108 20 CEMENT EASY MIX 20KG (Easy Mix GP Cement)

River Sands

Chemwatch Hazard Alert Code: **3**

Chemwatch: **31-9494**
Version No: **5.1.1.1**
Safety Data Sheet according to WHS and ADG requirements

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SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	108 20 CEMENT EASY MIX 20KG (Easy Mix GP Cement)
Synonyms	Not Available
Other means of identification	Not Available

Relevant identified uses of the substance or mixture and uses advised against

Relevant identified uses	A general-purpose construction binder, which will harden after the addition of water.

Details of the supplier of the safety data sheet

Registered company name	**River Sands**
Address	683 Beenleigh-Redland Bay Road Carbrook QLD 4130 Australia
Telephone	+61 7 3412 8111
Fax	+61 7 3287 6445
Website	www.riversands.com.au
Email	info@riversands.com.au

Emergency telephone number

Association / Organisation	**Not Available**
Emergency telephone numbers	13 11 26
Other emergency telephone numbers	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Skin Corrosion/Irritation Category 1A, Serious Eye Damage Category 1, Germ cell mutagenicity Category 2
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HSIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

Hazard pictogram(s)	
SIGNAL WORD	**DANGER**

Hazard statement(s)

H314	Causes severe skin burns and eye damage.
H341	Suspected of causing genetic defects.

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Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P281	Use personal protective equipment as required.

Precautionary statement(s) Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353	IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower.
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 or doctor/physician.
P363	Wash contaminated clothing before reuse.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.

Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
65997-15-1	>30	portland cement
65996-69-2	0-65	blast furnace slag
68131-74-8.	0-30	fly ash - low quartz
1317-65-3	0-5	limestone
13397-24-5	4-7	gypsum
Not Available		NOTE: hexavalent chromium may be present at trace amounts

SECTION 4 FIRST AID MEASURES

Description of first aid measures

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

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	▸ pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor. ▸ If dust is inhaled, remove from contaminated area. ▸ Encourage patient to blow nose to ensure clear breathing passages. ▸ Ask patient to rinse mouth with water but to not drink water. ▸ Seek immediate medical attention.
Ingestion	▸ For advice, contact a Poisons Information Centre or a doctor at once. ▸ Urgent hospital treatment is likely to be needed. ▸ 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. ▸ Transport to hospital or doctor without delay.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

For acute or short term repeated exposures to iron and its derivatives:

- Always treat symptoms rather than history.
- In general, however, toxic doses exceed 20 mg/kg of ingested material (as elemental iron) with lethal doses exceeding 180 mg/kg.
- Control of iron stores depend on variation in absorption rather than excretion. Absorption occurs through aspiration, ingestion and burned skin.
- Hepatic damage may progress to failure with hypoprothrombinaemia and hypoglycaemia. Hepatorenal syndrome may occur.
- Iron intoxication may also result in decreased cardiac output and increased cardiac pooling which subsequently produces hypotension.
- Serum iron should be analysed in symptomatic patients. Serum iron levels (2-4 hrs post-ingestion) greater that 100 ug/dL indicate poisoning with levels, in excess of 350 ug/dL, being potentially serious. Emesis or lavage (for obtunded patients with no gag reflex)are the usual means of decontamination.
- Activated charcoal does not effectively bind iron.
- Catharsis (using sodium sulfate or magnesium sulfate) may only be used if the patient already has diarrhoea.
- Deferoxamine is a specific chelator of ferric (3+) iron and is currently the antidote of choice. It should be administered parenterally. [Ellenhorn and Barceloux: Medical Toxicology]

For acute or short term repeated exposures to dichromates and chromates:

- Absorption occurs from the alimentary tract and lungs.
- The kidney excretes about 60% of absorbed chromate within 8 hours of ingestion. Urinary excretion may take up to 14 days.
- Establish airway, breathing and circulation. Assist ventilation.
- Induce emesis with Ipecac Syrup if patient is not convulsing, in coma or obtunded and if the gag reflex is present.
- Otherwise use gastric lavage with endotracheal intubation.
- Fluid balance is critical. Peritoneal dialysis, haemodialysis or exchange transfusion may be effective although available data is limited.
- British Anti-Lewisite, ascorbic acid, folic acid and EDTA are probably not effective.
- There are no antidotes.
- Primary irritation, including chrome ulceration, may be treated with ointments comprising calcium-sodium-EDTA. This, together with the use of frequently renewed dressings, will ensure rapid healing of any ulcer which may develop.

The mechanism of action involves the reduction of Cr (VI) to Cr(III) and subsequent chelation; the irritant effect of Cr(III)/ protein complexes is thus avoided. [ILO Encyclopedia]

[Ellenhorn and Barceloux: Medical Toxicology]

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]

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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	None known.

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	▸ Non combustible. ▸ Not considered a significant fire risk, however containers may burn. Decomposition may produce toxic fumes of: sulfur oxides (SOx) silicon dioxide (SiO2) When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles. May emit poisonous fumes. May emit corrosive fumes.
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	▸ Remove all ignition sources. ▸ Clean up all spills immediately. ▸ Avoid contact with skin and eyes. ▸ Control personal contact with the substance, by using protective equipment. ▸ Use dry clean up procedures and avoid generating dust. ▸ Place in a suitable, labelled container for waste disposal.
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.

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.

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

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, acid chlorides, acid anhydrides and chloroformates. ▸ Avoid contact with copper, aluminium and their alloys.

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	portland cement	Portland cement	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	limestone	Calcium carbonate	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	gypsum	Calcium sulphate	10 mg/m3	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
limestone	Limestone; (Calcium carbonate; Dolomite)	45 mg/m3	500 mg/m3	3,000 mg/m3
limestone	Carbonic acid, calcium salt	45 mg/m3	210 mg/m3	1,300 mg/m3
gypsum	Calcium sulfate anhydrous; (Drierite; Gypsum; Plaster of Paris)	30 mg/m3	330 mg/m3	2,000 mg/m3

Ingredient	Original IDLH	Revised IDLH
portland cement	5,000 mg/m3	Not Available
blast furnace slag	Not Available	Not Available
fly ash - low quartz	Not Available	Not Available
limestone	Not Available	Not Available
gypsum	Not Available	Not Available

MATERIAL DATA

None assigned. Refer to individual constituents.

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

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

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.
An approved self contained breathing apparatus (SCBA) may be required in some situations.
Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Personal protection

Eye and face protection

- Chemical goggles.
- Full face shield may be required for supplementary but never for primary protection of eyes.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection

See Hand protection below

Hands/feet protection

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.

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	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 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. ▸ Protective gloves eg. Leather gloves or gloves with Leather facing ▸ 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	▸ Overalls. ▸ P.V.C. apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.

Respiratory protection

Type AX-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	AX P1 Air-line*	- -	AX PAPR-P1 -
up to 50 x ES	Air-line**	AX P2	AX PAPR-P2
up to 100 x ES	-	AX P3	-
		Air-line*	-
100+ x ES	-	Air-line**	AX 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 =

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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)

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	White/grey coloured free flowing fine powder; insoluble in water.

Physical state	Divided Solid	**Relative density (Water = 1)**	3.0-3.3
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	11-13 (wet cement)	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	1200	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Applicable
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	Inhalation of dusts, generated by the material during the course of normal handling, may be damaging to the health of the individual. Levels above 10 ug/m3 of suspended inorganic sulfates in the air may cause an excess risk of asthmatic attacks in susceptible persons 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.

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Ingestion	The material can produce chemical burns within the oral cavity and gastrointestinal tract following ingestion. Acute toxic responses to aluminium are confined to the more soluble forms.
Skin Contact	The material can produce chemical burns following direct contact with the skin. Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus. Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles. Four students received severe hand burns whilst making moulds of their hands with dental plaster substituted for Plaster of Paris. The dental plaster known as "Stone" was a special form of calcium sulfate hemihydrate containing alpha-hemihydrate crystals that provide high compression strength to the moulds. Beta-hemihydrate (normal Plaster of Paris) does not cause skin burns in similar circumstances. Handling wet cement can cause dermatitis. Cement when wet is quite alkaline and this alkali action on the skin contributes strongly to cement contact dermatitis since it may cause drying and defatting of the skin which is followed by hardening, cracking, lesions developing, possible infections of lesions and penetration by soluble salts. 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	The material can produce chemical burns to the eye following direct contact. Vapours or mists may be extremely irritating. 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	Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis. 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. Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals. Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminum oxide during abrasives production. Very fine Al2O3 powder was not fibrogenic in rats, guinea pigs, or hamsters when inhaled for 6 to 12 months and sacrificed at periods up to 12 months following the last exposure. When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C. The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis(aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity. Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction. There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveolii (sub 5 um) are able to produce pathogenic effects in the lungs. 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)

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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
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.
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*]

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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
Overexposure to respirable dust may cause coughing, wheezing, difficulty in breathing and impaired lung function. Chronic symptoms may include decreased vital lung capacity, chest infections
Repeated exposures, in an occupational setting, 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 (1/50,000 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 (exertional dyspnea), increased chest expansion, weakness and weight loss. As the disease progresses the cough produces a stringy mucous, vital capacity decreases further and shortness of breath becomes more severe. Other signs or symptoms include altered breath sounds, diminished lung capacity, diminished oxygen uptake during exercise, emphysema and pneumothorax (air in lung cavity) as a rare complication.
Removing workers from possibility of further exposure to dust generally leads to halting the progress of the lung abnormalities. Where worker-exposure potential is high, periodic examinations with emphasis on lung dysfunctions should be undertaken
Dust inhalation over an extended number of years may produce pneumoconiosis.. Pneumoconiosis is the accumulation of dusts in the lungs and the tissue reaction in its presence. It is further classified as being of noncollagenous or collagenous types. Noncollagenous pneumoconiosis, the benign form, is identified by minimal stromal reaction, consists mainly of reticulin fibres, an intact alveolar architecture and is potentially 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]
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

	TOXICITY	IRRITATION
108 20 CEMENT EASY MIX 20KG (Easy Mix GP Cement)	Not Available	Not Available
portland cement	Not Available	Not Available
blast furnace slag	dermal (rat) LD50: >4000 mg/kg[1]	Not Available

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fly ash - low quartz	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
limestone	**TOXICITY**	**IRRITATION**
	Oral (rat) LD50: 6450 mg/kg[2]	Skin (rabbit): 500 mg/24h-moderate
gypsum	**TOXICITY**	**IRRITATION**
	Oral (rat) LD50: >1581 mg/kg[1]	Not Available
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 tested.
BLAST FURNACE SLAG	For silica amorphous: When experimental animals inhale synthetic amorphous silica (SAS) dust, it dissolves in the lung fluid and is rapidly eliminated. If swallowed, the vast majority of SAS is excreted in the faeces and there is little accumulation in the body. Following absorption across the gut, SAS is eliminated via urine without modification in animals and humans. SAS is not expected to be broken down (metabolised) in mammals. After ingestion, there is limited accumulation of SAS in body tissues and rapid elimination occurs. Intestinal absorption has not been calculated, but appears to be insignificant in animals and humans. SASs injected subcutaneously are subjected to rapid dissolution and removal. There is no indication of metabolism of SAS in animals or humans based on chemical structure and available data. In contrast to crystalline silica, SAS is soluble in physiological media and the soluble chemical species that are formed are eliminated via the urinary tract without modification. Both the mammalian and environmental toxicology of SASs are significantly influenced by the physical and chemical properties, particularly those of solubility and particle size. SAS has no acute intrinsic toxicity by inhalation. Adverse effects, including suffocation, that have been reported were caused by the presence of high numbers of respirable particles generated to meet the required test atmosphere. These results are not representative of exposure to commercial SASs and should not be used for human risk assessment. Though repeated exposure of the skin may cause dryness and cracking, SAS is not a skin or eye irritant, and it is not a sensitiser. Repeated-dose and chronic toxicity studies confirm the absence of toxicity when SAS is swallowed or upon skin contact. Long-term inhalation of SAS caused some adverse effects in animals (increases in lung inflammation, cell injury and lung collagen content), all of which subsided after exposure. Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted with SAS 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. The difference in values may be explained by different particle size, and therefore the number of particles administered per unit dose. In general, as particle size decreases so does the NOAEL/LOAEL. Neither inhalation nor oral administration caused neoplasms (tumours). SAS is not mutagenic in vitro. No genotoxicity was detected in in vivo assays. SAS does not impair development of the foetus. Fertility was not specifically studied, but the reproductive organs in long-term studies were not affected. In humans, SAS is essentially non-toxic by mouth, skin or eyes, and by inhalation. Epidemiology studies show little evidence of adverse health effects due to SAS. Repeated exposure (without personal protection) may cause mechanical irritation of the eye and drying/cracking of the skin. There is no evidence of cancer or other long-term respiratory health effects (for example, silicosis) in workers employed in the manufacture of SAS. Respiratory symptoms in SAS workers have been shown to correlate with smoking but not with SAS exposure, while serial pulmonary function values and chest radiographs are not adversely affected by long-term exposure to SAS.
LIMESTONE	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. Eye (rabbit) 0.75: mg/24h - No evidence of carcinogenic properties. No evidence of mutagenic or teratogenic effects.
GYPSUM	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

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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 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 pneumoconiosis 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

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	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. 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 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; developmental effects were also not seen.
PORTLAND CEMENT & BLAST FURNACE SLAG & GYPSUM	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
PORTLAND CEMENT & BLAST FURNACE SLAG & FLY ASH - LOW QUARTZ & GYPSUM	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

108 20 CEMENT EASY MIX 20KG (Easy Mix GP Cement)

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

blast furnace slag

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	>100000mg/L	2
EC50	48	Crustacea	>100mg/L	2
EC50	72	Algae or other aquatic plants	>100mg/L	2
EC10	504	Crustacea	5-mg/L	2
NOEC	504	Crustacea	1-563mg/L	2

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fly ash - low quartz	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	NOEC	48	Fish	ca.700.0-2000mg/L	1
limestone	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>56000mg/L	4
	EC50	72	Algae or other aquatic plants	>14mg/L	2
	EC10	72	Algae or other aquatic plants	>14mg/L	2
	NOEC	72	Algae or other aquatic plants	14mg/L	2
gypsum	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>1-970mg/L	2
	EC50	72	Algae or other aquatic plants	>79mg/L	2
	EC0	96	Crustacea	=1255.000mg/L	1
	NOEC	504	Crustacea	360mg/L	4
Legend:	*Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*				

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
gypsum	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
gypsum	LOW (LogKOW = -2.2002)

Mobility in soil

Ingredient	Mobility
gypsum	LOW (KOC = 6.124)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Management Authority for disposal. ▸ Bury residue in an authorised landfill. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO Not Applicable
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

Continued...

SECTION 15 REGULATORY INFORMATION

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

PORTLAND CEMENT(65997-15-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Inventory of Chemical Substances (AICS)

BLAST FURNACE SLAG(65996-69-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

FLY ASH - LOW QUARTZ(68131-74-8.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

LIMESTONE(1317-65-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix F (Part 3)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

GYPSUM(13397-24-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Inventory of Chemical Substances (AICS)

National Inventory Status

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (blast furnace slag; portland cement; gypsum; fly ash - low quartz)
China - IECSC	No (blast furnace slag)
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (blast furnace slag; portland cement; fly ash - low quartz)
Korea - KECI	No (blast furnace slag)
New Zealand - NZIoC	Yes
Philippines - PICCS	No (blast furnace slag; portland cement)
USA - TSCA	Yes
Legend:	*Yes = All ingredients are on the inventory* *No = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 OTHER INFORMATION

Revision Date	09/05/2018
Initial Date	04/02/2013

SDS Version Summary

Version	Issue Date	Sections Updated
4.1.1.1	12/03/2013	Acute Health (eye), Acute Health (inhaled), Acute Health (skin), Acute Health (swallowed), Advice to Doctor, Chronic Health, Classification, Fire Fighter (fire/explosion hazard), First Aid (inhaled), First Aid (skin), First Aid (swallowed), Ingredients, Personal Protection (Respirator), Personal Protection (eye), Personal Protection (hands/feet), Spills (major)
5.1.1.1	09/05/2018	Classification

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.

Continued...

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
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