Antas FR50 Fireproof MS Sealant

Antas Sealants and Adhesives

Part Number: **66352FR**
Version No: **1.2.8.7**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

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

Product Identifier

Product name	Antas FR50 Fireproof MS Sealant
Chemical Name	Not Applicable
Synonyms	ATFR50
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	fire resistant sealing for ontrol joints

Details of the supplier of the safety data sheet

Registered company name	**Antas Sealants and Adhesives**
Address	21-23 Pavesi Street Smithfield NSW 2164 Australia
Telephone	+61 2 9157 0368
Fax	Not Available
Website	www.antas.com.au
Email	info@antas.com.au

Emergency telephone number

Association / Organisation	**Not Available**
Emergency telephone numbers	**+61 2 9157 0368**
Other emergency telephone numbers	**National Poison Information Centre 13 11 26**

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Poisons Schedule	Not Applicable
Classification [1]	Chronic Aquatic Hazard Category 2, Serious Eye Damage/Eye Irritation Category 1, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Skin Corrosion/Irritation Category 2, Carcinogenicity Category 1A, Reproductive Toxicity Category 1B, Germ cell mutagenicity Category 2
Legend:	*1. Classification by vendor; 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**

Hazard statement(s)

H411	Toxic to aquatic life with long lasting effects.
H318	Causes serious eye damage.
H335	May cause respiratory irritation.
H315	Causes skin irritation.
H350	May cause cancer.
H360FD	May damage fertility. May damage the unborn child.
H341	Suspected of causing genetic defects.

Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P391	Collect spillage.
P302+P352	IF ON SKIN: Wash with plenty of water.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

P405	Store locked up.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
26761-40-0	15-25	diisodecyl phthalate
471-34-1	35-45	calcium carbonate
13463-67-7	<3	titanium dioxide
1333-86-4	<3	carbon black
2768-02-7	<2	trimethoxyvinylsilane
6683-19-8	<2	pentaerythritol tetrabutylhydroxyhydrocinnamate
77-58-7	<1	dibutyltin dilaurate

Continued...

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

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ **IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY.** ▸ For advice, contact a Poisons Information Centre or a doctor. ▸ Urgent hospital treatment is likely to be needed. ▸ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. ▸ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist. ▸ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS. **Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:** ▸ **INDUCE** vomiting with fingers down the back of the throat, **ONLY IF CONSCIOUS**. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. **NOTE:** Wear a protective glove when inducing vomiting by mechanical means.

Indication of any immediate medical attention and special treatment needed

As in all cases of suspected poisoning, follow the ABCDEs of emergency medicine (airway, breathing, circulation, disability, exposure), then the ABCDEs of toxicology (antidotes, basics, change absorption, change distribution, change elimination).
For poisons (where specific treatment regime is absent):

BASIC TREATMENT

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

ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Treat seizures with diazepam.

- Proparacaine hydrochloride should be used to assist eye irrigation.

BRONSTEIN, A.C. and CURRANCE, P.L.
EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994
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

Advice for firefighters

Fire Fighting	‣ 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	Combustible. Will burn if ignited. Combustion products include: , carbon monoxide (CO) , carbon dioxide (CO2) , metal oxides , other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes. Heating calcium carbonate at high temperatures(825 C.) causes decomposition, releases carbon dioxide gas and leaves a residue of alkaline lime
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	Environmental hazard - contain spillage. ‣ 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	Environmental hazard - contain spillage. ‣ 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.

SECTION 7 Handling and storage

Precautions for safe handling

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

Conditions for safe storage, including any incompatibilities

Suitable container	‣ Metal can or drum ‣ Packaging as recommended by manufacturer. ‣ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Calcium carbonate: ‣ is incompatible with acids, ammonium salts, fluorine, germanium, lead diacetate, magnesium, mercurous chloride, silicon, silver nitrate, titanium. Contact with acid generates carbon dioxide gas, which may pressurise and then rupture closed containers Phthalates: ‣ react with strong acids, strong oxidisers, permanganates and nitrates ‣ attack some form of plastics ‣ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates. ‣ Avoid reaction with oxidising agents



+	x	+	x	+	+	+

X — *Must not be stored together*
0 — *May be stored together with specific preventions*
+ — *May be stored together*

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

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 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 Exposure Standards	carbon black	Carbon black	3 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	dibutyltin dilaurate	Tin, organic compounds (as Sn)	0.1 mg/m3	0.2 mg/m3	Not Available	(g) Some compounds in these groups are classified as carcinogenic or as sensitisers. Check individual classification details on the safety data sheet for information on classification.

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
calcium carbonate	45 mg/m3	210 mg/m3	1,300 mg/m3
titanium dioxide	30 mg/m3	330 mg/m3	2,000 mg/m3
carbon black	9 mg/m3	99 mg/m3	590 mg/m3
trimethoxyvinylsilane	9.5 ppm	100 ppm	120 ppm
dibutyltin dilaurate	1.1 mg/m3	8 mg/m3	48 mg/m3

Ingredient	Original IDLH	Revised IDLH
diisodecyl phthalate	Not Available	Not Available
calcium carbonate	Not Available	Not Available
titanium dioxide	5,000 mg/m3	Not Available
carbon black	1,750 mg/m3	Not Available
trimethoxyvinylsilane	Not Available	Not Available
pentaerythritol tetrabutylhydroxyhydrocinnamate	Not Available	Not Available
dibutyltin dilaurate	25 mg/m3	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
trimethoxyvinylsilane	E	≤ 0.1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

For calcium carbonate:
The TLV-TWA is thought to be protective against the significant risk of physical irritation associated with exposure.
Animals exposed by inhalation to 10 mg/m3 titanium dioxide show no significant fibrosis, possibly reversible tissue reaction. The architecture of lung air spaces remains intact.

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. An approved self contained breathing apparatus (SCBA) may be required in some situations.

	Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant. Type of Contaminant: / Air Speed: solvent, vapours, degreasing etc., evaporating from tank (in still air). / 0.25-0.5 m/s (50-100 f/min.) aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation) / 0.5-1 m/s (100-200 f/min.) direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion) / 1-2.5 m/s (200-500 f/min.) grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion). / 2.5-10 m/s (500-2000 f/min.) Within each range the appropriate value depends on: Lower end of the range / Upper end of the range 1: Room air currents minimal or favourable to capture / 1: Disturbing room air currents 2: Contaminants of low toxicity or of nuisance value only. / 2: Contaminants of high toxicity 3: Intermittent, low production. / 3: High production, heavy use 4: Large hood or large air mass in motion / 4: Small hood-local control only Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
Personal protection	
Eye and face protection	‣ Safety glasses with side shields. ‣ Chemical goggles. ‣ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	‣ Wear chemical protective gloves, e.g. PVC. ‣ Wear safety footwear or safety gumboots, e.g. Rubber
Body protection	See Other protection below
Other protection	‣ Overalls. ‣ P.V.C apron. ‣ Barrier cream. ‣ Skin cleansing cream. ‣ Eye wash unit.

Respiratory protection

Particulate. (AS/NZS 1716 & 1715, EN 143:2000 & 149:001, ANSI Z88 or national equivalent)

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	P1 Air-line*	- -	PAPR-P1 -
up to 50 x ES	Air-line**	P2	PAPR-P2
up to 100 x ES	-	P3	-
		Air-line*	-
100+ x ES	-	Air-line**	PAPR-P3

* - Negative pressure demand ** - Continuous flow

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

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	The phthalates have a clear syrupy liquid consistency and show low water solubility, high oil solubility, and low volatility. The polar carboxyl group contributes little to the physical properties of the phthalates, except when R and R' are very small (such as ethyl or methyl groups). Phthalates are colourless, odourless liquids produced by reacting phthalic anhydride with an appropriate alcohol (usually 6- to 13-carbon). Phthalate esters are the dialkyl or alkyl aryl esters of phthalic acid (also called 1,2-benzenedicarboxylic acid). When added to plastics, phthalates allow the long polyvinyl molecules to slide against one another. Dark

Physical state	Non Slump Paste	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature**	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 Available
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Not Available	**pH as a solution (%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	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.
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. 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. 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.
Skin Contact	The material may accentuate any pre-existing dermatitis condition Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either ▸ 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. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	On the basis of epidemiological data, it has been concluded that prolonged inhalation of the material, in an occupational setting, may produce cancer in humans. 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. Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure. 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. 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. 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 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 add 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, monoethyhexyl 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 carbohdrate 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.
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.

Antas FR50 Fireproof MS Sealant	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

diisodecyl phthalate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2900 mg/kg[2]	Not Available
	Inhalation(Rat) LC50; >12.54 mg/l4h[2]	
	Oral(Rat) LD50; >15000 mg/kg[2]	

calcium carbonate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit): 0.75 mg/24h - SEVERE
	Inhalation(Rat) LC50; >3 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral(Rat) LD50; >2000 mg/kg[1]	Skin (rabbit): 500 mg/24h-moderate
		Skin: no adverse effect observed (not 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/l4h[1]	Skin (human): 0.3 mg /3D (int)-mild *
	Oral(Rat) LD50; >=2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
carbon black	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral(Rat) LD50; >8000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
trimethoxyvinylsilane	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 3249.12 mg/kg[2]	Eye (rabbit): 500 mg/24h - mild
	Inhalation(Rat) LC50; 2773 ppm4h[1]	Eye (rabbit): 500 mg/24h mild
	Oral(Rat) LD50; >300<2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
		Skin (rabbit): 500 mg/24h - mild
		Skin (rabbit): 500 mg/24h mild
		Skin: no adverse effect observed (not irritating)[1]
pentaerythritol tetrabutylhydroxyhydrocinnamate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >3160 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50; >1.95 mg/L4h[2]	Eye: non-irritating **
	Oral(Rat) LD50; >2000 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
		Skin: non-irritating **
dibutyltin dilaurate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit): 100 mg/24h -moderate
	Oral(Rat) LD50; >=33<=300 mg/kg[1]	Skin (rabbit): 500 mg/24h - mild
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*	

DIISODECYL PHTHALATE	for bis(2-propylheptyl)phthalate A substance thought to be comparable to bis(2-propylheptyl)phthalate is diisodecyl phthalate (syn: DIDP) **Acute toxicity:** Bis(2-propylheptyl)phthalate is of low acute oral, dermal and inhalation toxicity and is slightly irritating to eyes and skin. The result of the non-adjuvant skin sensitisation test provided for assessment was negative and additional information available in the EU report for DIDP indicates that the material has low sensitising potential. **Repeat dose toxicity** : Based on repeated dose studies using DIDP, the more complex analogue of the substance, the target organ in subacute and subchronic studies in rats is the liver, the effects observed being increased liver weight and changes in liver peroxisome proliferator enzyme activities. As the NOAELs derived are due to the latter, which is considered to be species-specific and of little relevance to humans, the NOAEL of 15 mg/kg/day from a 90-day dog study was used in the EU risk assessment. However, this study was considered to be of poor reliability. In the DIDP dietary study provided to NICNAS for assessment, the NOAEL was 39 mg/kg/day, based on liver effects and hypertrophy of the follicular epithelium of the thyroid glands. The effects observed in the repeated dose toxicity tests do not justify classification with R48 according to the Approved criteria. **Developmental toxicity:** An EU report concluded that DIDP was a developmental toxicant, based on a decrease in survival indices in two-generation studies; a NOAEL of 0.06% (33 mg/kg/day) was used in the risk assessment. Developmental toxicity: For developmental effects, NOAELs of 500 mg/kg/day, for skeletal variations, and 253 mg/kg/day, for body weight decrease in offspring, were used in the risk assessment. No fertility effects were observed in any studies. Overall, the effects observed were not severe enough to warrant classification against the EU criteria. Based on the absence of statistically significant effects in dams, the No Observed Adverse Effect Level (NOAEL) for maternal toxicity was established as 1000 mg/kg bw/day in this screening study. Based on the statistically significant and dose-related incidence of skeletal variations in foetuses, the NOAEL for

developmental toxicity is 400 mg/kg bw/day.
An expert panel of the US National Toxicology Program (NTP) concluded that there was sufficient evidence from the toxicology database to determine that DIDP can cause foetotoxicity after oral exposure. The NOAEL for developmental toxicity in the two prenatal developmental studies in rats was 40-100 mg/kg/day based on the effects on the developing skeletal system. In the two oral two-generation reproductive toxicity studies in rats, adverse effects on pup survival and growth were observed, the NOAELs being 38-44 mg/kg/day during pregnancy and 52-114 mg/kg/day during lactation.
However, the reproductive studies showed that DIDP had no effect on reproductive structure or function, and the top doses, 427-929 mg/kg/day for males and 508-927 mg/kg/day for females, were selected as the NOAELs
The developmental effects of several phthalates are exerted via alternations in testosterone-synthesising ability of the foetal testes. The mode of action of the testicular toxicity is via the monoester with the target cell in the testis being the Sertoli cell, although the precise biochemical interaction has yet to be identified.
Attention has also been focused on the endocrine-active effects of phthalates including interactions with both oestrogen and androgen action. The experimental results indicate that only selected phthalates esters exhibit weak oestrogen receptor-mediated activity in some in vitro assays at high concentrations (IPCS, 2002). No overt effect related to endocrine disruption of the reproductive system was observed in any of the studies considered in the EU report on DIDP.
Genotoxicity: DIDP is not mutagenic in vitro in bacterial mutation assays with and without metabolic activation, and is negative in a mouse lymphoma assay. It is not clastogenic in a mouse micronucleus assay in vivo either. DIDP is not genotoxic.
Carcinogenicity: No carcinogenicity long term study is available for DIDP, but an increase in incidence of hepatocellular tumours in rats related to peroxisome proliferation might be anticipated. An increased incidence in tumour liver cells was observed in rats treated with DEHP and di-isononyl phthalate (DINP). However, the carcinogenic effects of peroxisome proliferators are generally considered to be specific to rodent species, while humans are essentially non-responsive or refractory
High Molecular Weight Phthalate Esters (HMWPEs) Category as defined by the Phthalate Esters Panel HPV Testing Group (2001) and OECD (2004). The HMWPE group includes chemically similar substances produced from alcohols having backbone carbon lengths of >= 7. Due to their similar chemical structure, category members are generally similar with respect to physicochemical, biological and toxicological properties or display an expected trend. Thus, read-across for toxicity endpoints is an appropriate approach to characterise selected endpoints for members of this category.
In some cases the substances have ester side group constituents that span two subcategories (i.e., transitional and high molecular weight constituents). If the level of C4 to C6 constituents in the substance exceeded 10%,the substance was conservatively placed in the transitional subcategory.
High molecular weight phthalates are used nearly exclusively as plasticisers of PVC.
They are very poorly soluble in water, and have very low vapor pressure. The extant database demonstrates that these substances have few biological effects. A notable exception to this generalisation is that hepatocarcinogenicity has been observed for diisononyl phthalate (DINP). The hepatocarcinogencity effects of DINP are by a mechanism (peroxisomal proliferation) to which rodents are particularly sensitive. However, it does not appear to be relevant to humans.
The high molecular weight phthalates all demonstrate minimal acute toxicity, are not genotoxic, exhibit some liver and kidney effects at high doses, and are negative for reproductive and developmental effects. Further, the available data indicate that the toxicological activity of these molecules diminishes with increasing molecular weight.
Studies on HMWPEs indicate that they are rapidly metabolised in the gastrointestinal tract to the corresponding monoester, absorbed and excreted primarily in the urine.
Acute toxicity: The available data on phthalates spanning the carbon range from C8-C13 indicate that phthalate esters in the high molecular weight subcategory are not toxic by acute oral and dermal administration; LD50 values of all substances tested exceed the maximum amounts which can be administered to the animals. There are fewer data available on inhalation toxicity; only di-iso-nonyl phthalate (DINP) and di-iso-decyl phthalate (DIDP) have been tested. However, the phthalates in the high molecular weight subcategory have extremely low vapor pressures, and exposure by inhalation at potentially hazardous levels is not anticipated.
Repeat dose toxicity. Several substances ranging from C8-C11 have been tested for repeated dose toxicity in studies ranging from 21 days to two years. Ditridecyl phthalate (CAS 119-06-2) has been studied by the Japan Ministry of Health and Welfare (unpublished report) and data for this substance is used as read-across data for DTDP*. In addition results from repeat dose studies examining DINP (CAS 685 15-48-0) and DIDP (CAS 68515-49-1) are used as read across for the di C9-C11 phthalates (CAS 68515-43-5). The principal effects found are those associated with peroxisomal proliferation, including liver enlargement and induction of peroxisomal enzymes. As shown for example in a comparative study of liver effects, the strongest inducers of peroxisomal proliferation were DEHP, DINP, and DIDP with substances of shorter and longer ester side chains (e.g., 610P*, 711P*, and diundecyl phthalate - DUP) showing less pronounced effects. Thus, it is reasonable to conclude that other members of this subcategory would show effects similar to but not more pronounced than those associated with DINP and 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 (PPARa;), and that levels of PPARa are much higher in rodents than humans . Thus, one would expect humans to be substantially less responsive than rodents to peroxisome proliferating agents. Empirical evidence supporting this postulation is provided by studies in primates in which repeated administration of DEHP and DINP had no effects on liver, kidney or testicular parameters.
In this regard it should also be noted that kidney enlargement is also commonly observed but normally without any pathological changes. There is a component of the kidney changes which is also PPARa-related. It has

	also been shown that in male rats, DINP induces an alpha 2u-globulin nephropathy which is male rat- specific but without relevance to humans. Thus, as was true for the liver changes, the relevance of the kidney changes to human health is also questionable Finally, some of the lower molecular weight phthalates can induce testicular atrophy when administered to juvenile rats at high levels. However, the higher molecular weight phthalates including di-n-octyl phthalate (DnOP), DINP, DIDP, 610P, and 71 1P do not induce testicular atrophy. Further, the testis was not a target organ for DINP in either marmosets or cynomolgus monkeys . Thus, testicular atrophy is not an effect associated with phthalates in the high molecular weight subcategory **Reproductive toxicity:** Reproductive toxicity tests in rats have been carried out with DINP, DIDP a linear C7-C9 phthalate (CAS 68515-41-3), a linear C9-C11 phthalate, and ditridecyl phthalate (Japan Ministry of Health and Welfare, unpublished report). None of these affected fertility or profoundly affected male reproductive development. A slight decrease in offspring viability was reported for both DIDP and ditridecyl phthalate at levels associated with maternal effects. DnOP was tested for effects on fertility in a continuous breeding protocol in mice, and, like the other members of this subcategory, did not reduce fertility. Thus, it can be concluded that the subcategory of high molecular weight phthalates do not affect fertility. **Developmental toxicity:** Developmental toxicity tests in rats have been carried out with DINP; DIDP; C7-9 phthalate (CAS 68515-41-3); C9-11 phthalate (CAS 68515-43-5); and ditridecyl phthalate (CAS 119-06-2). None of the substances tested affected litter size, foetal survival or bodyweight, and none produced teratogenic effects. Increased frequencies of developmental variants including dilated renal pelvis, and supernumerary lumbar and cervical ribs were found at levels associated with maternal effects. The toxicological significance of these developmental variants is unclear. DnOP was not teratogenic in mice when tested at very high levels. Thus, it can be concluded that this subcategory of high molecular weight phthalates do not produce profound developmental effects in rodents **Genotoxicity:** The majority of the substances in the subcategory of high molecular weight phthalates have been tested for genetic activity in the Salmonella assay, and all were inactive. One large program covering many of these substances was carried out by the National Institute of Environmental Health Sciences. Similarly, a range of substances covering the majority of the carbon numbers in this subcategory were found to be inactive in mouse lymphoma tests Chromosomal Aberrations. Two representative members of the subcategory of high molecular weight phthalates (DINP and DIDP) have been tested for chromosomal mutation in the mouse micronucleus test, and both were inactive. Ditridecyl phthalate (CAS 119-06-2) induced neither structural chromosomal aberrations nor polyploidy in CHL cells up to the limit concentration of 4.75 mg/ rnl, in the absence or presence of an exogenous metabolic activation system (Japan Ministry of Health and Welfare, unpublished report). Further, all of the low molecular weight and transitional phthalates that have been tested were inactive. *610P - mixed decyl, hexyl and octyl esters (CAS Rn: 68648-93-1) *711P - C7,C11, branched and linear esters (CAS Rn: 111381-90-9) * DTDP - di-C11-14, C13 rich ester (CAS 68515-47-9) Effects, Chronic Exposure General liver damage reported in rodents and dogs fed DIDP; not a route of industrial exposure Sensitising not a sensitiser in humans or animals; very few reports of human sensitisation usually associated with monomers or oligomers in incompletely cured polymer, not the plasticiser Carcinogen/Tumorigen not considered a tumorigen or a carcinogen in humans or animals Reproductive Effect rodent fetotoxicity on prolonged feeding; no known effect in humans or animals Mutagen no known effect on humans or animals
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. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
TITANIUM DIOXIDE	* IUCLID The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
CARBON BLACK	Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported
TRIMETHOXYVINYLSILANE	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. The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. Manufacturers Data:
PENTAERYTHRITOL TETRABUTYLHYDROXYHYDROCINNAMATE	For hindered phenols: Available data shows that acute toxicity of these substances is low. **Mutagenicity.** Data from bacterial reverse mutation assays and *in vitro* and *in vivo* chromosome aberration studies were reviewed. All assays, with and without metabolic activation, were negative. The weight of evidence for mutagenic potential for this category indicates these substances are not mutagenic. **In Vitro Chromosome Aberration Studies.** In vitro chromosome aberration studies are available for several members All except 2,6-di-tert-butyl-p-cresol were negative **In Vivo Chromosome Aberration Studies.** In vivo studies evaluating chromosome damage are available for six of the hindered phenols. All in vivo evaluations were negative. **Repeated Dose Toxicity**. Repeated dose toxicity data of approximately three months (90-day, 12- and 13-week) are available for some of the substances in this group. The liver was the target organ in rats for almost all of the substances with subchronic toxicity data in that species. Other target organs included thyroid and kidney and mesenteric lymph nodes. NOAELs in rats ranged from 100 ppm (approximately 5 mg/kg/day) to 10,000 ppm (500 mg/kg/day **Carcinogenicity:** Data is available for 2,6-di-tert-butyl-p-cresol (128-37-0); and 4,4'-thiobis-6-(t-butyl-m-cresol) (96-69-5). Liver adenomas were reported for 2,6-di-tert-butyl-p-cresol (128-37-0) and a NOAEL was established for the study at 25 mg/kg/day. 4,4'-Thiobis-6-(t-butyl-m-cresol) (96-69-5) was not carcinogenic in rats or mice, but the kidney was identified as a target organ in female rats Non-sensitising in Guinea pig skin assay ** * CG ** Clariant
Antas FR50 Fireproof MS Sealant & TITANIUM DIOXIDE & DIBUTYLTIN DILAURATE	Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.
Antas FR50 Fireproof MS Sealant & CALCIUM CARBONATE & TITANIUM DIOXIDE & TRIMETHOXYVINYLSILANE	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. 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. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
Antas FR50 Fireproof MS Sealant & TITANIUM DIOXIDE	For titanium dioxide: Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin. Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica. No data were available on genotoxic effects in titanium dioxide-exposed humans. Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts. Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences

	are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium. Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light. **Animal carcinogenicity data** Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats. In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative. Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice. In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative.
Antas FR50 Fireproof MS Sealant & DIISODECYL PHTHALATE	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.
TITANIUM DIOXIDE & CARBON BLACK	No significant acute toxicological data identified in literature search. **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
TITANIUM DIOXIDE & TRIMETHOXYVINYLSILANE	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

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

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

SECTION 12 Ecological information

Toxicity

Antas FR50 Fireproof MS Sealant	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available

diisodecyl phthalate	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	BCF	1344h	Fish	<*3.6	7
	EC50	72h	Algae or other aquatic plants	>500mg/l	1
	LC50	96h	Fish	>0.46mg/L	4
	NOEC(ECx)	48h	Crustacea	0.026mg/l	4

	Endpoint	Test Duration (hr)	Species	Value	Source
calcium carbonate	NOEC(ECx)	6h	Fish	4-320mg/l	4
	EC50	72h	Algae or other aquatic plants	>14mg/l	2
	LC50	96h	Fish	>165200mg/L	4

	Endpoint	Test Duration (hr)	Species	Value	Source
titanium dioxide	EC50	72h	Algae or other aquatic plants	3.75-7.58mg/l	4
	BCF	1008h	Fish	<1.1-9.6	7
	EC50	48h	Crustacea	1.9mg/l	2
	LC50	96h	Fish	1.85-3.06mg/l	4
	NOEC(ECx)	504h	Crustacea	0.02mg/l	4
	EC50	96h	Algae or other aquatic plants	179.05mg/l	2

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

	Endpoint	Test Duration (hr)	Species	Value	Source
trimethoxyvinylsilane	EC50	72h	Algae or other aquatic plants	>89mg/l	2
	LC50	96h	Fish	>92.2mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	NOEC(ECx)	48h	Crustacea	1mg/l	2

	Endpoint	Test Duration (hr)	Species	Value	Source
pentaerythritol tetrabutylhydroxyhydrocinnamate	BCF	1008h	Fish	<0.2	7
	EC50(ECx)	24h	Crustacea	>86mg/l	1
	EC50	72h	Algae or other aquatic plants	>100mg/l	1
	LC50	96h	Fish	>100mg/l	2

	Endpoint	Test Duration (hr)	Species	Value	Source
dibutyltin dilaurate	LC50	96h	Fish	21.2mg/l	2
	EC50	48h	Crustacea	1.7-3.4mg/l	2
	EC10(ECx)	96h	Algae or other aquatic plants	>0.5mg/l	4
	BCF	1344h	Fish	2.2-40	7
	EC50	72h	Algae or other aquatic plants	>1mg/l	2

Legend:	*Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.
Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
for phthalate esters:
Phthalates are easily released into the environment. In general, they do not persist due to rapid biodegradation, photodegradation, and anaerobic degradation. Outdoor air concentrations are higher in urban and suburban areas than in rural and remote areas. They also pose no acute toxicity. In general, children's exposure to phthalates is greater than that of adults
Environmental fate;
Under aerobic and anaerobic conditions, studies reveal that many phthalate esters are degraded by a wide range of bacteria and actinomycetes. Standardized aerobic biodegradation tests with sewage sludge inocula show that within 28 days approximately 50% ultimate degradation occurs. Biodegradation is, therefore, expected to be the dominant pathway in surface soils and sediments. In the atmosphere, photodegradation via free radical attack is the anticipated dominant pathway. The half-life of many phthalate esters is ca. 1 day in the air, from < 1 day to 2 weeks in surface and marine waters, and from < 1 week to several months in soils.

Phthalates are high molecular weight chemicals, and are not expected to partition significantly to air. However for the minor amount that may partition to air, modelled predictions indicate that they would be rapidly oxidised: with a predicted atmospheric oxidation half-life of around 0.52 days . They are expected to react appreciably with other photo oxidative species in the atmosphere, such as O3. Therefore, it is expected that reactions with hydroxyl radicals will be the most important fate process in the atmosphere for phthalates.
Bioaccumulation of phthalate esters in the aquatic and terrestrial food chain is limited by biotransformation.
Most phthalates have experimental bioaccumulation factor (BCFs) and bioconcentration factor (BAFs) below 5000 L/kg, as they are readily metabolised by fish
A study of 18 commercial phthalate esters with alkyl chains ranging from one to 13 carbons found an eight order of magnitude increase in octanol-water coefficients (Kow) and a four order of magnitude decrease in vapor pressure with increasing length. This increase in Kow and decrease in vapor pressure results in increased partitioning of the phthalate esters to suspended solids, soils, sediments, and aerosols
The phthalate esters are distributed throughout the environment ubiquitously. They are found complexed with fulvic acid components of the humic substances in soil and marine and estuarine waters. Fulvic acid appears to act as a solubiliser for the otherwise insoluble ester and serves to mediate its transport and mobilisation in water or immobilisation in soil. Phthalate esters have been found in open ocean environments, in deep sea jelly fish, Atlantic herring and in mackerel. Phthalic ester plasticisers are clearly recognised as general contaminants of almost every soil and water ecosystem. In general they have low acute toxicity but the weight of evidence supporting their carcinogenicity is substantial. Other subtle chronic effects have also been reported. As little as 4 ug/ml in culture medium is lethal to chick embryo heart cells. This concentration is similar to that reached in human blood stored in vinyl plastic bags for as little as one day. As phthalates are present in drinking water and food, concerns have been raised about their long term effects on humans.
Ecotoxicity:
Some phthalates (notably di-2-ethylhexyl phthalate and dibutyl phthalate) may be detrimental to the reproduction of the water flea (Daphnia magna), zebra fish and guppies
While phthalates may have very low true water solubilities, they possess the ability to form suspensions which may cause adverse effects through physical contact with *Daphnia* at very low concentrations.
Available toxicity and water solubility information suggest that the high molecular weight phthalates, form these suspensions and are able to elicit chronic toxic effects at concentrations of approximately 0.05 mg/L . Therefore, these substances are considered to have the potential to harm aquatic organisms at relatively low concentrations
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
diisodecyl phthalate	HIGH	HIGH
titanium dioxide	HIGH	HIGH
trimethoxyvinylsilane	HIGH	HIGH
pentaerythritol tetrabutylhydroxyhydrocinnamate	HIGH	HIGH
dibutyltin dilaurate	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
diisodecyl phthalate	HIGH (BCF = 3500)
titanium dioxide	LOW (BCF = 10)
trimethoxyvinylsilane	LOW (LogKOW = -0.3169)
pentaerythritol tetrabutylhydroxyhydrocinnamate	LOW (BCF = 2.3)
dibutyltin dilaurate	LOW (BCF = 110)

Mobility in soil

Ingredient	Mobility
diisodecyl phthalate	LOW (KOC = 1589000)
titanium dioxide	LOW (KOC = 23.74)
trimethoxyvinylsilane	LOW (KOC = 757.6)
dibutyltin dilaurate	LOW (KOC = 64610000)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.

	▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

Marine Pollutant	
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

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

Not Applicable

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

Product name	Group
diisodecyl phthalate	Not Available
calcium carbonate	Not Available
titanium dioxide	Not Available
carbon black	Not Available
trimethoxyvinylsilane	Not Available
pentaerythritol tetrabutylhydroxyhydrocinnamate	Not Available
dibutyltin dilaurate	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
diisodecyl phthalate	Not Available
calcium carbonate	Not Available
titanium dioxide	Not Available
carbon black	Not Available
trimethoxyvinylsilane	Not Available
pentaerythritol tetrabutylhydroxyhydrocinnamate	Not Available
dibutyltin dilaurate	Not Available

SECTION 15 Regulatory information

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

diisodecyl phthalate is found on the following regulatory lists

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

calcium carbonate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

titanium dioxide is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
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)

carbon black is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
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)

trimethoxyvinylsilane is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

pentaerythritol tetrabutylhydroxyhydrocinnamate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

dibutyltin dilaurate 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 7
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (diisodecyl phthalate; carbon black; trimethoxyvinylsilane; pentaerythritol tetrabutylhydroxyhydrocinnamate; dibutyltin dilaurate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (trimethoxyvinylsilane)
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 Other information

Revision Date	30/06/2021
Initial Date	30/06/2021

SDS Version Summary

Version	Date of Update	Sections Updated
0.0.2.1	26/04/2021	Regulation Change
0.0.3.1	03/05/2021	Regulation Change

Version	Date of Update	Sections Updated
0.0.4.1	06/05/2021	Regulation Change
0.0.5.1	10/05/2021	Regulation Change
0.0.5.2	30/05/2021	Template Change
0.0.5.3	04/06/2021	Template Change
0.0.5.4	05/06/2021	Template Change
0.0.6.4	07/06/2021	Regulation Change
0.0.6.5	09/06/2021	Template Change
0.0.6.6	11/06/2021	Template Change
0.0.6.7	15/06/2021	Template Change
0.0.7.7	17/06/2021	Regulation Change
0.0.8.7	21/06/2021	Regulation Change

Other information

Ingredients with multiple cas numbers

Name	CAS No
diisodecyl phthalate	26761-40-0, 68515-49-1
calcium carbonate	471-34-1, 13397-26-7, 15634-14-7, 1317-65-3, 72608-12-9, 878759-26-3, 63660-97-9, 459411-10-0, 198352-33-9, 146358-95-4, 1357-85-3
titanium dioxide	13463-67-7, 1317-70-0, 1317-80-2, 12188-41-9, 1309-63-3, 100292-32-8, 101239-53-6, 116788-85-3, 12000-59-8, 12701-76-7, 12767-65-6, 12789-63-8, 1344-29-2, 185323-71-1, 185828-91-5, 188357-76-8, 188357-79-1, 195740-11-5, 221548-98-7, 224963-00-2, 246178-32-5, 252962-41-7, 37230-92-5, 37230-94-7, 37230-95-8, 37230-96-9, 39320-58-6, 39360-64-0, 39379-02-7, 416845-43-7, 494848-07-6, 494848-23-6, 494851-77-3, 494851-98-8, 55068-84-3, 55068-85-4, 552316-51-5, 62338-64-1, 767341-00-4, 97929-50-5, 98084-96-9, 51745-87-0, 12035-95-9, 52624-13-2
pentaerythritol tetrabutylhydroxyhydrocinnamate	6683-19-8, 5106-16-1, 12634-41-2, 60005-82-5, 67894-72-8, 68882-58-6, 70695-00-0, 98584-37-3, 103843-13-6, 127337-64-8, 137500-49-3, 145526-73-4, 154508-64-2

Classification of the preparation and its individual components has drawn on official and authoritative sources 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
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