting of linear and methyl-branched C7, C9, and C11 ester side chains. This test substance is considered by EPA under the following CAS Numbers.: 68515-44-6 (di C7), 68515-45-7 (di C9), 3648-20-2 (di C11), 111381-89-6 (C7, C9), 111381-90-9 (C7, C11), and 111381-91-0 (C9, C11). The overall content of C4 to C6 isomers in "711P" is approximately 10%, based on the contribution from methyl-branched C7 isomers e.g., di C7 (30% C4-C6); C7, C9 (15% C4-C6); and C7, C11 (15 % C4-C6). Test data on 711P were used selectively as read-across data to the C7-containing substances in the mixture, based on the C4 to C6 content of each substance in the mixture.

NOTE: Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.

Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen
[National Toxicology Program: U.S. Dep. of Health & Human Services 2002]

TITANIUM DIOXIDE

* IUCLID

Laboratory (in vitro) and animal studies show, exposure to the material may result in a possible risk of irreversible effects, with the possibility of producing mutation.

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

	<p>female mice.</p> <p>In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative. No significant acute toxicological data identified in literature search.</p> <p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
<p style="text-align: center;">GAMMA-GLYCIDOXYPROPYLTRIMETHOXYSILANE</p>	<p>For alkoxysilanes: Low molecular weight alkoxysilanes (including alkyl orthosilicates) are a known concern for lung toxicity, due to inhalation of vapours or aerosols causing irreversible lung damage at low doses. Alkoxysilane groups that rapidly hydrolyse when in contact with water, result in metabolites that may only cause mild skin irritation. Although there appears to be signs of irritation under different test conditions, based on the available information, the alkoxysilanes cannot be readily classified as a skin irritant. The trimethoxysilane group of chemicals have previously been associated with occupational eye irritation in exposed workers who experienced severe inflammation of the cornea. Based on the collective information, these substances are likely to be severe irritants to the eyes. Methoxysilanes are generally reported to possess higher reactivity and toxicity compared to ethoxysilanes; some methoxysilanes appear to be carcinogenic. In the US, alkoxysilanes with alkoxy groups greater than C2 are classified as moderate concern. Based on available information on methoxysilanes, the possibility that this family causes skin sensitisation cannot be ruled out. Amine-functional methoxysilanes have previously been implicated as a cause of occupational contact dermatitis, often as a result of repeated skin exposure with workers involved in the manufacture or use of the resins containing the chemical during fibreglass production.</p> <p>For gamma-glycidopropyltrimethoxysilane (GPTMS) GPTMS is subject to rapid hydrolysis, and the observed toxicity is expected to be due primarily to methanol and silanetriols. GPTMS has been tested for acute toxicity by the oral, dermal, and inhalation routes of exposure. Reported acute oral LD50s in rats range from 7010 to 16900 mg/kg bw and > 5 ml/kg bw to 22.6 ml/kg bw. The dermal LD50s are 6800 mg/kg bw and 4.0 ml/kg bw. The 4-hour inhalation LC50 was greater than 2.7 mg/L in one study and greater than 5.3 mg/L in another study. GPTMS is mildly irritating to the skin and eyes and is not a known skin sensitiser in humans or in animals. Following inhalation exposures of rats to target aerosol concentrations of 0, 75, 225 and 750 mg/m3 (actual concentrations were 0, 77, 226, 707 mg/m3 (males) and 0, 73, 226, 734 mg/m3 (females)), GPTMS in 9 repeated exposures administered over two weeks, 6 animals in the high dose group died or were sacrificed from three to five days after initiation of the study. These animals had signs of inanition but no acute tissue toxicity. At both the mid and high doses, rats exhibited some clinical signs including a dose-related decrease in body weight. Under the conditions of this study, the No Observed Adverse Effect Concentration is 225 mg/m3. Repeated exposure of rats by gavage to GPTMS doses of 40, 400 and 1000 mg/kg bw/day for 5 days/week for 4 weeks resulted in no test substance-related organ weights effects or gross or microscopic pathological changes. Under the conditions of this study, the NOAEL for the test substance was found to be 1000 mg/kg bw/day. Genotoxicity: GPTMS did not induce chromosomal damage in mouse bone marrow cells by gavage at doses of 500, 1670 and 5000 mg/kg bw/day, or when administered by intraperitoneal (i.p.) injection at 1600 mg/kg bw/day. However, chromosomal damage was induced in mouse bone marrow cells when administered by i.p. in water at doses of 500, 1000 and 2000 mg/kg bw/day. GPTMS induced gene mutations in bacteria. GPTMS induced gene mutations in mouse lymphoma L1578Y TK cells but did not induce forward mutations in CHO cells. GPTMS induced SCE in vitro. There are no in vivo gene mutation data. Carcinogenicity: GPTMS was not considered tumourigenic when applied to the clipped skin of mice (25 ul dose of 25% GPTMS in acetone) three times per week for approximately 78 weeks. Note that there was only one dose level, and this dose was relatively low. Reproductive toxicity: In a one-generation reproduction toxicity study in rats, no reproductive effects were observed at any of the doses tested (250, 500, or 1000 mg/kg bw/day). At 1000 mg/kg bw/day, treatment with GPTMS resulted in the following signs in parental animals: discomfort after dosing (noted for females from early/mid gestation onwards), decreased body weight gain (males), increased mean relative liver and kidney weights (noted for males and females), and histopathological effects on livers and kidneys (males). Based on these data, a NOAEL for parental animals was established at 500 mg/kg bw/day. A NOAEL for reproductive effects was established at 1000 mg/kg bw/day. Developmental toxicity: Three developmental studies have been conducted using GPTMS. In a rabbit study, the maternal NOAEL was 200 mg/kg bw/day and the developmental NOAEL was 400 mg/kg bw/day (the highest dose tested). In a rat study, the NOAELs for both maternal and developmental toxicity were also at the highest dose tested (1000 mg/kg bw/day). In another rat study, developmental effects were observed at the maternally toxic dose of 3000 mg/kg bw/day (again, the highest dose tested). Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit many common characteristics with respect to animal toxicology. One such oxirane is ethyloxirane; data presented here may be taken as representative. for 1,2-butylene oxide (ethyloxirane): Ethyloxirane increased the incidence of tumours of the respiratory system in male and female rats exposed via inhalation. Significant increases in nasal papillary adenomas and combined alveolar/bronchiolar adenomas and carcinomas were observed in male rats exposed to 1200 mg/m3 ethyloxirane via inhalation for 103 weeks. There was also a significant positive trend in the incidence of combined alveolar/bronchiolar adenomas and carcinomas. Nasal papillary adenomas were also observed in 2/50 high-dose female rats with none occurring in control or low-dose animals. In mice exposed chronically via inhalation, one male mouse developed a squamous cell papilloma in the nasal cavity (300 mg/m3) but other tumours were not observed. Tumours were not observed in mice exposed chronically via dermal exposure. When trichloroethylene containing 0.8% ethyloxirane was administered orally to mice for up to 35 weeks, followed by 0.4% from weeks 40 to 69, squamous-cell carcinomas of the forestomach occurred in 3/49 males (p=0.029, age-adjusted) and 1/48 females at week 106. Trichloroethylene administered alone did not induce these tumours and they were not observed in control animals. Two structurally related substances, oxirane (ethylene oxide) and methyloxirane (propylene oxide), which are also direct-acting alkylating agents, have been classified as carcinogenic</p>
<p style="text-align: center;">CALCIUM CARBONATE & TITANIUM DIOXIDE</p>	<p>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.</p>
<p style="text-align: center;">CALCIUM CARBONATE & DI-SEC-OCTYL PHTHALATE & TITANIUM DIOXIDE</p>	<p>The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.</p>
<p style="text-align: center;">DI-SEC-OCTYL PHTHALATE & TITANIUM DIOXIDE</p>	<p>WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.</p>
<p style="text-align: center;">Acute Toxicity</p>	<p style="text-align: center;">×</p>
<p style="text-align: center;">Skin Irritation/Corrosion</p>	<p style="text-align: center;">✓</p>
<p style="text-align: center;">Carcinogenicity</p>	<p style="text-align: center;">✓</p>
<p style="text-align: center;">Reproductivity</p>	<p style="text-align: center;">✓</p>

Serious Eye Damage/Irritation	✓	STOT - Single Exposure	✓
Respiratory or Skin sensitisation	✓	STOT - Repeated Exposure	✗
Mutagenicity	✓	Aspiration Hazard	✗

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

SECTION 12 Ecological information

Toxicity

Tremflex 25	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
polyurethane polymer	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
calcium carbonate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>14mg/l	2
	NOEC(ECx)	1h	Fish	4-320mg/l	4
	LC50	96h	Fish	>165200mg/L	4
di-sec-octyl phthalate	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1344h	Fish	<0.7-29.7	7
	EC50	72h	Algae or other aquatic plants	>130mg/l	1
	EC50	48h	Crustacea	>0.16mg/l	1
	NOEC(ECx)	1680h	Fish	0.007mg/l	1
	EC50	96h	Algae or other aquatic plants	>0.1mg/l	1
	LC50	96h	Fish	>0.16mg/l	2
ErC50	72h	Algae or other aquatic plants	>130mg/l	1	
titanium dioxide	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1008h	Fish	<1.1-9.6	7
	EC50	72h	Algae or other aquatic plants	3.75-7.58mg/l	4
	EC50	48h	Crustacea	1.9mg/l	2
	NOEC(ECx)	672h	Fish	>=0.004mg/L	2
	EC50	96h	Algae or other aquatic plants	179.05mg/l	2
LC50	96h	Fish	1.85-3.06mg/l	4	
gamma-glycidoxypropyltrimethoxysilane	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	96h	Fish	1.5mg/l	2
	EC50	72h	Algae or other aquatic plants	>420mg/l	2
	EC50	48h	Crustacea	473mg/l	2
	EC50	96h	Algae or other aquatic plants	250mg/l	2
LC50	96h	Fish	4.9mg/l	2	

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

Toxic to aquatic organisms.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
di-sec-octyl phthalate	HIGH (Half-life = 389 days)	LOW (Half-life = 1.21 days)
titanium dioxide	HIGH	HIGH
gamma-glycidoxypropyltrimethoxysilane	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
di-sec-octyl phthalate	HIGH (BCF = 24500)
titanium dioxide	LOW (BCF = 10)

Ingredient	Bioaccumulation
gamma-glycidoxypropyltrimethoxysilane	LOW (LogKOW = 0.5)

Mobility in soil

Ingredient	Mobility
di-sec-octyl phthalate	LOW (Log KOC = 165400)
titanium dioxide	LOW (Log KOC = 23.74)
gamma-glycidoxypropyltrimethoxysilane	LOW (Log KOC = 90.22)

SECTION 13 Disposal considerations**Waste treatment methods**

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority. ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Authority for disposal. ▶ Bury or incinerate residue at an approved site. ▶ Recycle containers if possible, or dispose of in an authorised landfill.
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SECTION 14 Transport information**Labels Required**

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

14.7. Maritime transport in bulk according to IMO instruments**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
polyurethane polymer	Not Applicable
calcium carbonate	Not Applicable
di-sec-octyl phthalate	Not Applicable
titanium dioxide	Not Applicable
gamma-glycidoxypropyltrimethoxysilane	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
polyurethane polymer	Not Applicable
calcium carbonate	Not Applicable
di-sec-octyl phthalate	Not Applicable
titanium dioxide	Not Applicable
gamma-glycidoxypropyltrimethoxysilane	Not Applicable

SECTION 15 Regulatory information**Safety, health and environmental regulations / legislation specific for the substance or mixture****polyurethane polymer is found on the following regulatory lists**

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

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)

di-sec-octyl phthalate 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

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

titanium dioxide is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

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

gamma-glycidoxypropyltrimethoxysilane is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (polyurethane polymer)
Canada - DSL	No (polyurethane polymer)
Canada - NDSL	No (polyurethane polymer; di-sec-octyl phthalate; gamma-glycidoxypropyltrimethoxysilane)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (polyurethane polymer)
Japan - ENCS	No (polyurethane polymer)
Korea - KECI	No (polyurethane polymer)
New Zealand - NZIoC	Yes
Philippines - PICCS	No (polyurethane polymer)
USA - TSCA	TSCA Inventory 'Active' substance(s) (calcium carbonate; di-sec-octyl phthalate; titanium dioxide; gamma-glycidoxypropyltrimethoxysilane); No (polyurethane polymer)
Taiwan - TCSI	Yes
Mexico - INSQ	No (polyurethane polymer; gamma-glycidoxypropyltrimethoxysilane)
Vietnam - NCI	Yes
Russia - FBEPH	Yes
UAE - Control List (Banned/Restricted Substances)	No (polyurethane polymer; calcium carbonate; di-sec-octyl phthalate; titanium dioxide; gamma-glycidoxypropyltrimethoxysilane)
Legend:	<i>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.</i>

SECTION 16 Other information

Revision Date	10/11/2025
Initial Date	10/11/2025

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
- ▶ MARPOL: International Convention for the Prevention of Pollution from Ships
- ▶ IMSBC: International Maritime Solid Bulk Cargoes Code
- ▶ IGC: International Gas Carrier Code
- ▶ IBC: International Bulk Chemical Code
- ▶ AIIC: Australian Inventory of Industrial Chemicals

- ▶ DSL: Domestic Substances List
- ▶ NDSL: Non-Domestic Substances List
- ▶ IECSC: Inventory of Existing Chemical Substance in China
- ▶ EINECS: European INventory of Existing Commercial chemical Substances
- ▶ ELINCS: European List of Notified Chemical Substances
- ▶ NLP: No-Longer Polymers
- ▶ ENCS: Existing and New Chemical Substances Inventory
- ▶ KECI: Korea Existing Chemicals Inventory
- ▶ NZIoC: New Zealand Inventory of Chemicals
- ▶ PICCS: Philippine Inventory of Chemicals and Chemical Substances
- ▶ TSCA: Toxic Substances Control Act
- ▶ TCSI: Taiwan Chemical Substance Inventory
- ▶ INSQ: Inventario Nacional de Sustancias Químicas
- ▶ NCI: National Chemical Inventory
- ▶ FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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