



RAPID REPAIR H

SUPER HIGH EARLY STRENGTH HORIZONTAL STRUCTURAL CONCRETE REPAIR MORTAR & CONCRETE FLOOR TOPPING.

Chemwatch Hazard Alert Code: 3

Chemwatch: 5253-37
Version No: 2.1.1.1
Safety Data Sheet according to HSNO Regulations

Issue Date: 14/06/2017
Print Date: 19/08/2017
L.GHS.NZL.EN

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

Product Identifier

Product name	Cemix Rapid Repair H
Synonyms	Not Available
Other means of identification	Not Available

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

Relevant identified uses	Structural repair mortar construction jobs.
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Details of the supplier of the safety data sheet

Registered company name	Cemix (a part of Ardex NZ)
Address	19 Alfred Street Onehunga Auckland 1061 New Zealand
Telephone	+64 9 636 1000
Fax	+64 9 636 0000
Website	www.cemix.co.nz
Email	Not Available

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	0800 ASK CEMIX
Other emergency telephone numbers	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

Considered a Hazardous Substance according to the criteria of the New Zealand Hazardous Substances New Organisms legislation. Not regulated for transport of Dangerous Goods.

Classification [1]	Acute Toxicity (Oral) Category 5, Acute Toxicity (Inhalation) Category 5, Skin Corrosion/Irritation Category 2, Serious Eye Damage Category 1, Skin Sensitizer Category 1, Carcinogenicity Category 1B, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Specific target organ toxicity - repeated exposure Category 1, Acute Aquatic Hazard Category 3, Chronic Aquatic Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI
Determined by Chemwatch using GHS/HSNO criteria	6.1E (inhalation), 6.1E (oral), 6.3A, 6.5B (contact), 6.7A, 6.9 (respiratory), 6.9A (inhalation), 8.3A, 9.1C, 9.1D

Label elements

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

H303	May be harmful if swallowed.
H333	May be harmful if inhaled
H315	Causes skin irritation.
H318	Causes serious eye damage.
H317	May cause an allergic skin reaction.
H350	May cause cancer.
H335	May cause respiratory irritation.
H372	Causes damage to organs through prolonged or repeated exposure.

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H412	Harmful to aquatic life with long lasting effects.
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Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P281	Use personal protective equipment as required.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
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 or doctor/physician.
P362	Take off contaminated clothing and wash before reuse.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P304+P312	IF INHALED: Call a POISON CENTER or doctor/physician if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
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.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.
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SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available	40-50	sand and aggregates
65997-15-1	35-45	<u>portland cement</u>
Not Available	0-10	silica as,
7631-86-9		<u>silica amorphous</u>
14808-60-7		<u>silica crystalline - quartz</u>
14464-46-1		<u>crystalite</u>
Not Available	0-10	alkali earth aluminate compound
Not Available	0-10	plasticisers
Not Available	0-30	Ingredients determined not to be hazardous

SECTION 4 FIRST AID MEASURES

NZ Poisons Centre 0800 POISON (0800 764 766) | NZ Emergency Services: 111

Description of first aid measures

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

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Ingestion	<ul style="list-style-type: none"> ▶ necessary. ▶ Transport to hospital, or doctor, without delay. <hr/> <ul style="list-style-type: none"> ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice.
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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 than 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]

- ▶ Manifestation of aluminium toxicity include hypercalcaemia, anaemia, Vitamin D refractory osteodystrophy and a progressive encephalopathy (mixed dysarthria-apraxia of speech, asterixis, tremulousness, myoclonus, dementia, focal seizures). Bone pain, pathological fractures and proximal myopathy can occur.
- ▶ Symptoms usually develop insidiously over months to years (in chronic renal failure patients) unless dietary aluminium loads are excessive.
- ▶ Serum aluminium levels above 60 ug/ml indicate increased absorption. Potential toxicity occurs above 100 ug/ml and clinical symptoms are present when levels exceed 200 ug/ml.
- ▶ Deferoxamine has been used to treat dialysis encephalopathy and osteomalacia. CaNa2EDTA is less effective in chelating aluminium.

[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, cricothyrotomy 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

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves.
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	<ul style="list-style-type: none"> ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Non combustible. ▶ Not considered a significant fire risk, however containers may burn. <p>Decomposes on heating and produces toxic fumes of:</p> <p>silicon dioxide (SiO₂)</p> <p>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.</p> <p>May emit poisonous fumes.</p> <p>May emit corrosive fumes.</p>

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none"> ▶ 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	<p>Moderate hazard.</p> <ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ DO NOT allow material to contact humans, exposed food or food utensils. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ Polyethylene or polypropylene container. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<p>For aluminas (aluminium oxide):</p> <p>Incompatible with hot chlorinated rubber.</p> <p>In the presence of chlorine trifluoride may react violently and ignite.</p> <p>-May initiate explosive polymerisation of olefin oxides including ethylene oxide.</p> <p>-Produces exothermic reaction above 200 C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals.</p> <p>-Produces exothermic reaction with oxygen difluoride.</p> <p>-May form explosive mixture with oxygen difluoride.</p>

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-Forms explosive mixtures with sodium nitrate.

-Reacts vigorously with vinyl acetate.

Aluminium oxide is an amphoteric substance, meaning it can react with both acids and bases, such as hydrofluoric acid and sodium hydroxide, acting as an acid with a base and a base with an acid, neutralising the other and producing a salt.

Calcium oxide:

- ▶ reacts violently with water, evolving high quantities of heat
- ▶ reacts violently, with possible ignition or explosion, with acids, anilinium perchlorate, bromine pentafluoride, chlorine trifluoride, fluorine, hydrogen fluoride, hydrazine, hydrogen sulfide, hydrogen trisulfide, isopropyl isocyanide dichloride, light metals, lithium, magnesium, powdered aluminium, phosphorus, potassium, sulfur trioxide
- ▶ increase the explosive sensitivity of azides, nitroalkanes (e.g. nitroethane, nitromethane, 1-nitropropane etc.)
- ▶ is incompatible with boric acid, boron trifluoride, carbon dioxide, ethanol, halogens (such as fluorine), metal halides, phosphorus pentoxide, selenium oxychloride, sulfur dioxide and many organic materials

Calcium sulfate:

- ▶ reacts violently with reducing agents, acrolein, alcohols, chlorine trifluoride, diazomethane, ethers, fluorine, hydrazine, hydrazinium perchlorate, hydrogen peroxide, finely divided aluminium or magnesium, peroxyfuroic acid, red phosphorus, sodium acetylde
- ▶ sensitises most organic azides which are unstable shock- and heat- sensitive explosives
- ▶ may form explosive materials with 1,3-di(5-tetrazolyl)triazene
- ▶ is incompatible with glycidol, isopropyl chlorocarbonate, nitrosyl perchlorate, sodium borohydride
- ▶ is hygroscopic; reacts with water to form gypsum and Plaster of Paris

For iron oxide (ferric oxide):

- ▶ Avoid storage with aluminium, calcium hypochlorite and ethylene oxide.
- ▶ Risk of explosion occurs following reaction with powdered aluminium, calcium silicide, ethylene oxide (polymerises), carbon monoxide, magnesium and perchlorates.
- ▶ Risk of ignition or formation of flammable gases or vapours occurs following reaction with carbides, for example caesium carbide, (produces heat), hydrogen sulfide, hydrogen peroxide (decomposes).
- ▶ An intimately powdered mixture with aluminium, usually ignited by magnesium ribbon, reacts with an intense exotherm to produce molten iron in the commercial "thermit" welding process
- ▶ WARNING: Avoid or control reaction with peroxides. All *transition metal* peroxides should be considered as potentially explosive. For example transition metal complexes of alkyl hydroperoxides may decompose explosively.
- ▶ The pi-complexes formed between chromium(0), vanadium(0) and other transition metals (haloarene-metal complexes) and mono-or poly-fluorobenzene show extreme sensitivity to heat and are explosive.
- ▶ Avoid reaction with borohydrides or cyanoborohydrides

Silicas:

- ▶ react with hydrofluoric acid to produce silicon tetrafluoride gas
- ▶ react with xenon hexafluoride to produce explosive xenon trioxide
- ▶ reacts exothermically with oxygen difluoride, and explosively with chlorine trifluoride (these halogenated materials are not commonplace industrial materials) and other fluorine-containing compounds
- ▶ may react with fluorine, chlorates
- ▶ are incompatible with strong oxidisers, manganese trioxide, chlorine trioxide, strong alkalis, metal oxides, concentrated orthophosphoric acid, vinyl acetate
- ▶ may react vigorously when heated with alkali carbonates.
- ▶ 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
New Zealand Workplace Exposure Standards (WES)	portland cement	Portland cement	10 mg/m ³	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	silica amorphous	Silica fume	2 mg/m ³	Not Available	Not Available	(r) - The value for respirable dust.
New Zealand Workplace Exposure Standards (WES)	silica amorphous	Silica fused	0.2 mg/m ³	Not Available	Not Available	(r) - The value for respirable dust.
New Zealand Workplace Exposure Standards (WES)	silica amorphous	Silica-Amorphous: Precipitated silica	10 mg/m ³	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	silica amorphous	Silica-Amorphous: Silica gel	10 mg/m ³	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	silica amorphous	Silica-Amorphous: Diatomaceous earth (not calcined)	10 mg/m ³	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	crystalite	Silica-Crystalline (all forms) quartz and cristobalite are confirmed carcinogens	0.1 mg/m ³	Not Available	Not Available	6.7A - Confirmed carcinogen; (r) - The value for respirable dust.

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
silica amorphous	Silica gel, amorphous synthetic	18 mg/m ³	200 mg/m ³	1,200 mg/m ³
silica amorphous	Silica, amorphous fumed	18 mg/m ³	100 mg/m ³	630 mg/m ³
silica amorphous	Siloxanes and silicones, dimethyl, reaction products with silica; (Hydrophobic silicon dioxide, amorphous)	120 mg/m ³	1,300 mg/m ³	7,900 mg/m ³
silica amorphous	Silica, amorphous fume	45 mg/m ³	500 mg/m ³	3,000 mg/m ³
silica amorphous	Silica amorphous hydrated	18 mg/m ³	220 mg/m ³	1,300 mg/m ³
silica crystalline - quartz	Silica, crystalline-quartz; (Silicon dioxide)	0.075 mg/m ³	33 mg/m ³	200 mg/m ³
cristobalite	Cristobalite	0.075 mg/m ³	33 mg/m ³	200 mg/m ³


Ingredient	Original IDLH	Revised IDLH
sand and aggregates	Not Available	Not Available

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portland cement	N.E. mg/m3 / N.E. ppm	5,000 mg/m3
silica as,	Not Available	Not Available
silica amorphous	N.E. mg/m3 / N.E. ppm	3,000 mg/m3
silica crystalline - quartz	N.E. mg/m3 / N.E. ppm	50 mg/m3
crystalite	N.E. mg/m3 / N.E. ppm	25 mg/m3
alkali earth aluminate compound	Not Available	Not Available
plasticisers	Not Available	Not Available
Ingredients determined not to be hazardous	Not Available	Not Available

MATERIAL DATA

Exposure controls

Appropriate engineering controls	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <ul style="list-style-type: none"> ▶ Local exhaust ventilation is required where solids are handled as powders or crystals; even when particulates are relatively large, a certain proportion will be powdered by mutual friction. ▶ Exhaust ventilation should be designed to prevent accumulation and recirculation of particulates in the workplace. ▶ If in spite of local exhaust an adverse concentration of the substance in air could occur, respiratory protection should be considered. Such protection might consist of: <ul style="list-style-type: none"> (a): particle dust respirators, if necessary, combined with an absorption cartridge; (b): filter respirators with absorption cartridge or canister of the right type; (c): fresh-air hoods or masks ▶ Build-up of electrostatic charge on the dust particle, 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. <p>Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to efficiently remove the contaminant.</p>															
	<table border="1"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>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 4-10 m/s (800-2000 f/min) for extraction of crusher dusts generated 2 metres 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.</p>	Type of Contaminant:	Air Speed:	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.)	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
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Personal protection																
Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] 															
Skin protection	See Hand protection below															
Hands/feet protection	<p>NOTE:</p> <ul style="list-style-type: none"> ▶ 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. <p>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.</p> <p>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.</p>															

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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 moisturizer 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.

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.
- ▶ fluorocautchouc.
- ▶ polyvinyl chloride.

Gloves should be examined for wear and/ or degradation constantly.

Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C. apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit.
Thermal hazards	Not Available

Respiratory protection

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

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

* - Negative pressure demand ** - Continuous flow

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Grey powder; insoluble in water.		
Physical state	Divided Solid	Relative density (Water = 1)	1.5-2.5
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	10-11 (wetted)	Decomposition temperature	Not Applicable
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Applicable
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	>350	Taste	Not Available

Continued...

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Evaporation rate	Not Applicable	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 Applicable
Vapour pressure (kPa)	Not Applicable	Gas group	Not Available
Solubility in water (g/L)	Immiscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	Not Applicable	VOC g/L	Not Applicable

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	<p>Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Inhalation may result in chrome ulcers or sores of nasal mucosa and lung damage.</p> <p>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.</p> <p>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.</p> <p>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.</p>
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	<p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus.</p> <p>Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	<p>On the basis of epidemiological data, it has been concluded that prolonged inhalation of the material, in an occupational setting, may produce cancer in humans.</p> <p>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.</p> <p>Toxic: danger of serious damage to health by prolonged exposure through inhalation.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers.</p> <p>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.</p> <p>The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis(aluminosis) in experimental</p>

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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 alveoli (sub 5 µm) 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)

In an inhalation study in rats no increase in tumour incidence was observed but the number of fibres with lengths exceeding 5 µm and a diameter of less than 3 µm 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 µm in length and less than 0.5 µm in diameter.

In two studies by intraperitoneal injection in rats using wollastonite with median fibre lengths of 8.1 µm and 5.6 µm 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

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

Chronic symptoms produced by crystalline silicas included decreased vital lung capacity and chest infections. Lengthy exposure may cause silicosis a disabling form of pneumoconiosis which may lead to fibrosis, a scarring of the lining of the air sacs in the lung. Symptoms may appear 8 to 18 months after initial exposure. Smoking increases this risk. Classic silicosis is a chronic disease characterised by the formation of scattered, rounded or stellate silica-containing nodules of scar tissue in the lungs ranging from microscopic to 1.0 cm or more. The nodules isolate the inhaled silica particles and protect the surrounding normal and functioning tissue from continuing injury. Simple silicosis (in which the nodules are less than 1.0 cm in diameter) is generally asymptomatic but may be slowly progressive even in the absence of continuing exposure. Simple silicosis can develop in complicated silicoses (in which nodules are greater than 1.0 cm in diameter) and can produce disabilities including an associated tuberculous infection (which 50 years ago accounted for 75% of the deaths among silicotic workers). Crystalline silica deposited in the lungs causes epithelial and macrophage injury and activation. Crystalline silica translocates to the interstitium and the regional lymph nodes and cause the recruitment of inflammatory cells in a dose dependent manner. In humans, a large fraction of crystalline silica persists in the lungs. The question of potential carcinogenicity associated with chronic inhalation of crystalline silica remains equivocal with some studies supporting the proposition and others finding no significant association. The results of recent epidemiological studies suggest that lung cancer risk is elevated only in those patients with overt silicosis. A relatively large number of epidemiological studies have been undertaken and in some, increased risk gradients have been observed in relation to dose surrogates - cumulative exposure, duration of exposure, the presence of radiographically defined silicosis, and peak intensity exposure. Chronic inhalation in rats by single or repeated intratracheal instillation produced a significant increase in the incidences of adenocarcinomas and squamous cell carcinomas of the lung. Lifetime inhalation of crystalline silica (87% alpha-quartz) at 1 mg/m³ (74% respirable) by rats, produced an increase in animals with keratinising cystic squamous cell tumours, adenomas, adenocarcinomas, adenosquamous cell carcinomas, squamous cell carcinoma and nodular bronchiolar alveolar hyperplasia accompanied by extensive subpleural and peribronchiolar fibrosis, increased pulmonary collagen content, focal lipoproteinosis and macrophage infiltration. Thoracic and abdominal malignant lymphomas developed in rats after single intrapleural and intraperitoneal injection of suspensions of several types of quartz.

Some studies show excess numbers of cases of scleroderma, connective tissue disorders, lupus, rheumatoid arthritis chronic kidney diseases, and end-stage kidney disease in workers

NOTE: Some jurisdictions require health surveillance be conducted on workers occupationally exposed to silica, crystalline. Such surveillance should emphasise

- ▶ demography, occupational and medical history and health advice
- ▶ standardised respiratory function tests such as FEV₁, FVC and FEV₁/FVC
- ▶ standardised respiratory function tests such as FV₁, FVC and FEV₁/FVC
- ▶ chest X-ray, full size PA view
- ▶ records of personal exposure

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

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	<p>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.</p> <p>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 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]</p> <p>Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.</p>
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	TOXICITY	IRRITATION
Cemix Rapid Repair H	Dermal (Rat) LD50: >5000 mg/kg ^[2]	Not Available
	Inhalation (Rat) LC50: >5 mg/l (dust/mist) ^{*[2]}	
	Oral (Rat) LD50: >5000 mg/kg ^{*[2]}	
portland cement	TOXICITY	IRRITATION
	Not Available	Not Available
silica amorphous	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^{*[2]}	Eye (rabbit): non-irritating *
	Inhalation (rat) LC50: >0.139 mg/l/14hr ^{**[2]}	Skin (rabbit): non-irritating *
silica crystalline - quartz	TOXICITY	IRRITATION
	Not Available	Not Available
cristobalite	TOXICITY	IRRITATION
	Not Available	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

	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>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.</p>
PORTLAND CEMENT	<p>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. No significant acute toxicological data identified in literature search.</p>
SILICA AMORPHOUS	<p>For silica amorphous:</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>Repeated-dose and chronic toxicity studies confirm the absence of toxicity when SAS is swallowed or upon skin contact.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>There is no evidence of cancer or other long-term respiratory health effects (for example, silicosis) in workers employed in the manufacture of SAS.</p> <p>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.</p> <p>The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans.</p>

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	Evidence of carcinogenicity may be inadequate or limited in animal testing. Reports indicate high/prolonged exposures to amorphous silicas induced lung fibrosis in experimental animals; in some experiments these effects were reversible. [PATTYS]
CRISTOBALITE	Inhalation (human) TCLo: 16 mppcf*/8H/17.9y-l * Millions of particles per cubic foot
SILICA CRYSTALLINE - QUARTZ & CRISTOBALITE	WARNING: For inhalation exposure <u>ONLY</u> : This substance has been classified by the IARC as Group 1: CARCINOGENIC TO HUMANS
SILICA CRYSTALLINE - QUARTZ & CRISTOBALITE	The International Agency for Research on Cancer (IARC) has classified occupational exposures to respirable (<5 µm) crystalline silica as being carcinogenic to humans. This classification is based on what IARC considered sufficient evidence from epidemiological studies of humans for the carcinogenicity of inhaled silica in the forms of quartz and cristobalite. Crystalline silica is also known to cause silicosis, a non-cancerous lung disease. Intermittent exposure produces; focal fibrosis, (pneumoconiosis), cough, dyspnoea, liver tumours. * Millions of particles per cubic foot (based on impinger samples counted by light field techniques). NOTE : the physical nature of quartz in the product determines whether it is likely to present a chronic health problem. To be a hazard the material must enter the breathing zone as respirable particles.

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 available but does not fill the criteria for classification
✓ – Data available to make classification
⊘ – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Cemix Rapid Repair H	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
silica amorphous	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	ca.2000mg/L	1
	EC50	48	Crustacea	ca.7600mg/L	1
	EC50	72	Algae or other aquatic plants	440mg/L	1
	EC10	72	Algae or other aquatic plants	140mg/L	1
NOEC	72	Algae or other aquatic plants	60mg/L	1	
silica crystalline - quartz	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
cristobalite	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available

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.

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
silica amorphous	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
silica amorphous	LOW (LogKOW = 0.5294)

Mobility in soil

Ingredient	Mobility
silica amorphous	LOW (KOC = 23.74)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product. <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. In most instances the supplier of the material should be consulted.</p> <ul style="list-style-type: none"> ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority. ▶ Recycle wherever possible. ▶ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▶ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material) ▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.
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Ensure that the disposal of material is carried out in accordance with Hazardous Substances (Disposal) Regulations 2001.

SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (UN): 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

SECTION 15 REGULATORY INFORMATION

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

This substance is to be managed using the conditions specified in an applicable Group Standard

HSR Number	Group Standard
HSR002545	Construction Products (Toxic [6.7A]) Group Standard 2006

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

New Zealand Inventory of Chemicals (NZIoC)

New Zealand Workplace Exposure Standards (WES)

SILICA AMORPHOUS(7631-86-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

New Zealand Inventory of Chemicals (NZIoC)

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Workplace Exposure Standards (WES)

SILICA CRYSTALLINE - QUARTZ(14808-60-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

New Zealand Inventory of Chemicals (NZIoC)

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Workplace Exposure Standards (WES)

CRISTOBALITE(14464-46-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Workplace Exposure Standards (WES)

New Zealand Inventory of Chemicals (NZIoC)

Location Test Certificate

Subject to Regulation 55 of the Hazardous Substances (Classes 1 to 5 Controls) Regulations, a location test certificate is required when quantity greater than or equal to those indicated below are present.

Hazard Class	Quantity beyond which controls apply for closed containers	Quantity beyond which controls apply when use occurring in open containers
Not Applicable	Not Applicable	Not Applicable

Approved Handler

Subject to Regulation 56 of the Hazardous Substances (Classes 1 to 5 Controls) Regulations and Regulation 9 of the Hazardous Substances (Classes 6, 8, and 9 Controls) Regulations, the substance must be under the personal control of an Approved Handler when present in a quantity greater than or equal to those indicated below.

Class of substance	Quantities
6.7A	10 kg or more, if solid 10 L or more, if liquid

Refer Group Standards for further information

Tracking Requirements

Not Applicable

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (portland cement; silica crystalline - quartz; cristobalite)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	N (portland cement; silica amorphous; silica crystalline - quartz)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	N (portland cement)
USA - TSCA	Y
Legend:	Y = All ingredients are on the inventory N = 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

Other information

Ingredients with multiple cas numbers

Name	CAS No
silica amorphous	7631-86-9, 112945-52-5, 67762-90-7, 68611-44-9, 68909-20-6, 112926-00-8, 61790-53-2, 60676-86-0, 91053-39-3, 69012-64-2, 844491-94-7
silica crystalline - quartz	14808-60-7, 122304-48-7, 122304-49-8, 12425-26-2, 1317-79-9, 70594-95-5, 87347-84-0, 308075-07-2

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
 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

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