

# Ardex Quickbond ARDEX (Ardex Australia)

Chemwatch: 5696-91

Version No: 2.1
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

### Chemwatch Hazard Alert Code: 3

Issue Date: **26/08/2024** Print Date: **27/09/2024** L.GHS.AUS.EN.E

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

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Product name	Ardex Quickbond
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

### Details of the manufacturer or supplier of the safety data sheet

Registered company name	ARDEX (Ardex Australia)	
Address	2 Buda Way Kemps Creek NSW 2147 Australia	
Telephone	1300 788 780	
Fax	1300 780 102	
Website	www.ardexaustralia.com	
Email	technical.services@ardexaustralia.com	

### Emergency telephone number

Association / Organisation	ARDEX (ARDEX Australia)	
Emergency telephone numbers	1800 224 070 (Mon-Fri, 9am-5pm)	
Other emergency telephone numbers	Not Available	

### **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]	Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Germ Cell Mutagenicity Category 2, Carcinogenicity Category 1A, Specific Target Organ Toxicity - Repeated Exposure 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)







Signal word

Danger

### Hazard statement(s)

` '	
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.

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H318	Causes serious eye damage.
H335	May cause respiratory irritation.
H341	Suspected of causing genetic defects.
H350	May cause cancer.
H373	May cause damage to organs through prolonged or repeated exposure.

### Precautionary statement(s) Prevention

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P272	Contaminated work clothing should not be allowed out of the workplace.
P264 Wash all exposed external body areas thoroughly after handling.	
P280 Wear protective gloves, protective clothing, eye protection and face protection.	
P271	Use only outdoors or in a well-ventilated area.
P260	Do not breathe dust/fume.
P201	Obtain special instructions before use.

### Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.		
P308+P313	IF exposed or concerned: Get medical advice/ attention.		
P310	Immediately call a POISON CENTER/doctor/physician/first aider.		
P302+P352	IF ON SKIN: Wash with plenty of water.		
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.		
P362+P364	Take off contaminated clothing and wash it before reuse.		
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.		

### 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
14808-60-7.	30-60	graded sand
65997-15-1	10-30	portland cement
12004-14-7	10-30	calcium aluminate sulfate
7778-18-9	1-10	<u>calcium sulfate</u>
13463-67-7	<3	titanium dioxide
14808-60-7	<0.3	silica crystalline - quartz
Not Available	1-10	Ingredients determined not to be hazardous
Legend:	Classified by Chemwatch; 2. Classification drawn from C&L	Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4.

### **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. Immediately hold eyelids apart and flush the eye continuously with running water. Immediately hold eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. Transport to hospital or doctor without delay. Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs:  Immediately flush body and clothes with large amounts of water, using safety shower if available.  Quickly remove all contaminated clothing, including footwear.  Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre.  Transport to hospital, or doctor.
Inhalation	<ul> <li>If fumes or combustion products are inhaled remove from contaminated area.</li> <li>Lay patient down. Keep warm and rested.</li> <li>Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.</li> <li>Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.</li> <li>Transport to hospital, or doctor, without delay.</li> </ul>
Ingestion	► If swallowed do <b>NOT</b> induce vomiting.

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- 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

For acute or short-term repeated exposures to highly alkaline materials:

- Respiratory stress is uncommon but present occasionally because of soft tissue edema.
- Unless endotracheal intubation can be accomplished under direct vision, cricothyroidotomy or tracheotomy may be necessary.
- Oxygen is given as indicated.
- The presence of shock suggests perforation and mandates an intravenous line and fluid administration.
- Damage due to alkaline corrosives occurs by liquefaction necrosis whereby the saponification of fats and solubilisation of proteins allow deep penetration into the tissue. Alkalis continue to cause damage after exposure.

### INGESTION:

▶ Milk and water are the preferred diluents

No more than 2 glasses of water should be given to an adult.

- Neutralising agents should never be given since exothermic heat reaction may compound injury.
   \* Catharsis and emesis are absolutely contra-indicated.
- \* Activated charcoal does not absorb alkali.
- \* Gastric lavage should not be used.

Supportive care involves the following:

- Withhold oral feedings initially.
- If endoscopy confirms transmucosal injury start steroids only within the first 48 hours.
- Carefully evaluate the amount of tissue necrosis before assessing the need for surgical intervention.
- Patients should be instructed to seek medical attention whenever they develop difficulty in swallowing (dysphagia).

### SKIN AND EYE:

Injury should be irrigated for 20-30 minutes.

Eye injuries require saline. [Ellenhorn & Barceloux: Medical Toxicology]

### **SECTION 5 Firefighting measures**

### Extinguishing media

Advice for firefighters

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

### Special hazards arising from the substrate or mixture

Fire Incompatibility	<ul> <li>Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result</li> </ul>
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- Alert Fire Brigade and tell them location and nature of hazard.
- Wear breathing apparatus plus protective gloves in the event of a fire.
- Prevent, by any means available, spillage from entering drains or water courses.
- Use fire fighting procedures suitable for surrounding area. Fire Fighting
  - ▶ 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.

### ▶ Solid which exhibits difficult combustion or is difficult to ignite. Avoid generating dust, particularly clouds of dust in a confined or unventilated space as dusts may form an explosive mixture with air, and any source of ignition, i.e. flame or spark, will cause fire or explosion.

- Dust clouds generated by the fine grinding of the solid are a particular hazard; accumulations of fine dust (420 micron or less) may burn
- rapidly and fiercely if ignited; once initiated larger particles up to 1400 microns diameter will contribute to the propagation of an explosion. A dust explosion may release large quantities of gaseous products; this in turn creates a subsequent pressure rise of explosive force
- capable of damaging plant and buildings and injuring people.
- ▶ Usually the initial or primary explosion takes place in a confined space such as plant or machinery, and can be of sufficient force to damage or rupture the plant. If the shock wave from the primary explosion enters the surrounding area, it will disturb any settled dust layers, forming a second dust cloud, and often initiate a much larger secondary explosion. All large scale explosions have resulted from chain reactions of this type.
- Dry dust can also be charged electrostatically by turbulence, pneumatic transport, pouring, in exhaust ducts and during transport.
- Build-up of electrostatic charge may be prevented by bonding and grounding.
- Powder handling equipment such as dust collectors, dryers and mills may require additional protection measures such as explosion venting.
- All movable parts coming in contact with this material should have a speed of less than 1-metre/sec. Decomposition may produce toxic fumes of:

sulfur oxides (SOx)

silicon dioxide (SiO2)

metal oxides

May emit poisonous fumes. May emit corrosive fumes.

HAZCHEM

Not Applicable

### **SECTION 6 Accidental release measures**

Fire/Explosion Hazard

### Personal precautions, protective equipment and emergency procedures

See section 8

### **Environmental precautions**

See section 12

### Methods and material for containment and cleaning up

Minor Spills

Clean up waste regularly and abnormal spills immediately.

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	<ul> <li>Avoid breathing dust and contact with skin and eyes.</li> <li>Wear protective clothing, gloves, safety glasses and dust respirator.</li> <li>Use dry clean up procedures and avoid generating dust.</li> <li>Vacuum up or sweep up. NOTE: Vacuum cleaner must be fitted with an exhaust micro filter (H-Class HEPA type) (consider explosion-proof machines designed to be grounded during storage and use). H-Class HEPA filtered industrial vacuum cleaners should NOT be used on wet materials or surfaces.</li> <li>Dampen with water to prevent dusting before sweeping.</li> <li>Place in suitable containers for disposal.</li> </ul>
Major Spills	Moderate hazard.  CAUTION: Advise personnel in area.  Alert Emergency Services and tell them location and nature of hazard.  Control personal contact by wearing protective clothing.  Prevent, by any means available, spillage from entering drains or water courses.  Recover product wherever possible.  IF DRY: Use dry clean up procedures and avoid generating dust. Collect residues and place in sealed plastic bags or other containers for disposal. IF WET: Vacuum/shovel up and place in labelled containers for disposal.  ALWAYS: Wash area down with large amounts of water and prevent runoff into drains.  If contamination of drains or waterways occurs, advise Emergency Services.

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

### Conditions for safe storage, including any incompatibilities

Suitable container	<ul> <li>Polyethylene or polypropylene container.</li> <li>Check all containers are clearly labelled and free from leaks.</li> </ul>
Storage incompatibility	<ul> <li>Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.</li> <li>Avoid contact with copper, aluminium and their alloys.</li> <li>Avoid reaction with oxidising agents</li> </ul>

### SECTION 8 Exposure controls / personal protection

### **Control parameters**

Occupational Exposure Limits (OEL)

INGREDIENT DATA

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	graded sand	Quartz (respirable dust)	0.05 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	graded sand	Silica - Crystalline: Quartz (respirable dust)	0.05 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	portland cement	Portland cement	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	calcium sulfate	Calcium sulphate	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	silica crystalline - quartz	Quartz (respirable dust)	0.05 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silica crystalline - quartz	Silica - Crystalline: Quartz (respirable dust)	0.05 mg/m3	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
graded sand	25 mg/m3 / 50 mg/m3	Not Available
portland cement	5,000 mg/m3	Not Available
calcium aluminate sulfate	Not Available	Not Available
calcium sulfate	Not Available	Not Available
titanium dioxide	5,000 mg/m3	Not Available
silica crystalline - quartz	25 mg/m3 / 50 mg/m3	Not Available

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Exposure controls	
Appropriate engineering controls	None under normal operating conditions.
Individual protection measures, such as personal protective equipment	
Eye and face protection	<ul> <li>Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure.</li> <li>Chemical goggles. Whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted. [AS/NZS 1337.1, EN166 or national equivalent]</li> <li>Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection.</li> <li>Alternatively a gas mask may replace splash goggles and face shields.</li> <li>Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].</li> </ul>
Skin protection	See Hand protection below
Hands/feet protection	NOTE:  The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.  Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.  The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.  The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.  Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.  Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:  • frequency and duration of contact,  • chemical resistance of glove material

- · chemical resistance of glove material,
- glove thickness and
- dexterity

- Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).

  When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
- · Contaminated gloves should be replaced.

As defined in ASTM F-739-96 in any application, gloves are rated as:

- · Excellent when breakthrough time > 480 min
- Good when breakthrough time > 20 min
   Fair when breakthrough time < 20 min</li>
   Poor when glove material degrades

For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.

It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also

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be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended Experience indicates that the following polymers are suitable as glove materials for protection against undissolved, dry solids, where abrasive particles are not present. polychloroprene. nitrile rubber. butyl rubber. fluorocaoutchouc. polyvinyl chloride. Gloves should be examined for wear and/ or degradation constantly. **Body protection** See Other protection below Overalls. ▶ P.V.C apron Other protection Barrier cream. Skin cleansing cream.

### Respiratory protection

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

Eye wash unit.

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)

- · Respirators may be necessary when engineering and administrative controls do not adequately prevent exposures.
- The decision to use respiratory protection should be based on professional judgment that takes into account toxicity information, exposure measurement data, and frequency and likelihood of the worker's exposure ensure users are not subject to high thermal loads which may result in heat stress or distress due to personal protective equipment (powered, positive flow, full face apparatus may be an option).
- · Published occupational exposure limits, where they exist, will assist in determining the adequacy of the selected respiratory protection. These may be government mandated or vendor recommended.
- · Certified respirators will be useful for protecting workers from inhalation of particulates when properly selected and fit tested as part of a complete respiratory protection program.
- · Where protection from nuisance levels of dusts are desired, use type N95 (US) or type P1 (EN143) dust masks. Use respirators and components tested and approved under appropriate government standards such as NIOSH (US) or CEN (EU)
- $\cdot$  Use approved positive flow mask if significant quantities of dust becomes airborne.
- · Try to avoid creating dust conditions.

Where significant concentrations of the material are likely to enter the breathing zone, a Class P3 respirator may be required.

Class P3 particulate filters are used for protection against highly toxic or highly irritant particulates

Filtration rate: Filters at least 99.95% of airborne particles

Suitable for:

- · Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.
- Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.
- · Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS
- · Highly toxic particles e.g. Organophosphate Insecticides, Radionuclides, Asbestos

Note: P3 Rating can only be achieved when used with a Full Face Respirator or Powered Air-Purifying Respirator (PAPR). If used with any other respirator, it will only provide filtration protection up to a P2 rating.

### **SECTION 9 Physical and chemical properties**

### Information on basic physical and chemical properties

Appearance	Fine off-white powder; partly soluble in water.		
Physical state	Divided Solid	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 Applicable
pH (as supplied)	Not Applicable	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Applicable
Initial boiling point and boiling range (°C)	Not Applicable	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available

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Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Applicable
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Applicable	Gas group	Not Available
Solubility in water	Partly miscible	pH as a solution (1%)	11
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**

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

### **SECTION 11 Toxicological information**

### Information on toxicological effects

	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
nhaled	derived from the vascular system.
maicu	Persons with impaired respiratory function, airway diseases and conditions such as emphysema or chronic bronchitis, may incur further
	disability if excessive concentrations of particulate are inhaled.

If prior damage to the circulatory or nervous systems has occurred or if kidney damage has been sustained, proper screenings should be

conducted on individuals who may be exposed to further risk if handling and use of the material result in excessive exposures. Effects on lungs are significantly enhanced in the presence of respirable particles. Overexposure to respirable dust may produce wheezing, coughing and breathing difficulties leading to or symptomatic of impaired respiratory function.

### Ingestion

Inl

Not normally a hazard due to the physical form of product. The material is a physical irritant to the gastro-intestinal tract

## Skin Contact

Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.

The material may accentuate any pre-existing dermatitis condition
Skin contact may result in severe irritation particularly to broken skin. Ulceration known as "chrome ulcers" may develop. Chrome ulcers and skin cancer are significantly related.

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.

### 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

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 Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.

Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.

Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.

Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. 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.

Cement contact dermatitis (CCD) may occur when contact shows an allergic response, which may progress to sensitisation. Sensitisation is due to soluble chromates (chromate compounds) present in trace amounts in some cements and cement products. Soluble chromates readily penetrate intact skin. Cement dermatitis can be characterised by fissures, eczematous rash, dystrophic nails, and dry skin; acute contact with highly alkaline mixtures may cause localised necrosis.

Cement eczema may be due to chromium in feed stocks or contamination from materials of construction used in processing the cement. Sensitisation to chromium may be the leading cause of nickel and cobalt sensitivity and the high alkalinity of cement is an important factor in Chemwatch: 5696-91 Page 8 of 14
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cement dermatoses [ILO].

Repeated, prolonged severe inhalation exposure may cause pulmonary oedema and rarely, pulmonary fibrosis. Workers may also suffer from dust-induced bronchitis with chronic bronchitis reported in 17% of a group occupationally exposed to high dust levels. Respiratory symptoms and ventilatory function were studied in a group of 591 male Portland cement workers employed in four Taiwanese cement plants, with at least 5 years of exposure (1). This group had a significantly lowered mean forced vital capacity (FCV), forced expiratory volume at 1 s (FEV1) and forced expiratory flows after exhalation of 50% and 75% of the vital capacity (FEF50, FEF75). The data suggests that occupational exposure to Portland cement dust may lead to a higher incidence of chronic respiratory symptoms and a reduction of ventilatory capacity.

Chun-Yuh et al; Journal of Toxicology and Environmental Health 49: 581-588, 1996

On the basis of epidemiological data, the material is regarded as carcinogenic to humans. There is sufficient data to establish a causal association between human exposure to the material and the development of cancer.

Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.

Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.

TOXICITY	IRRITATION
Not Available	Not Available
TOXICITY	IRRITATION
Oral (Rat) LD50: 500 mg/kg <sup>[2]</sup>	Not Available
TOXICITY	IRRITATION
Not Available	Not Available
TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
Inhalation (Rat) LC50: >3.26 mg/l4h <sup>[1]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
Oral (Rat) LD50: >1581 mg/kg <sup>[1]</sup>	
TOXICITY	IRRITATION
Inhalation (Rat) LC50: >3.26 mg/l4h <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
Oral (Rat) LD50: >1581 mg/kg <sup>[1]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
TOXICITY	IRRITATION
dermal (hamster) LD50: >=10000 mg/kg <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
Inhalation (Rat) LC50: >2.28 mg/l4h <sup>[1]</sup>	Skin (human): 0.3 mg /3D (int)-mild *
Oral (Rat) LD50: >=2000 mg/kg <sup>[1]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
TOXICITY	IRRITATION
	TOXICITY  Oral (Rat) LD50: 500 mg/kg <sup>[2]</sup> TOXICITY  Not Available  TOXICITY  Not Available  TOXICITY  dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup> Inhalation (Rat) LC50: >3.26 mg/l4h <sup>[1]</sup> Oral (Rat) LD50: >1581 mg/kg <sup>[1]</sup> TOXICITY  Inhalation (Rat) LC50: >3.26 mg/l4h <sup>[1]</sup> Oral (Rat) LD50: >1581 mg/kg <sup>[1]</sup> TOXICITY  Inhalation (Rat) LC50: >2.28 mg/l4h <sup>[1]</sup> Oral (Rat) LD50: >=2000 mg/kg <sup>[1]</sup> Oral (Rat) LD50: >=2000 mg/kg <sup>[1]</sup>

Legend:

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

### PORTLAND CEMENT

The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons

### CALCIUM ALUMINATE SULFATE

tested.

for calcium:

Toxicity from calcium is not common because the gastrointestinal tract normally limits the amount of calcium absorbed. Therefore, short-term

intake of large amounts of calcium does not generally produce any ill effects aside from **constipation** and an increased risk of kidney stones. However, more severe toxicity can occur when excess calcium is ingested over long periods, or when calcium is combined with increased amounts of vitamin D, which increases calcium absorption. Calcium toxicity is also sometimes found after excessive intravenous administration of calcium. Toxicity is manifested by abnormal deposition of calcium in tissues and by elevated blood calcium levels (hypercalcaemia). However, hypercalcaemia is often due to other causes, such as abnormally high amounts of parathyroid hormone (PTH). Usually, under these circumstances, bone density is lost and the resulting hypercalcaemia can cause kidney stones and abdominal pain. Some cancers can also cause hypercalcaemia, either by secreting abnormal proteins that act like PTH or by invading and killing bone cells causing them to release calcium. Very high levels of calcium can result in appetite loss, nausea, vomiting, abdominal pain, confusion, seizures, and even coma.

for calcium chloride:

Acute toxicity: The acute oral toxicity of calcium chloride is low: LD50 in mice is 1940-2045 mg/kg bw, 3798-4179 mg/kg bw in rats, and 500-1000 mg/kg bw in rabbits. The acute oral toxicity is attributed to the severe irritating property of the original substance or its high-concentration solutions to the gastrointestinal tract. In humans, however, acute oral toxicity is rare because large single doses induce nausea and vomiting. The dermal acute toxicity is negligible: LD50 in rabbits >5000 mg/kg bw. No significant change was found by gross necropsy examination except skin lesions at or near the site of administration. Hypercalcaemia may occur only when there exists other factors that alter calcium homeostasis, such as renal inefficiency and primary hyperthyroidism.

Irritation/corrosiveness studies conducted under OECD test guidelines indicate that calcium chloride is not/slightly irritating to skin but severely irritating to eyes of rabbits. Prolonged exposure and application of moistened material or concentrated solutions resulted in considerable skin irritation, however. Irritating effect of the substance was observed in human skin injuries caused by incidental contact with the substance or its high-concentration solutions.

Repeat dose toxicity: A limited oral repeated dose toxicity study shows no adverse effect of calcium chloride on rats fed on 1000-2000 mg/kg bw/day for 12 months. Calcium and chloride are both essential nutrients for humans and a daily intake of more than 1000 mg each of the ions is recommended. The establishment of the ADI for calcium chloride has not been deemed necessary by JECFA (Joint FAO/WHO Expert Committee on Food Additives)

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**Genotoxicity:** Genetic toxicity of calcium chloride was negative in the bacterial mutation tests and the mammalian chromosome aberration test.

Reproductive and developmental toxicity: No reproductive toxicity study has been reported. A developmental toxicity study equivalent to an OECD Guideline study, on the other hand, reveals no toxic effects on dams or foetuses at doses up to 189 mg/kg bw/day (mice), 176 mg/kg bw/day (rats) and 169 mg/kg bw/day (rabbits).

Gypsum (calcium sulfate dihydrate) is a skin, eye, mucous membrane, and respiratory system irritant. Early studies of gypsum miners did not relate pneumoconiosis with chronic exposure to gypsum. Other studies in humans (as well as animals) showed no lung fibrosis produced by natural dusts of calcium sulfate except in the presence of silica. However, a series of studies reported chronic nonspecific respiratory diseases in gypsum industry workers in Gacki. Poland.

Unlike other fibers, gypsum is very soluble in the body; its half-life in the lungs has been estimated as minutes. In four healthy men receiving calcium supplementation with calcium sulfate (CaSO4-1/2H2O) (200 or 220 mg) for 22 days, an average absorption of 28.3% was reported. Several feeding studies in pigs on the bioavailability of calcium in calcium supplements, including gypsum, have been conducted. The bioavailability of calcium in gypsum was similar to that for calcitic limestone, oyster shell flour, marble dust, and aragonite, ranging from 85 to 102%. In mice, the i.p. and intragastric LD50 values were 6200 and 4704 mg/kg, respectively, for phosphogypsum (98% CaSO4-H2O). For Plaster of Paris, the values were 4415 and 5824, respectively. In

rats, an intragastric LD50 of 9934 mg/kg was reported for phosphogypsum

Repeat dose toxicity: In a study of 241 underground male workers employed in four gypsum mines in Nottinghamshire and Sussex for a year (November 1976-December 1977), results of chest X-rays, lung function tests, and respiratory systems suggested an association of the observed lung shadows with the higher quartz content in dust rather than to gypsum; the small round opacities in the lungs were characteristic of silica exposure.

Prophylactic examinations of workers in a gypsum extraction and production plant (dust concentration exceeded TLV 2.5- to 10-fold) reported no risk of pneumoconiosis due to gypsum exposure, while another study of gypsum manufacturing plant workers reported that chronic occupational exposure to gypsum dust had resulted in pulmonary ventilatory defect of the restrictive form.

Three cases of idiopathic interstitial pneumonia with multiple bullae throughout the lungs were seen in Japanese schoolteachers (lifetime occupation) exposed to chalk; 2/3 of the chalk was made from gypsum and small amounts of silica and other minerals. In rats exposed to an aerosol of anhydrous calcium sulfate fibers (15 mg/m3) or a combination of milled and fibrous calcium sulfate (60

In rats exposed to an aerosol of anhydrous calcium sulfate fibers (15 mg/m3) or a combination of milled and fibrous calcium sulfate (60 mg/m3) six hours per day, five days per week for three weeks, gypsum dust was quickly cleared from the lungs of via dissolution and mechanisms of particle clearance.

In guinea pigs given intraperitoneal (i.p.) injections of gypsum (doses not provided), gypsum was absorbed followed by the dissolution of gypsum in surrounding tissues. In another study, after i.p. injection of gypsum (2 cm3 of a 5 or 10% suspension in saline) into guinea pigs, which were sacrificed at intervals up to 180 days, most of the dust was found distributed in the peritoneum of the anterior abdominal wall. Gypsum dust produced irregular and clustered nodules, which decreased in size over time.

Direct administration of WTC PM2.5 [mostly composed of calcium-based compounds, including calcium sulfate (gypsum) and calcium carbonate (calcite)] (10, 32, or 100 µg) into the airways of mice produced mild to moderate lung inflammation and airway hyperresponsiveness at the high dose. [It was noted that WTC PM2.5 is composed of many chemical species and that their interactions may be related with development of airway hyperresponsiveness.] In female SPF Wistar rats intratracheally (i.t.) instilled with anhydrite dust (35 mg) and sacrificed three months later, an increase in total lipid or hydroxyproline content in the lungs was not observed compared to controls.

In inhalation (nose-only) experiments in which male F344 rats were exposed to calcium sulfate fiber aerosols (100 mg/m3) for six hours per day, five days per week for three weeks, there were no effects on the number of macrophages per alveolus, bronchoalveolar lavage fluid (BALF) protein concentration, or BALF g-glutamyl transpeptidase activity (g-GT). Following three weeks of recovery, nonprotein thiol levels (NPSH), mainly glutathione, were increased in animals. In follow-up experiments, rats were exposed to an aerosol of anhydrous calcium sulfate fibers (15 mg/m3) or a combination of milled and fibrous calcium sulfate (60 mg/m3) for the same duration. Calcium levels in the lungs were similar to those of controls; however, gypsum fibers were detected in the lungs of treated animals. Significant increases in NSPH levels in BALF were observed in rats killed immediately after exposure at both doses and in recovery group animals at the higher dose. At 15 mg/m3, almost all NPSH was lost in macrophages from all treated animals (including those in recovery), but a significant decrease in extracellular g-GT activity was seen only in recovery group animals. Overall, the findings were "considered to be non-pathological local effects due to physical factors related to the shape of the gypsum fibers and not to calcium sulphate per se."

Intratracheal administration of man-made calcium sulfate fiber (2.0 mg) once per week for five weeks resulted in no deaths or significant

body weight changes in female Syrian hamsters compared to controls.

Inflammation (specifically, chronic alveolitis with macrophage and neutrophil aggregation) was observed in the lung.

In guinea pigs, inhalation of calcined gypsum dust (1.6 x 104 particles/mL) for 44 hours per week in 5.5 days for two years, followed with or without a recovery period of up to 22 months, produced only minor effects in the lungs. There were 12 of 21 deaths over the entire experimental period. These were due to pneumonia or other pulmonary lesions; however, no significant gross signs of pulmonary disease or nodular or diffuse pneumocniosis became significant. Beginning near 11 months, pigmentation and atelectasis were seen. During the recovery period, four of ten guinea pigs died; two died of pneumonia. Pigmentation continued in most animals but not atelectasis. Low-grade chronic inflammation, occurring in the first two months, also disappeared.

Mercury emissions controls on coal-fired power plants have increased the likelihood of the presence of mercury in synthetic gypsum formed in wet flue gas desulfurisation (FGD) systems and the finished wallboard produced from the FGD gypsum. In a study at a commercial wallboard plant, the raw FGD gypsum, the product stucco (beta form of CaSO4-1/2H2O), and the finished dry wallboard each contained about 1 ug Hg/g dry weight. Total mercury loss from the original FGD gypsum content was about 0.045 g Hg/ton dry gypsum processed Synergistic/Antagonistic Effects: In rats, i.t. administration of anhydrite (5-35 mg) successively and simultaneously with quartz reduced the toxic effect of quartz in lung tissue. This protective effect on quartz toxicity was also seen in guinea pigs; calcined gypsum dust prevented or hindered the development of fibrosis. Natural anhydrite, however, increased the fibrogenic effect of cadmium sulfide in rats. Additionally, calcined gypsum dust had a stimulatory effect on experimental tuberculosis in guinea pigs.

Cytotoxicity: In Syrian hamster embryo cells, gypsum (up to 10 ug/cm2) did not induce apoptosis. Negative results were also found in mouse peritoneal macrophages (tested at 150 ug/mL gypsum dust) and in Chinese hamster lung V79-4 cells (tested up to 100 ug/mL). Carcinogenicity: In female Sprague-Dawley rats, i.p. injection of natural anhydrite dusts from German coal mines (doses not provided) induced granulomas; whether gypsum was the causal factor was not established. In Wistar rats, four i.p. injections of gypsum (25 mg each) induced abdominal cavity tumours, mostly sarcomatous mesothelioma, in 5% of animals; first tumour was seen at 546 days. In a subsequent experiment using the same procedure, female Wistar rats exhibited the first tumour at 579 days after the last injection. Mean survival of the tumour-bearing rats (5.7% of test group) was 583 days, while mean survival of the test group was 587 days. Tumour types seen were a sarcoma having cellular polymorphism, a carcinoma, and a reticulosarcoma.

Intratracheal administration of man-made calcium sulfate fiber (2.0 mg) once per week for five weeks produced tumours in three of 20 female Syrian hamsters observed two years later. An anaplastic carcinoma was found in the heart, and one dark cell carcinoma was seen in the kidney. Two tumours of unspecified types were observed in the rib.

the kidney. Two tumours of unspecified types were observed in the rib. In guinea pigs, inhalation of gypsum (doses not provided) for 24 months produced no lung tumours.

In rats, i.t. administration of gypsum (doses not provided in abstract) from FGD for up to 18 months produced no arterial blood gas changes or indications of secondary heart damage as compared to controls.

In another study, a single i.t. dose (25 mg) of flue gas gypsum dust did not produce a pathological reaction when observed for up to 18 months. There were also no signs of developing granuloma of fibrosis of the lungs. Lead quickly accumulated in the femur after injection but was eliminated during the observation period. In the Ames test, the flue gas gypsum dust was negative.

Genotoxicity: Calcium sulfate (up to 2.5%) was negative in Salmonella typhimurium strains TA1535, TA1537, and TA1538 and in

**Genotoxicity:** Calcium sulfate (up to 2.5%) was negative in Salmonella typhimurium strains TA1535, TA1537, and TA1538 and i Saccharomyces cerevisiae strain D4 with and without metabolic activation.

**Developmental toxicity:** In pregnant mice, rats, and rabbits, daily oral administration of calcium sulfate (16-1600 mg/kg bw) beginning on gestation day 6 up to 18 produced no effects on maternal body weights, maternal or foetal survival, or nidation (embryo implantation); developmental effects were also not seen.

### TITANIUM DIOXIDE

**CALCIUM SULFATE** 

\* IUCLID

Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of

appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.

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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 invitro genotoxicity studies with titanium dioxide were negative.

The material may produce moderate eye irritation leading to 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 epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.

## SILICA CRYSTALLINE -

WARNING: For inhalation exposure ONLY: This substance has been classified by the IARC as Group 1: CARCINOGENIC TO HUMANS

The International Agency for Research on Cancer (IARC) has classified occupational exposures to **respirable** (<5 um) crystalline silica as being carcinogenic to humans . This classification is based on what IARC considered sufficient evidence from epidemiological studies of humans for the carcinogenicity of inhaled silica in the forms of quartz and cristobalite. Crystalline silica is also known to cause silicosis, a non-cancerous lung disease.

Intermittent exposure produces; focal fibrosis, (pneumoconiosis), cough, dyspnoea, liver tumours.

\* Millions of particles per cubic foot (based on impinger samples counted by light field techniques).

NOTE: the physical nature of quartz in the product determines whether it is likely to present a chronic health problem. To be a hazard the material must enter the breathing zone as respirable particles.

# GRADED SAND & PORTLAND CEMENT & CALCIUM ALUMINATE SULFATE & TITANIUM DIOXIDE

No significant acute toxicological data identified in literature search.

# PORTLAND CEMENT & CALCIUM SULFATE & TITANIUM DIOXIDE

Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.

Acute Toxicity	X	Carcinogenicity	✓
Skin Irritation/Corrosion	✓	Reproductivity	×
Serious Eye Damage/Irritation	<b>~</b>	STOT - Single Exposure	<b>~</b>
Respiratory or Skin sensitisation	<b>~</b>	STOT - Repeated Exposure	<b>~</b>
Mutagenicity	✓	Aspiration Hazard	×

Legend:

💢 – Data either not available or does not fill the criteria for classification

– Data available to make classification

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### **SECTION 12 Ecological information**

### Toxicity

	Endpoint	Test Duration (hr)	Species	Value	Source
Ardex Quickbond	Not Available	Not Available	Not Available	Not Available	Not Available
	Endpoint	Test Duration (hr)	Species	Value	Source
graded sand	Not Available	Not Available	Not Available	Not Available	Not Available
	Endpoint	Test Duration (hr)	Species	Value	Source
portland cement	Not Available	Not Available	Not Available	Not Available	Not Available
	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	4.8mg/l	2
calcium aluminate sulfate	EC50	48h	Crustacea	6.8mg/l	2
	LC50	96h	Fish	>83mg/l	2
	EC10(ECx)	72h	Algae or other aquatic plants	2.3mg/l	2
	Endpoint	Test Duration (hr)	Species	Value	Sourc
	EC50	72h	Algae or other aquatic plants	>79mg/l	2
calcium sulfate	LC50	96h	Fish	>79mg/l	2
	EC50	96h	Algae or other aquatic plants	3200mg/L	4
	NOEC(ECx)	0.25h	Fish	75mg/l	4
	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
titanium dioxide	EC50	48h	Crustacea	1.9mg/l	2
	LC50	96h	Fish	1.85- 3.06mg/l	4
	NOEC(ECx)	672h	Fish	>=0.004mg/L	2
	EC50	96h	Algae or other aquatic plants	179.05mg/l	2
	Endpoint	Test Duration (hr)	Species	Value	Source
silica crystalline - quartz	Not Available	Not Available	Not Available	Not Available	Not Available

### DO NOT discharge into sewer or waterways.

### Persistence and degradability

Ingredient Persistence: Water/Soil		Persistence: Air
calcium sulfate	HIGH	HIGH
titanium dioxide	HIGH	HIGH

### **Bioaccumulative potential**

Ingredient	Bioaccumulation	
calcium sulfate	LOW (LogKOW = -2.2002)	
titanium dioxide	LOW (BCF = 10)	

### Mobility in soil

Ingredient	Mobility
calcium sulfate	LOW (Log KOC = 6.124)
titanium dioxide	LOW (Log KOC = 23.74)

### **SECTION 13 Disposal considerations**

### Waste treatment methods

### Product / Packaging disposal

- ▶ DO NOT allow wash water from cleaning or process equipment to enter drains.
- ▶ It may be necessary to collect all wash water for treatment before disposal.
- In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
   Where in doubt contact the responsible authority.
- Recycle wherever possible or consult manufacturer for recycling options.

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- ▶ Consult State Land Waste Authority for disposal.
- ▶ Bury or incinerate residue at an approved site.
- · Recycle containers if possible, or dispose of in an authorised landfill.

### **SECTION 14 Transport information**

### Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

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

Product name	Group
graded sand	Not Available
portland cement	Not Available
calcium aluminate sulfate	Not Available
calcium sulfate	Not Available
titanium dioxide	Not Available
silica crystalline - quartz	Not Available

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

Product name	Ship Type
graded sand	Not Available
portland cement	Not Available
calcium aluminate sulfate	Not Available
calcium sulfate	Not Available
titanium dioxide	Not Available
silica crystalline - quartz	Not Available

### **SECTION 15 Regulatory information**

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

### graded sand is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans

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

### portland cement is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

### calcium aluminate sulfate 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)

### calcium sulfate 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 - Group 2B: Possibly carcinogenic to humans

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

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

### silica crystalline - quartz is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans

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

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### **Additional Regulatory Information**

Not Applicable

### **National Inventory Status**

National Inventory	Status	
Australia - AIIC / Australia Non- Industrial Use	Yes	
Canada - DSL	Yes	
Canada - NDSL	No (graded sand; portland cement; calcium aluminate sulfate; calcium sulfate; silica crystalline - quartz)	
China - IECSC	Yes	
Europe - EINEC / ELINCS / NLP	es	
Japan - ENCS	No (portland cement; calcium aluminate sulfate)	
Korea - KECI	/es	
New Zealand - NZIoC	Yes	
Philippines - PICCS	No (portland cement; calcium aluminate sulfate)	
USA - TSCA	Yes	
Taiwan - TCSI	Yes	
Mexico - INSQ	No (calcium aluminate sulfate)	
Vietnam - NCI	Yes	
Russia - FBEPH	No (calcium aluminate sulfate)	
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.	

### **SECTION 16 Other information**

Revision Date	26/08/2024
Initial Date	26/08/2024

### **SDS Version Summary**

Version	Date of Update	Sections Updated
2.1	26/08/2024	Handling and storage - Storage (suitable container)

### 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

### **Definitions and abbreviations**

- ▶ PC TWA: Permissible Concentration-Time Weighted Average
- ▶ PC STEL: Permissible Concentration-Short Term Exposure Limit
- ▶ IARC: International Agency for Research on Cancer
- ACGIH: American Conference of Governmental Industrial Hygienists
- STEL: Short Term Exposure Limit
- ► TEEL: Temporary Emergency Exposure Limit。
- ▶ IDLH: Immediately Dangerous to Life or Health Concentrations
- ▶ ES: Exposure Standard
- OSF: Odour Safety Factor
- NOAEL: No Observed Adverse Effect Level
- LOAEL: Lowest Observed Adverse Effect Level
- TLV: Threshold Limit Value
- LOD: Limit Of Detection
- OTV: Odour Threshold Value
- ▶ BCF: BioConcentration Factors
- ▶ BEI: Biological Exposure Index
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration
- ▶ AIIC: Australian Inventory of Industrial Chemicals
- DSL: Domestic Substances ListNDSL: 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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**Ardex Quickbond** 

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