Ardex FG8, Flexible Coloured Grout

Ardex (Ardex Australia)

Chemwatch Hazard Alert Code: **3**

Chemwatch: **5410-55**
Version No: **5.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **23/12/2022**
Print Date: **22/06/2023**
L.GHS.AUS.EN.E

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

Product Identifier

Product name	Ardex FG8, Flexible Coloured Grout
Chemical Name	Not Applicable
Synonyms	Grouting Material
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	Cement based grouting material for filling joints around ceramic floor and wall tiles.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**Ardex (Ardex Australia)**
Address	20 Powers Road Seven Hills NSW 2147 Australia
Telephone	1800 224 070
Fax	1300 780 102
Website	www.ardexaustralia.com
Email	technicalservices@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.

Chemwatch Hazard Ratings

	Min	Max
Flammability	1	
Toxicity	1	
Body Contact	3	
Reactivity	1	
Chronic	2	

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

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

Label elements

Page 1 continued...

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

Hazard statement(s)

H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H318	Causes serious eye damage.
H335	May cause respiratory irritation.
H341	Suspected of causing genetic defects.

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 dust/fumes.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

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 and soap.
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
471-34-1	30-60	calcium carbonate
14808-60-7.	30-60	graded sand
65997-15-1	10-30	portland cement
1317-61-9	0-2	C.I. Pigment Black 11
13463-67-7	<2	C.I. Pigment White 6
Not Available	balance	Ingredients determined not to be hazardous
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available*	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

Continued...

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. For thermal burns: ▸ Decontaminate area around burn. ▸ Consider the use of cold packs and topical antibiotics. For first-degree burns (affecting top layer of skin) ▸ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides. ▸ Use compresses if running water is not available. ▸ Cover with sterile non-adhesive bandage or clean cloth. ▸ Do NOT apply butter or ointments; this may cause infection. ▸ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur. For second-degree burns (affecting top two layers of skin) ▸ Cool the burn by immerse in cold running water for 10-15 minutes. ▸ Use compresses if running water is not available. ▸ Do NOT apply ice as this may lower body temperature and cause further damage. ▸ Do NOT break blisters or apply butter or ointments; this may cause infection. ▸ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape. To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort): ▸ Lay the person flat. ▸ Elevate feet about 12 inches. ▸ Elevate burn area above heart level, if possible. ▸ Cover the person with coat or blanket. ▸ Seek medical assistance. For third-degree burns Seek immediate medical or emergency assistance. In the mean time: ▸ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound. ▸ Separate burned toes and fingers with dry, sterile dressings. ▸ Do not soak burn in water or apply ointments or butter; this may cause infection. ▸ To prevent shock see above. ▸ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. ▸ Have a person with a facial burn sit up. ▸ Check pulse and breathing to monitor for shock until emergency help arrives.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.
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

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

Special hazards arising from the substrate or mixture

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

Continued...

Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves in the event of a fire. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ Use fire fighting procedures suitable for surrounding area. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire. ▸ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	▸ 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. Decomposes on heating and produces: carbon monoxide (CO) carbon dioxide (CO_2) silicon dioxide (SiO_2) 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	▸ Clean up waste regularly and abnormal spills immediately. ▸ Avoid breathing dust and contact with skin and eyes. ▸ Wear protective clothing, gloves, safety glasses and dust respirator. ▸ Use dry clean up procedures and avoid generating dust. ▸ 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. ▸ Dampen with water to prevent dusting before sweeping. ▸ Place in suitable containers for disposal.
Major Spills	▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by all means available, spillage from entering drains or water courses. ▸ Consider evacuation (or protect in place). ▸ No smoking, naked lights or ignition sources. ▸ Increase ventilation. ▸ Stop leak if safe to do so. ▸ Water spray or fog may be used to disperse / absorb vapour. ▸ Contain or absorb spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▸ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ 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.

Continued...

	▸ 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. ▸ Organic powders when finely divided over a range of concentrations regardless of particulate size or shape and suspended in air or some other oxidizing medium may form explosive dust-air mixtures and result in a fire or dust explosion (including secondary explosions) ▸ Minimise airborne dust and eliminate all ignition sources. Keep away from heat, hot surfaces, sparks, and flame. ▸ Establish good housekeeping practices. ▸ Remove dust accumulations on a regular basis by vacuuming or gentle sweeping to avoid creating dust clouds. ▸ Use continuous suction at points of dust generation to capture and minimise the accumulation of dusts. Particular attention should be given to overhead and hidden horizontal surfaces to minimise the probability of a "secondary" explosion. According to NFPA Standard 654, dust layers 1/32 in.(0.8 mm) thick can be sufficient to warrant immediate cleaning of the area. ▸ Do not use air hoses for cleaning. ▸ Minimise dry sweeping to avoid generation of dust clouds. Vacuum dust-accumulating surfaces and remove to a chemical disposal area. Vacuums with explosion-proof motors should be used. ▸ Control sources of static electricity. Dusts or their packages may accumulate static charges, and static discharge can be a source of ignition. ▸ Solids handling systems must be designed in accordance with applicable standards (e.g. NFPA including 654 and 77) and other national guidance. ▸ Do not empty directly into flammable solvents or in the presence of flammable vapors. ▸ The operator, the packaging container and all equipment must be grounded with electrical bonding and grounding systems. Plastic bags and plastics cannot be grounded, and antistatic bags do not completely protect against development of static charges. Empty containers may contain residual dust which has the potential to accumulate following settling. Such dusts may explode in the presence of an appropriate ignition source. ▸ Do NOT cut, drill, grind or weld such containers. ▸ In addition ensure such activity is not performed near full, partially empty or empty containers without appropriate workplace safety authorisation or permit.
Other information	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ Store in a cool, dry area protected from environmental extremes. ▸ 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. For major quantities: ▸ Consider storage in bunded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams}. ▸ Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities.

Conditions for safe storage, including any incompatibilities

Suitable container	Multi-ply paper bag with sealed plastic liner or heavy gauge plastic bag. **NOTE:** Bags should be stacked, blocked, interlocked, and limited in height so that they are stable and secure against sliding or collapse. Check that all containers are clearly labelled and free from leaks. Packing as recommended by manufacturer.
Storage incompatibility	▸ Avoid strong acids, bases. ▸ Avoid contact with copper, aluminium and their alloys. ▸ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	calcium carbonate	Calcium carbonate	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia 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	C.I. Pigment White 6	Titanium dioxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
calcium carbonate	45 mg/m3	210 mg/m3	1,300 mg/m3
graded sand	0.075 mg/m3	33 mg/m3	200 mg/m3
C.I. Pigment Black 11	21 mg/m3	230 mg/m3	1,400 mg/m3
C.I. Pigment White 6	30 mg/m3	330 mg/m3	2,000 mg/m3

Ingredient	Original IDLH	Revised IDLH
calcium carbonate	Not Available	Not Available

Continued...

Ingredient	Original IDLH	Revised IDLH
graded sand	25 mg/m3 / 50 mg/m3	Not Available
portland cement	5,000 mg/m3	Not Available
C.I. Pigment Black 11	Not Available	Not Available
C.I. Pigment White 6	5,000 mg/m3	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
C.I. Pigment Black 11	E	≤ 0.01 mg/m³
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

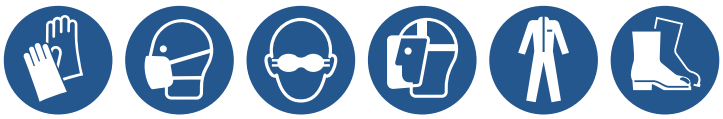
Exposure controls

Appropriate engineering controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.

- Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area.
- Work should be undertaken in an isolated system such as a "glove-box" . Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system.
- Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within.
- Open-vessel systems are prohibited.
- Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation.
- Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system.
- For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood.
- Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas).
- Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air.
- Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed.

Individual protection measures, such as personal protective equipment



Eye and face protection

- Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure.
- Chemical goggles. Whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted. [AS/NZS 1337.1, EN166 or national equivalent]
- Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection.
- Alternatively a gas mask may replace splash goggles and face shields.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].

Skin protection

See Hand protection below

Hands/feet protection

- Elbow length PVC gloves

NOTE:

- The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
· frequency and duration of contact,
· chemical resistance of glove material,
· glove thickness and
· dexterity
Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240

Continued...

	minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. ▸ Neoprene rubber gloves 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
Other protection	▸ Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent] ▸ Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent] ▸ Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely. ▸ Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. ▸ Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▸ Overalls. ▸ P.V.C apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.

Respiratory protection

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

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

If inhalation risk above the TLV exists, wear approved dust respirator.
Use respirators with protection factors appropriate for the exposure level.
▸ Up to 5 X TLV, use valveless mask type; up to 10 X TLV, use 1/2 mask dust respirator
▸ Up to 50 X TLV, use full face dust respirator or demand type C air supplied respirator
▸ Up to 500 X TLV, use powered air-purifying dust respirator or a Type C pressure demand supplied-air respirator
▸ Over 500 X TLV wear full-face self-contained breathing apparatus with positive pressure mode or a combination respirator with a Type C positive pressure supplied-air full-face respirator and an auxiliary self-contained breathing apparatus operated in pressure demand or other positive pressure mode
· 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

Continued...

appropriate government standards such as NIOSH (US) or CEN (EU)
· Use approved positive flow mask if significant quantities of dust becomes airborne.
· Try to avoid creating dust conditions.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Coloured powder; insoluble 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
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	Immiscible	**pH as a solution (1%)**	Not Applicable
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. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Inhalation of dusts, generated by the material during the course of normal handling, may be damaging to the health of the individual. Inhalation may result in chrome ulcers or sores of nasal mucosa and lung damage. 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	Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	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. 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

Continued...

	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. On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body 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. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Red blood cells and rabbit alveolar macrophages exposed to calcium silicate insulation materials in vitro showed haemolysis in one study but not in another. Both studies showed the substance to be more cytotoxic than titanium dioxide but less toxic than asbestos. In a small cohort mortality study of workers in a wollastonite quarry, the observed number of deaths from all cancers combined and lung cancer were lower than expected. Wollastonite is a calcium inosilicate mineral (CaSiO3). In some cases, small amounts of iron (Fe), and manganese (Mn), and lesser amounts of magnesium (Mg) substitute for calcium (Ca) in the mineral formulae (*e.g.*, rhodonite) In an inhalation study in rats no increase in tumour incidence was observed but the number of fibres with lengths exceeding 5 um and a diameter of less than 3 um was relatively low. Four grades of wollastonite of different fibre size were tested for carcinogenicity in one experiment in rats by intrapleural implantation. There was no information on the purity of the four samples used. A slight increase in the incidence of pleural sarcomas was observed with three grades, all of which contained fibres greater than 4 um in length and less than 0.5 um in diameter. In two studies by intraperitoneal injection in rats using wollastonite with median fibre lengths of 8.1 um and 5.6 um respectively, no intra-abdominal tumours were found. Evidence from wollastonite miners suggests that occupational exposure can cause impaired respiratory function and pneumoconiosis. However animal studies have demonstrated that wollastonite fibres have low biopersistence and induce a transient inflammatory response compared to various forms of asbestos. A two-year inhalation study in rats at one dose showed no significant inflammation or fibrosis Chromium(III) is considered an essential trace nutrient serving as a component of the "glucose tolerance factor" and a cofactor for insulin action. High concentrations of chromium are also found in RNA. Trivalent chromium is the most common form found in nature. Chronic inhalation of trivalent chromium compounds produces irritation of the bronchus and lungs, dystrophic changes to the liver and kidney, pulmonary oedema, and adverse effects on macrophages. Intratracheal administration of chromium(III) oxide, in rats, increased the incidence of sarcomas, and tumors and reticulum cell sarcomas of the lung. There is inadequate evidence of carcinogenicity of chromium(III) compounds in experimental animals and humans (IARC). Chronic exposure to hexavalent chromium compounds reportedly produces skin, eye and respiratory tract irritation, yellowing of the eyes and skin, allergic skin and respiratory reactions, diminished sense of smell and taste, blood disorders, liver and kidney damage, digestive disorders and lung damage. There is sufficient evidence of carcinogenicity of chromium(VI) compounds in experimental animals and humans to confirm these as Class 1 carcinogens (IARC). Exposure to chromium during chrome production and in the chrome pigment industry is associated with cancer of the respiratory tract. A slight increase in gastrointestinal cancer following exposure to chromium compounds has also been reported. The greatest risk is attributed to exposure to acid-soluble, water-insoluble hexavalent chromium which occurs in roasting and refining processes. Animal studies support the idea that the most potent carcinogenic compounds are the slightly soluble hexavalent compounds. The cells are more active in the uptake of the hexavalent forms compared to trivalent forms and this may explain the difference in occupational effect. It is the trivalent form, however, which is metabolically active and binds with nucleic acid within the cell suggesting that chromium mutagenesis first requires biotransformation of the hexavalent form by reduction. Hexavalent chromes produce chronic ulceration of skin surfaces (quite independent of other hypersensitivity reactions exhibited by the skin). Water-soluble chromium(VI) compounds come close to the top of any published "hit list" of contact allergens (eczematogens) producing positive results in 4 to 10% of tested individuals. On the other hand only chromium(III) compounds can bind to high molecular weight carriers such as proteins to form a complete allergen (such as a hapten). Chromium(VI) compounds cannot. It is assumed that reduction must take place for such compounds to manifest any contact sensitivity. The apparent contradiction that chromium(VI) salts cause allergies to chromium(III) compounds but that allergy to chromium(III) compounds is difficult to demonstrate is accounted for by the different solubilities and skin penetration of these compounds. Water-soluble chromium(VI) salts penetrate the horny layer of the skin more readily than chromium(III) compounds which are bound by cross-linking in the horny layer ("tanning", as for leather) and therefore do not reach the cells involved in antigen processing. The synthetic, amorphous silicas are believed to represent a very greatly reduced silicosis hazard compared to crystalline silicas and are considered to be nuisance dusts. When heated to high temperature and a long time, amorphous silica can produce crystalline silica on cooling. Inhalation of dusts containing crystalline silicas may lead to silicosis, a disabling pulmonary fibrosis that may take years to develop. Discrepancies between various studies showing that fibrosis associated with chronic exposure to amorphous silica and those that do not may be explained by assuming that diatomaceous earth (a non-synthetic silica commonly used in industry) is either weakly fibrogenic or nonfibrogenic and that fibrosis is due to contamination by crystalline silica content Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic. Because aluminium competes with calcium for absorption, increased amounts of dietary aluminium may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies, aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseus tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory These salts' estrogen-like effects have led to their classification as a metalloestrogen.Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.

Continued...

After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus. Aluminium may persist for a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans.
At high levels of exposure, some aluminium compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminium compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminium potassium sulphate at high levels in the diet.
Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer s disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produce lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, testes, embryotoxicity, and the developing nervous system of 52, 75, 100, and 50 mg aluminium/kg bw/day, respectively. Similarly, the lowest no-observed-adverse-effect levels (NOAELs) for effects on these endpoints were reported at 30, 27, 100, and for effects on the developing nervous system, between 10 and 42 mg aluminium/kg bw per day, respectively.
Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 mg/l aluminium compared with communities where the aluminium level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminium exposure to brain disease. Aluminium concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramid-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations.
Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of "tau" a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminium hyperphosphorylates tau in vitro. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade. Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA).
Aluminium enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminium include baking powder, antacids and aluminium products used for general food preparation and storage (over 12 months, aluminium levels in soft drink packed in aluminium cans rose from 0.05 to 0.9 mg/l). [*Walton, J and Bryson-Taylor, D. - Chemistry in Australia, August 1995*]
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 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
Repeated exposure to synthetic amorphous silicas may produce skin dryness and cracking.
Available data confirm the absence of significant toxicity by oral and dermal routes of exposure.
Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted in a number of species, at airborne concentrations ranging from 0.5 mg/m3 to 150 mg/m3. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m3. When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m3. Differences in values may be due to particle size, and therefore the number of particles administered per unit dose. Generally, as particle size diminishes so does the NOAEL/ LOAEL. Exposure produced transient increases in lung inflammation, markers of cell injury and lung collagen content. There was no evidence of interstitial pulmonary fibrosis.
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.
Overexposure to the breathable dust may cause coughing, wheezing, difficulty in breathing and impaired lung function. Chronic symptoms may include decreased vital lung capacity and chest infections. Repeated exposures in the workplace to high levels of fine-divided dusts may produce a condition known as pneumoconiosis, which is the lodgement of any inhaled dusts in the lung, irrespective of the effect. This is particularly true when a significant number of particles less than 0.5 microns (1/50000 inch) are present. Lung shadows are seen in the X-ray. Symptoms of pneumoconiosis may include a progressive dry cough, shortness of breath on exertion, increased chest expansion, weakness and weight loss. As the disease progresses, the cough produces stringy phlegm, vital capacity decreases further, and shortness of breath becomes more severe. Other signs or symptoms include changed breath sounds, reduced oxygen uptake during exercise, emphysema and rarely, pneumothorax (air in the lung cavity).
Removing workers from the possibility of further exposure to dust generally stops the progress of lung abnormalities. When there is high potential for worker exposure, examinations at regular period with emphasis on lung function should be performed.
Inhaling dust over an extended number of years may cause pneumoconiosis, which is the accumulation of dusts in the lungs and the subsequent tissue reaction. This may or may not be reversible.
Chronic excessive iron exposure has been associated with haemosiderosis and consequent possible damage to the liver and pancreas. Haemosiderin is a golden-brown insoluble protein produced by phagocytic digestion of haematin (an iron-based pigment). Haemosiderin is found in most tissues, especially in the liver, in the form of granules. Other sites of haemosiderin deposition include the pancreas and skin. A related condition, haemochromatosis, which involves a disorder of metabolism of these deposits, may produce cirrhosis of the liver, diabetes, and bronze pigmentation of the skin - heart failure may eventually occur.
Such exposure may also produce conjunctivitis, choroiditis, retinitis (both inflammatory conditions involving the eye) and siderosis of tissues if iron remains in these tissues. Siderosis is a form of pneumoconiosis produced by iron dusts. Siderosis also includes discoloration of organs, excess circulating iron and degeneration of the retina, lens and uvea as a result of the deposition of intraocular iron. Siderosis might also involve the lungs - involvement rarely develops before ten years of regular exposure. Often there is an accompanying inflammatory reaction of the bronchi. Permanent scarring of the lungs does not normally occur.
High levels of iron may raise the risk of cancer. This concern stems from the theory that iron causes oxidative damage to tissues and organs by generating highly reactive chemicals, called free radicals, which subsequently react with DNA. Cells may be disrupted and may be become cancerous. People whose genetic disposition prevents them from keeping tight control over iron (e.g. those with the inherited disorder, haemochromatosis) may be at increased risk.
Iron overload in men may lead to diabetes, arthritis, liver cancer, heart irregularities and problems with other organs as iron builds up.
[K. Schmidt, New Scientist, No. 1919 pp.11-12, 2nd April, 1994]

Continued...

	Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.

Ardex FG8, Flexible Coloured Grout	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
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]
graded sand	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 500 mg/kg[2]	Not Available
portland cement	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
C.I. Pigment Black 11	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >2000 mg/kg[1]	Not Available
C.I. Pigment White 6	**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 (rabbit) Draize 0.3mg/3hrInt Mild
	Oral (Rat) LD50: >=2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*	

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.
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 tested.
C.I. PIGMENT BLACK 11	No data of toxicological significance identified in literature search.
C.I. PIGMENT WHITE 6	Substance has been investigated as a mutagen, tumorigen and primary irritant. For titanium dioxide: Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin. Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica. No data were available on genotoxic effects in titanium dioxide-exposed humans. Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts. Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium. Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light.

Continued...

	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. The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
CALCIUM CARBONATE & PORTLAND CEMENT & C.I. PIGMENT BLACK 11	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.
GRADED SAND & PORTLAND CEMENT & C.I. PIGMENT BLACK 11	No significant acute toxicological data identified in literature search.

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

Ardex FG8, Flexible Coloured Grout

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

calcium carbonate

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

graded sand

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

portland cement

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

C.I. Pigment Black 11

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

C.I. Pigment White 6

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

Continued...

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

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
C.I. Pigment White 6	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
C.I. Pigment White 6	LOW (BCF = 10)

Mobility in soil

Ingredient	Mobility
C.I. Pigment White 6	LOW (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.

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

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
calcium carbonate	Not Available
graded sand	Not Available
portland cement	Not Available
C.I. Pigment Black 11	Not Available
C.I. Pigment White 6	Not Available

Transport in bulk in accordance with the IGC Code

Product name	Ship Type
calcium carbonate	Not Available
graded sand	Not Available
portland cement	Not Available
C.I. Pigment Black 11	Not Available
C.I. Pigment White 6	Not Available

SECTION 15 Regulatory information

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

calcium carbonate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

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

graded sand is found on the following regulatory lists

Continued...

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

portland cement is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

C.I. Pigment Black 11 is found on the following regulatory lists

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

C.I. Pigment White 6 is found on the following regulatory lists

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

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (graded sand; portland cement; C.I. Pigment Black 11; C.I. Pigment White 6)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (portland cement)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (portland cement)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
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. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	23/12/2022
Initial Date	03/07/2020

SDS Version Summary

Version	Date of Update	Sections Updated
4.1	09/07/2020	Composition / information on ingredients - Ingredients
5.1	23/12/2022	Classification review due to GHS Revision change.

Other information

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

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

Definitions and abbreviations

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

Continued...

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

end of SDS