

Overcrete Complete Resurfacing Compound



Overcrete Complete Resurfacing Compound

On-Crete Australia Pty Ltd

Chemwatch Hazard Alert Code: 4

Version No: 1.2

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	Overcrete Complete Resurfacing Compound
Synonyms	Not Available
Other means of identification	Not Available

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

Relevant identified uses	Overcrete Resurfacing Compound is a dry powder component of a 3 part system specially designed to rejuvenate existing concrete.
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Details of the supplier of the safety data sheet

Registered company name	On-Crete Australia Pty Ltd	Oncrete New Zealand Pty Ltd
Address	4/489 Scottsdale Drive Queensland 4227 Australia	15A Vega Place, Rosedale Auckland 0632 New Zealand
Telephone	+617 5593 6884	+649 930 8829
Fax	Not Available	Not Available
Website	www.on-crete.com.au	www.oncrete.co.nz
Email	sales@on-crete.com.au	sales@oncrete.co.nz

Emergency telephone number

Association / Organisation	On-Crete Australia Pty Ltd	Oncrete New Zealand Pty Ltd
Emergency telephone numbers	1300 292 504	0800 120 854
Other emergency telephone numbers	+613 6121 9073	+649 553 8356

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

CHEMWATCH HAZARD RATINGS

	Min	Max
Flammability	0	
Toxicity	0	
Body Contact	3	
Reactivity	0	
Chronic	4	

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

Poisons Schedule	Not Applicable
Classification ^[1]	Specific target organ toxicity - repeated exposure Category 2, Serious Eye Damage Category 1, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Skin Corrosion/Irritation Category 2, Carcinogenicity Category 1A, Skin Sensitizer Category 1, Germ cell mutagenicity Category 2

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

Label elements

Hazard pictogram(s)	
SIGNAL WORD	DANGER

Hazard statement(s)

H373	May cause damage to organs through prolonged or repeated exposure. (Respiratory system) (Inhalation)
H318	Causes serious eye damage.
H335	May cause respiratory irritation.
H315	Causes skin irritation.
H350	May cause cancer.
H317	May cause an allergic skin reaction.
H341	Suspected of causing genetic defects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume.
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.
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.
P321	Specific treatment (see advice on this label).
P362	Take off contaminated clothing and wash before reuse.
P302+P352	IF ON SKIN: Wash with plenty of water.
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 to authorised hazardous or special waste collection point in accordance with any local regulation.
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SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
65997-15-1	30-60	<u>portland cement</u>
14808-60-7*	<10	<u>silica crystalline - quartz</u>
1317-65-3	<10	<u>limestone</u>

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13397-24-5	<10	<u>gypsum</u>
1305-78-8	<10	<u>calcium oxide</u>
65997-16-2	<10	<u>calcium aluminate cement</u>

SECTION 4 FIRST AID MEASURES

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 or hair contact occurs:</p> <ul style="list-style-type: none"> ▶ 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. <p>For thermal burns:</p> <ul style="list-style-type: none"> ▶ Decontaminate area around burn. ▶ Consider the use of cold packs and topical antibiotics. <p>For first-degree burns (affecting top layer of skin)</p> <ul style="list-style-type: none"> ▶ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides. ▶ Use compresses if running water is not available. ▶ Cover with sterile non-adhesive bandage or clean cloth. ▶ Do NOT apply butter or ointments; this may cause infection. ▶ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur. <p>For second-degree burns (affecting top two layers of skin)</p> <ul style="list-style-type: none"> ▶ Cool the burn by immerse in cold running water for 10-15 minutes. ▶ Use compresses if running water is not available. ▶ Do NOT apply ice as this may lower body temperature and cause further damage. ▶ Do NOT break blisters or apply butter or ointments; this may cause infection. ▶ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape. <p>To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort):</p> <ul style="list-style-type: none"> ▶ Lay the person flat. ▶ Elevate feet about 12 inches. ▶ Elevate burn area above heart level, if possible. ▶ Cover the person with coat or blanket. ▶ Seek medical assistance. <p>For third-degree burns</p> <p>Seek immediate medical or emergency assistance.</p> <p>In the mean time:</p> <ul style="list-style-type: none"> ▶ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound. ▶ Separate burned toes and fingers with dry, sterile dressings. ▶ Do not soak burn in water or apply ointments or butter; this may cause infection. ▶ To prevent shock see above. ▶ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. ▶ Have a person with a facial burn sit up. ▶ Check pulse and breathing to monitor for shock until emergency help arrives.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay.
Ingestion	<ul style="list-style-type: none"> ▶ Immediately give a glass of water. ▶ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

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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, cricothyroidotomy or tracheotomy may be necessary.
- ▶ Oxygen is given as indicated.
- ▶ The presence of shock suggests perforation and mandates an intravenous line and fluid administration.
- ▶ Damage due to alkaline corrosives occurs by liquefaction necrosis whereby the saponification of fats and solubilisation of proteins allow deep penetration into the tissue.

Alkalis continue to cause damage after exposure.

INGESTION:

- ▶ Milk and water are the preferred diluents

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

* Neutralising agents should never be given since exothermic heat reaction may compound injury.

* Catharsis and emesis are absolutely contra-indicated.

* Activated charcoal does not absorb alkali.

* Gastric lavage should not be used.

Supportive care involves the following:

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

SKIN AND EYE:

- ▶ Injury should be irrigated for 20-30 minutes.

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

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

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

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Special hazards arising from the substrate or mixture

Fire Incompatibility	<ul style="list-style-type: none"> ▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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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. ▶ 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"> ▶ Combustible solid which burns but propagates flame with difficulty; it is estimated that most organic dusts are combustible (circa 70%) - according to the circumstances under which the combustion process occurs, such materials may cause fires and / or dust explosions. ▶ Organic powders when finely divided over a range of concentrations regardless of particulate size or shape and suspended in air or some other oxidizing medium may form explosive dust-air mixtures and result in a fire or dust explosion (including secondary explosions). ▶ Avoid generating dust, particularly clouds of dust in a confined or unventilated space as dusts may form an explosive mixture with air, and any source of ignition, i.e. flame or spark, will cause fire or explosion. Dust clouds generated by the fine grinding of the solid are a particular hazard; accumulations of fine dust (420 micron or less) may burn rapidly and fiercely if ignited - particles exceeding this limit will generally not form flammable dust clouds; once initiated, however, larger particles up to 1400 microns diameter will contribute to the propagation of an explosion. ▶ In the same way as gases and vapours, dusts in the form of a cloud are only ignitable over a range of concentrations; in principle, the concepts of lower explosive limit (LEL) and upper explosive limit (UEL) are applicable to dust clouds but only the LEL is of practical use; - this is because of the inherent difficulty of achieving homogeneous dust clouds at high temperatures (for dusts the LEL is often called the "Minimum Explosible Concentration", MEC). ▶ When processed with flammable liquids/vapors/mists, ignitable (hybrid) mixtures may be formed with combustible dusts. Ignitable mixtures will increase the rate of explosion pressure rise and the Minimum Ignition Energy (the minimum amount of energy required to ignite dust clouds - MIE) will be lower than the pure dust in air mixture. The Lower Explosive Limit (LEL) of the vapour/dust mixture will be lower than the individual LELs for the vapors/mists or dusts. ▶ A dust explosion may release of large quantities of gaseous products; this in turn creates a subsequent pressure rise of explosive force capable of damaging plant and buildings and injuring people. ▶ Usually the initial or primary explosion takes place in a confined space such as plant or machinery, and can be of sufficient force to damage or rupture the plant. If the shock wave from the primary explosion enters the surrounding area, it will disturb any settled dust layers, forming a second dust cloud, and often initiate a much larger secondary explosion. All large scale explosions have resulted from chain reactions of this type. ▶ Dry dust can be charged electrostatically by turbulence, pneumatic transport, pouring, in exhaust ducts and during transport. ▶ Build-up of electrostatic charge may be prevented by bonding and grounding. ▶ Powder handling equipment such as dust collectors, dryers and mills may require additional protection measures such as explosion venting. ▶ All movable parts coming in contact with this material should have a speed of less than 1-meter/sec. ▶ A sudden release of statically charged materials from storage or process equipment, particularly at elevated temperatures and/ or pressure, may result in ignition especially in the absence of an apparent ignition source. ▶ One important effect of the particulate nature of powders is that the surface area and surface structure (and often moisture content) can vary widely from sample to sample, depending of how the powder was manufactured and handled; this means that it is virtually impossible to use flammability data published in the literature for dusts (in contrast to that published for gases and vapours). ▶ Autoignition temperatures are often quoted for dust clouds (minimum ignition temperature (MIT)) and dust layers (layer ignition temperature (LIT)); LIT generally falls as the thickness of the layer increases. <p>Combustion products include: carbon monoxide (CO) carbon dioxide (CO₂) silicon dioxide (SiO₂) metal oxides other pyrolysis products typical of burning organic material.</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. May emit corrosive fumes.</p>
HAZCHEM	Not Applicable

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

See section 8

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Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none"> ▶ Clean up waste regularly and abnormal spills immediately. ▶ Avoid breathing dust and contact with skin and eyes. ▶ Wear protective clothing, gloves, safety glasses and dust respirator. ▶ Use dry clean up procedures and avoid generating dust. ▶ Vacuum up or sweep up. NOTE: Vacuum cleaner must be fitted with an exhaust micro filter (HEPA type) (consider explosion-proof machines designed to be grounded during storage and use). ▶ Dampen with water to prevent dusting before sweeping. ▶ Place in suitable containers for disposal.
Major Spills	<ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear full body protective clothing with breathing apparatus. ▶ Prevent, by all means available, spillage from entering drains or water courses. ▶ Consider evacuation (or protect in place). ▶ No smoking, naked lights or ignition sources. ▶ Increase ventilation. ▶ Stop leak if safe to do so. ▶ Water spray or fog may be used to disperse / absorb vapour. ▶ Contain or absorb spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<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. ▶ Organic powders when finely divided over a range of concentrations regardless of particulate size or shape and suspended in air or some other oxidizing medium may form explosive dust-air mixtures and result in a fire or dust explosion (including secondary explosions) ▶ Minimise airborne dust and eliminate all ignition sources. Keep away from heat, hot surfaces, sparks, and flame. ▶ Establish good housekeeping practices. ▶ Remove dust accumulations on a regular basis by vacuuming or gentle sweeping to avoid creating dust clouds. ▶ Use continuous suction at points of dust generation to capture and minimise the accumulation of dusts. Particular attention should be given to overhead and hidden horizontal surfaces to minimise the probability of a "secondary" explosion. According to NFPA Standard 654, dust layers 1/32 in.(0.8 mm) thick can be sufficient to warrant immediate cleaning of the area. ▶ Do not use air hoses for cleaning. ▶ Minimise dry sweeping to avoid generation of dust clouds. Vacuum dust-accumulating surfaces and remove to a chemical disposal area. Vacuums with explosion-proof motors should be used. ▶ Control sources of static electricity. Dusts or their packages may accumulate static charges, and static discharge can be a source of ignition. ▶ Solids handling systems must be designed in accordance with applicable standards (e.g. NFPA including 654 and 77) and other national guidance.
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	<ul style="list-style-type: none"> Do not empty directly into flammable solvents or in the presence of flammable vapors. The operator, the packaging container and all equipment must be grounded with electrical bonding and grounding systems. Plastic bags and plastics cannot be grounded, and antistatic bags do not completely protect against development of static charges. <p>Empty containers may contain residual dust which has the potential to accumulate following settling. Such dusts may explode in the presence of an appropriate ignition source.</p> <ul style="list-style-type: none"> Do NOT cut, drill, grind or weld such containers. In addition ensure such activity is not performed near full, partially empty or empty containers without appropriate workplace safety authorisation or permit.
Other information	<ul style="list-style-type: none"> Store in original containers. Keep containers securely sealed. Store in a cool, dry area protected from environmental extremes. Store away from incompatible materials and foodstuff containers. Protect containers against physical damage and check regularly for leaks. Observe manufacturer's storage and handling recommendations contained within this SDS. <p>For major quantities:</p> <ul style="list-style-type: none"> Consider storage in banded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams). Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities.

Conditions for safe storage, including any incompatibilities

Suitable container	<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> <ul style="list-style-type: none"> May initiate explosive polymerisation of olefin oxides including ethylene oxide. Produces exothermic reaction above 200 C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals. Produces exothermic reaction with oxygen difluoride. May form explosive mixture with oxygen difluoride. Forms explosive mixtures with sodium nitrate. Reacts vigorously with vinyl acetate. <p>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.</p> <p>Calcium oxide:</p> <ul style="list-style-type: none"> 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 <p>Calcium sulfate:</p> <ul style="list-style-type: none"> 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 acetylide 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 <p>For iron oxide (ferric oxide):</p> <ul style="list-style-type: none"> 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 <i>transition metal</i> 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 <p>Formaldehyde:</p> <ul style="list-style-type: none"> is a strong reducing agent may polymerise in air unless properly inhibited (usually with methanol up to 15%) and stored at controlled temperatures will polymerize with active organic material such as phenol

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- reacts violently with strong oxidisers, hydrogen peroxide, potassium permanganate, acrylonitrile, caustics (sodium hydroxide, yielding formic acid and flammable hydrogen), magnesium carbonate, nitromethane, nitrogen oxides (especially at elevated temperatures), peroxyformic acid
- is incompatible with strong acids (hydrochloric acid forms carcinogenic bis(chloromethyl)ether*), amines, ammonia, aniline, bisulfides, gelatin, iodine, magnesite, phenol, some monomers, tannins, salts of copper, iron, silver.
- acid catalysis can produce impurities: methylal, methyl formate

Aqueous solutions of formaldehyde:

- slowly oxidise in air to produce formic acid
- attack carbon steel

Concentrated solutions containing formaldehyde are:

- unstable, both oxidising slowly to form formic acid and polymerising; in dilute aqueous solutions formaldehyde appears as monomeric hydrate (methylene glycol) - the more concentrated the solution the more polyoxymethylene glycol occurs as oligomers and polymers (methanol and amine-containing compounds inhibit polymer formation)
- readily subject to polymerisation, at room temperature, in the presence of air and moisture, to form paraformaldehyde (8-100 units of formaldehyde), a solid mixture of linear polyoxymethylene glycols containing 90-99% formaldehyde; a cyclic trimer, trioxane (CH₂O₃), may also form

Flammable and/or toxic gases are generated by the combination of aldehydes with azo, diazo compounds, dithiocarbamates, nitrides, and strong reducing agents

*The empirical equation may be used to determine the concentration of bis(chloromethyl)ether (BCME) formed by reaction with HCl:

$$\log(\text{BCME})\text{ppb} = -2.25 + 0.67 \cdot \log(\text{HCHO})\text{ppm} + 0.77 \cdot \log(\text{HCl})\text{ppm}$$

Assume values for formaldehyde, in air, of 1 ppm and for HCl of 5 ppm, resulting BCME concentration, in air, would be 0.02 ppb.

Cellulose and its derivatives may react vigorously with calcium oxide, bleaching powder, perchlorates, perchloric acid, sodium chlorate, fluorine, nitric acid, sodium nitrate and sodium nitrite.

May be incompatible with aminacrine hydrochloride, chlorocresol, mercuric chloride, phenol, resorcinol, tannic acid and silver nitrate.

- Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.
- Avoid contact with copper, aluminium and their alloys.
- Avoid reaction with oxidising agents



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X — Must not be stored together

0 — May be stored together with specific precautions

+ — May be stored together

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/m ³	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica crystalline - quartz	Silica - Crystalline: Quartz (respirable dust)	0.05 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	limestone	Calcium carbonate	10 mg/m ³	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	gypsum	Calcium sulphate	10 mg/m ³	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	calcium oxide	Calcium oxide	2 mg/m ³	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
silica crystalline - quartz	Silica, crystalline-quartz; (Silicon dioxide)	0.075 mg/m ³	33 mg/m ³	200 mg/m ³
limestone	Carbonic acid, calcium salt	45 mg/m ³	210 mg/m ³	1,300 mg/m ³
calcium oxide	Calcium oxide	6 mg/m ³	110 mg/m ³	660 mg/m ³

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Overcrete Complete Resurfacing Compound

Ingredient	Original IDLH	Revised IDLH
portland cement	5,000 mg/m ³	Not Available
silica crystalline - quartz	25 mg/m ³ / 50 mg/m ³	Not Available
limestone	Not Available	Not Available
gypsum	Not Available	Not Available
calcium oxide	25 mg/m ³	Not Available
calcium aluminate cement	Not Available	Not Available

OCCUPATIONAL EXPOSURE BANDING

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
calcium aluminate cement	E	≤ 0.01 mg/m ³
Notes:	Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.	

MATERIAL DATA

for calcium silicate:

containing no asbestos and <1% crystalline silica

ES TWA: 10 mg/m³ inspirable dust

TLV TWA: 10 mg/m³ total dust (synthetic nonfibrous) A4

Although in vitro studies indicate that calcium silicate is more toxic than substances described as "nuisance dusts" is thought that adverse health effects which might occur following exposure to 10-20 mg/m³ are likely to be minimal. The TLV-TWA is thought to be protective against the physical risk of eye and upper respiratory tract irritation in workers and to prevent interference with vision and deposition of particulate in the eyes, ears, nose and mouth.

NOTE: This substance has been classified by the ACGIH as A4 **NOT** classifiable as causing Cancer in humans

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

The International Agency for Research on Cancer (IARC) has classified occupational exposures to **respirable** (<5 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.

For calcium oxide:

The TLV-TWA is thought to be protective against undue irritation and is analogous to that recommended for sodium hydroxide.

For aluminium oxide:

The experimental and clinical data indicate that aluminium oxide acts as an "inert" material when inhaled and seems to have little effect on the lungs nor does it produce significant organic disease or toxic effects when exposures are kept under reasonable control.

[Documentation of the Threshold Limit Values], ACGIH, Sixth Edition

Cellulose is considered a nuisance dust which has little adverse effect on lung and does not produce significant organic disease or toxic effects when appropriate controls are applied.

The concentration of dust, for application of respirable dust limits, is to be determined from the fraction that penetrates a separator whose size collection efficiency is described by a cumulative log-normal function with a median aerodynamic diameter of 4.0 µm (+-) 0.3 µm and with a geometric standard deviation of 1.5 µm (+-) 0.1 µm, i.e..generally less than 5 µm.

For amorphous crystalline silica (precipitated silicic acid):

Amorphous crystalline silica shows little potential for producing adverse effects on the lung and exposure standards should reflect a particulate of low intrinsic toxicity. Mixtures of amorphous silicas/ diatomaceous earth and crystalline silica should be monitored as if they comprise only the crystalline forms.

The dusts from precipitated silica and silica gel produce little adverse effect on pulmonary functions and are not known to produce significant disease or toxic effect.

IARC has classified silica, amorphous as Group 3: **NOT** classifiable as to its carcinogenicity to humans.

Evidence of carcinogenicity may be inadequate or limited in animal testing.

for formaldehyde:

Odour Threshold Value for formaldehyde: 0.98 ppm (recognition)

NOTE: Detector tubes for formaldehyde, measuring in excess of 0.2 ppm are available commercially.

Formaldehyde vapour exposure:

Primary irritation is dependent on duration of exposure and individual susceptibility.

The following are typical symptoms encountered at various exposure levels.

0.1 ppm - Lower level of mucous eye, nose and throat irritation

0.8 ppm - Typical threshold of perception

1-2 ppm - Typical threshold of irritation

2-3 ppm - Irritation of eyes, nose and throat

4-5 ppm - Increased irritation, tearing, headache, pungent odour

10-20 ppm - Profuse tearing, severe burning, coughing

50 ppm - Serious bronchial and alveolar damage

Continued...

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100 ppm - Formaldehyde induced chemical pneumonia and death

Despite the intent of the TLV Ceiling recommendation it is believed that 0.3 ppm will not protect that portion of the workforce (up to 20%) reported to be responsive to low ambient concentrations. Because of the dose-related carcinogenic activity for rat and mouse inhalation of formaldehyde, the report of macromolecular adducts in the upper and lower respiratory tracts of nonhuman primates following inhalation of formaldehyde, the human case reports of upper respiratory tract malignant melanoma associated with

formaldehyde inhalation and the suggestive epidemiologic data on human cancer risk, the TLV Committee recommends that workplace formaldehyde air concentrations be reduced to the lowest possible levels that can be achieved using engineering controls.

Odour Safety Factor(OSF)

OSF=0.36 (FORMALDEHYDE)

NOTE D: Certain substances which are susceptible to spontaneous polymerisation or decomposition are generally placed on the market in a stabilised form. It is in this form that they are listed on Annex I

When they are placed on the market in a non-stabilised form, the label must state the name of the substance followed by the words "non-stabilised"


European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

NOTE H: Special requirements exist in relation to classification and labelling of this substance. This note applies to certain coal- and oil -derived substances and to certain entries for groups of substances in Annex VI. European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

NOTE L: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 3% DMSO extract as measured by IP 346.

European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

Exposure controls

<p>Appropriate engineering controls</p>	<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.</p> <p>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. Employers may need to use multiple types of controls to prevent employee overexposure.</p> <ul style="list-style-type: none"> ▶ Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area. ▶ Work should be undertaken in an isolated system such as a "glove-box" . Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system. ▶ Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within. ▶ Open-vessel systems are prohibited. ▶ Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation. ▶ Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system. ▶ For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▶ Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas). ▶ Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air. ▶ Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed.
<p>Personal protection</p>	
<p>Eye and face protection</p>	<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]
<p>Skin protection</p>	<p>See Hand protection below</p>

Continued...

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NOTE:

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

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.

The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.

Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

- frequency and duration of contact,
- chemical resistance of glove material,
- glove thickness and
- dexterity

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

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

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

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

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

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

Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.

Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:

- Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.
- Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential

Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

- ▶ Neoprene rubber gloves

Experience indicates that the following polymers are suitable as glove materials for protection against undissolved, dry solids, where abrasive particles are not present.

- ▶ polychloroprene.
- ▶ nitrile rubber.
- ▶ butyl rubber.
- ▶ fluorocautchouc.
- ▶ polyvinyl chloride.

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

Hands/feet protection**Body protection**

See Other protection below

Other protection

- ▶ Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent]
- ▶ Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent]
- ▶ Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely.
- ▶ Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood.
- ▶ Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood.

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- ▶ Overalls.
- ▶ P.V.C. apron.
- ▶ Barrier cream.
- ▶ Skin cleansing cream.
- ▶ Eye wash unit.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:

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Material	CPI
BUTYL	A
NEOPRENE	A
NEOPRENE/NATURAL	A
NITRILE	A
PE	A
PE/EVAL/PE	A
PVC	A
TEFLON	A
VITON	A
NATURAL RUBBER	B
NATURAL+NEOPRENE	B

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Not Available		
Physical state	Solid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Available
Flash point (°C)	Not Available	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Available	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Applicable

Continued...

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Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Not Applicable	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	<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>Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation.</p> <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 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> <p>Inhalation of dusts, generated by the material during the course of normal handling, may be damaging to the health of the individual.</p>
Ingestion	<p>Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing.</p> <p>The material has NOT been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.</p> <p>Not normally a hazard due to the physical form of product. The material is a physical irritant to the gastro-intestinal tract. Accidental ingestion of the material may be damaging to the health of the individual.</p>
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.</p> <p>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>

Continued...

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	<p>Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.</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.</p> <p>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	<p>When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.</p>
Chronic	<p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>On the basis, primarily, of animal experiments, the material may be regarded as carcinogenic to humans. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in cancer on the basis of:</p> <ul style="list-style-type: none"> - appropriate long-term animal studies - other relevant information <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. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminum oxide during abrasives production.</p> <p>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.</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 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.</p> <p>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.</p> <p>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 um) are able to produce pathogenic effects in the lungs.</p> <p>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.</p> <p>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)</p> <p>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.</p> <p>In two studies by intraperitoneal injection in rats using wollastonite with median fibre lengths of 8.1 um and 5.6 um respectively,</p>

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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

Chromium(III) is considered an essential trace nutrient serving as a component of the "glucose tolerance factor" and a cofactor for insulin action. High concentrations of chromium are also found in RNA. Trivalent chromium is the most common form found in nature.

Chronic inhalation of trivalent chromium compounds produces irritation of the bronchus and lungs, dystrophic changes to the liver and kidney, pulmonary oedema, and adverse effects on macrophages. Intratracheal administration of chromium(III) oxide, in rats, increased the incidence of sarcomas, and tumors and reticulum cell sarcomas of the lung. There is inadequate evidence of carcinogenicity of chromium(III) compounds in experimental animals and humans (IARC).

Chronic exposure to hexavalent chromium compounds reportedly produces skin, eye and respiratory tract irritation, yellowing of the eyes and skin, allergic skin and respiratory reactions, diminished sense of smell and taste, blood disorders, liver and kidney damage, digestive disorders and lung damage. There is sufficient evidence of carcinogenicity of chromium(VI) compounds in experimental animals and humans to confirm these as Class 1 carcinogens (IARC).

Exposure to chromium during chrome production and in the chrome pigment industry is associated with cancer of the respiratory tract. A slight increase in gastrointestinal cancer following exposure to chromium compounds has also been reported. The greatest risk is attributed to exposure to acid-soluble, water-insoluble hexavalent chromium which occurs in roasting and refining processes. Animal studies support the idea that the most potent carcinogenic compounds are the slightly soluble hexavalent compounds. The cells are more active in the uptake of the hexavalent forms compared to trivalent forms and this may explain the difference in occupational effect. It is the trivalent form, however, which is metabolically active and binds with nucleic acid within the cell suggesting that chromium mutagenesis first requires biotransformation of the hexavalent form by reduction.

Hexavalent chromes produce chronic ulceration of skin surfaces (quite independent of other hypersensitivity reactions exhibited by the skin). Water-soluble chromium(VI) compounds come close to the top of any published "hit list" of contact allergens (eczematogens) producing positive results in 4 to 10% of tested individuals. On the other hand only chromium(III) compounds can bind to high molecular weight carriers such as proteins to form a complete allergen (such as a hapten). Chromium(VI) compounds cannot. It is assumed that reduction must take place for such compounds to manifest any contact sensitivity. The apparent contradiction that chromium(VI) salts cause allergies to chromium(III) compounds but that allergy to chromium(III) compounds is difficult to demonstrate is accounted for by the different solubilities and skin penetration of these compounds. Water-soluble chromium(VI) salts penetrate the horny layer of the skin more readily than chromium(III) compounds which are bound by cross-linking in the horny layer ("tanning", as for leather) and therefore do not reach the cells involved in antigen processing.

Overcrete Complete Resurfacing Compound	TOXICITY	IRRITATION
	Not Available	Not Available
portland cement	TOXICITY	IRRITATION
	Not Available	Not Available
silica crystalline - quartz	TOXICITY	IRRITATION
	Inhalation (Human)LCLo: 0.3 mg/m ³ /10Y ^[2]	Not Available
	Inhalation (Human)TCLo: 16 mppcf*8H/17.9Y ^[2]	
limestone	Inhalation (Rat)TCLo: 50 mg/m ³ /6H/71W ^[2]	
	TOXICITY	IRRITATION
	Oral (rat) LD50: 6450 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin (rabbit): 500 mg/24h-moderate
		Skin: no adverse effect observed (not irritating) ^[1]

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gypsum	TOXICITY	IRRITATION
	Oral (rat) LD50: >1581 mg/kg ^[1]	Not Available
calcium oxide	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: adverse effect observed (irreversible damage) ^[1]
	Oral (rat) LD50: ~500-2000 mg/kg ^[2]	Skin: adverse effect observed (irritating) ^[1]
calcium aluminate cement	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Not Available
	Oral (rat) LD50: >2000 mg/kg ^[1]	
Legend:	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. * Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

Overcrete Complete Resurfacing Compound	Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.
silica crystalline - quartz	<p>WARNING: For inhalation exposure <u>ONLY</u>: This substance has been classified by the IARC as Group 1: CARCINOGENIC TO HUMANS</p> <p>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.</p> <p>Intermittent exposure produces; focal fibrosis, (pneumoconiosis), cough, dyspnoea, liver tumours.</p> <p>* Millions of particles per cubic foot (based on impinger samples counted by light field techniques).</p> <p>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.</p>
LIMESTONE	<p>Eye (rabbit) 0.75: mg/24h - No evidence of carcinogenic properties. No evidence of mutagenic or teratogenic effects.</p> <p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>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.</p>
GYPSUM	<p>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.</p> <p>Unlike other fibers, gypsum is very soluble in the body; its half-life in the lungs has been estimated as minutes. In four healthy men receiving calcium supplementation with calcium sulfate (CaSO₄·1/2H₂O) (200 or 220 mg) for 22 days, an average absorption of 28.3% was reported.</p> <p>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</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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</p>

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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 NPSH 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 µg 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 µg/cm²) did not induce apoptosis. Negative results were also found in mouse peritoneal macrophages (tested at 150 µg/mL gypsum dust) and in Chinese hamster lung V79-4 cells (tested up to 100 µg/mL).

Carcinogenicity: In female Sprague-Dawley rats, i.p. injection of natural anhydrite dusts from German coal mines (doses not provided) induced granulomas; whether gypsum was the causal factor was not established. In Wistar rats, four i.p. injections of gypsum (25 mg each) induced abdominal cavity tumours, mostly sarcomatous mesothelioma, in 5% of animals; first tumour was seen at 546 days. In a subsequent experiment using the same procedure, female Wistar rats exhibited the first tumour at 579 days after the last injection. Mean survival of the tumour-bearing rats (5.7% of test group) was 583 days, while mean survival of the test group was 587 days. Tumour types seen were a sarcoma having cellular polymorphism, a carcinoma, and a reticulosarcoma.

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

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 or 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.

**Overcrete Complete
Resurfacing Compound &
PORTLAND CEMENT &
GYPSUM & CALCIUM
OXIDE & CALCIUM
ALUMINATE CEMENT**

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

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	nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
Overcrete Complete Resurfacing Compound & 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.
PORTLAND CEMENT & GYPSUM & CALCIUM ALUMINATE CEMENT	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

Overcrete Complete Resurfacing Compound	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 crystalline - quartz	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
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
calcium oxide	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	50.6mg/L	2
	EC50	48	Crustacea	49.1mg/L	2
	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

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calcium aluminate cement	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>100mg/L	2
	EC50	48	Crustacea	5.4mg/L	2
	EC50	72	Algae or other aquatic plants	3.6mg/L	2
	NOEC	72	Algae or other aquatic plants	2.6mg/L	2

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

Chromium in the oxidation state +3 (the trivalent form) is poorly absorbed by cells found in microorganisms, plants and animals. Chromate anions (CrO₄⁻, oxidation state +6, the hexavalent form) are readily transported into cells and toxicity is closely linked to the higher oxidation state.

Chromium Ecotoxicology:

Toxicity in Aquatic Organisms:

Chromium is harmful to aquatic organisms in very low concentrations. Fish food organisms are very sensitive to low levels of chromium. Chromium is toxic to fish although less so in warm water. Marked decreases in toxicity are found with increasing pH or water hardness; changes in salinity have little if any effect. Chromium appears to make fish more susceptible to infection. High concentrations can damage and/or accumulate in various fish tissues and in invertebrates such as snails and worms.

Reproduction of Daphnia is affected by exposure to 0.01 mg/kg hexavalent chromium/litre

Toxicity of chromium in fresh-water organisms (50% mortality)*

Compound	Category	Exposure	Toxicity Range (mg/litre)	Most sensitive species
hexavalent chrome	invertebrate	acute	0.067-59.9	scud
		long-term	-	-
	vertebrate	acute	17.6-249	fathead minnow
		long-term	0.265-2.0	rainbow trout
trivalent chrome	invertebrate	acute	2.0-64.0	cladoceran
		long-term	0.066	cladoceran
	vertebrate	acute	33.0-71.9	guppy
		long-term	1.0	fathead minnow

* from Environmental Health Criteria 61: WHO Publication.

Toxicity in Microorganisms:

In general, toxicity for most microorganisms occurs in the range of 0.05-5 mg chromium/kg of medium. Trivalent chromium is less toxic than the hexavalent form. The main signs of toxicity are inhibition of growth and the inhibition of various metabolic processes such as photosynthesis or protein synthesis. Gram-negative soil bacteria are generally more sensitive to hexavalent chromium (1-12 mg/kg) than the gram-positive types. Toxicity to trivalent chromium is not observed at similar levels. The toxicity of low levels of hexavalent chromium (1 mg/kg) indicates that soil microbial transformation, such as nitrification, may be affected. Chromium should not be introduced to municipal sewage treatment facilities.

Toxicity in Plants: Chromium in high concentrations can be toxic for plants. The main feature of chromium intoxication is chlorosis, which is similar to iron deficiency. Chromium affects carbohydrate metabolism and leaf chlorophyll concentration decreases with hexavalent chromium concentration (0.01-1 mg/l). The hexavalent form appears to more toxic than the trivalent species.

Biological half-life: The elimination curve for chromium, as measured by whole-body counting, has an exponential form. In rats, three different components of the curve have been identified, with half-lives of 0.5, 5.9 and 83.4 days, respectively.

Water Standards: Chromium is identified as a hazardous substance in the Federal (U.S.) Water Pollution Control Act and further regulated by Clean Air Water Act Amendments (US). These regulations apply to discharge. The US Primary drinking water Maximum Contaminant Level (MCL), for chromium, is 0.05 mg/l (total chromium).

Since chromium compounds cannot volatilize from water, transport of chromium from water to the atmosphere is not likely, except by transport in windblown sea sprays. Most of the chromium released into water will ultimately be deposited in the sediment. A very small percentage of chromium can be present in water in both soluble and insoluble forms. Soluble chromium generally accounts for a very small percentage of the total chromium. Most of the soluble chromium is present as chromium(VI) and soluble chromium(III) complexes. In the aquatic phase, chromium(III) occurs mostly as suspended solids adsorbed onto clayish materials, organics, or iron oxide (Fe₂O₃) present in water. Soluble forms and suspended chromium can undergo intramedia transport. Chromium(VI) in water will eventually be reduced to chromium(III) by organic matter in the water.

The reduction of chromium(VI) and the oxidation of chromium(III) in water has been investigated. The reduction of chromium(VI) by S⁻² or Fe⁺² ions under anaerobic conditions was fast, and the reduction half-life ranged from instantaneous to a few days. However, the reduction of chromium(VI) by organic sediments and soils was much slower and depended on the type and amount of organic material and on the redox condition of the water. The reaction was generally faster under anaerobic than aerobic conditions. The reduction half-life of chromium(VI) in water with soil and sediment ranged from 4 to 140 day. Dissolved oxygen by itself in natural waters did not cause any measurable oxidation of chromium(III) to chromium(VI) in 128 days. When chromium(III) was added to lake water, a slow oxidation of chromium(III) to chromium(VI) occurred, corresponding to an oxidation half-life of nine years. The oxidation of chromium(III) to chromium(VI) during chlorination of water was highest in the pH range of 5.5-6.0. However, the process would rarely occur during chlorination of drinking water because of the low

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concentrations of chromium(III) in these waters, and the presence of naturally occurring organics that may protect chromium(III) from oxidation, either by forming strong complexes with chromium(III) or by acting as a reducing agent to free available chlorine.

The bioconcentration factor (BCF) for chromium(VI) in rainbow trout (*Salmo gairdneri*) is 1. In bottom feeder bivalves, such as the oyster (*Crassostrea virginica*), blue mussel (*Mytilus edulis*), and soft shell clam (*Mya arenaria*), the BCF values for chromium(III) and chromium(VI) may range from 86 to 192.

The bioavailability of chromium(III) to freshwater invertebrates (*Daphnia pulex*) decreased with the addition of humic acid. This decrease in bioavailability was attributed to lower availability of the free form of the metal due to its complexation with humic acid. Based on this information, chromium is not expected to biomagnify in the aquatic food chain. Although higher concentrations of chromium have been reported in plants growing in high chromium-containing soils (e.g., soil near ore deposits or chromium-emitting industries and soil fertilized by sewage sludge) compared with plants growing in normal soils, most of the increased uptake in plants is retained in roots, and only a small fraction is translocated in the aboveground part of edible plants. Therefore, bioaccumulation of chromium from soil

to above-ground parts of plants is unlikely. There is no indication of biomagnification of chromium along the terrestrial food chain (soil-plant-animal).

The fate of chromium in soil is greatly dependent upon the speciation of chromium, which is a function of redox potential and the pH of the soil. In most soils, chromium will be present predominantly in the chromium(III) state. This form has very low solubility and low reactivity resulting in low mobility in the environment and low toxicity in living organisms. Under oxidizing conditions chromium(VI) may be present in soil as CrO_4^{2-} and HCrO_4^- . In this form, chromium is relatively soluble, mobile, and toxic to living organisms. In deeper soil where anaerobic conditions exist, chromium(VI) will be reduced to chromium(III) by S-2 and Fe+2 present in soil. The reduction of chromium(VI) to chromium(III) is possible in aerobic soils that contain appropriate organic energy sources to carry out the redox reaction. The reduction of chromium(VI) to chromium(III) is facilitated by low pH. From thermodynamic considerations, chromium(VI) may exist in the aerobic zone of some natural soil. The oxidation of chromium(III) to chromium(VI) in soil is facilitated by the presence of low oxidisable organic substances, oxygen, manganese dioxide, and moisture. Organic forms of chromium(III) (e.g., humic acid complexes) are more easily oxidised than insoluble oxides. Because most chromium(III) in soil is immobilized due to adsorption and complexation with soil materials, the barrier to this oxidation process is the lack of availability of mobile chromium(III) to immobile manganese dioxide in soil surfaces. Due to this lack of availability of mobile chromium(III) to manganese dioxide surfaces, a large portion of chromium in soil will not be oxidized to chromium(VI), even in the presence of manganese dioxide and favorable pH conditions.

The microbial reduction of chromium(VI) to chromium(III) has been discussed as a possible remediation technique in heavily contaminated environmental media or wastes. Factors affecting the microbial reduction of chromium(VI) to chromium(III) include biomass concentration, initial chromium(VI) concentration, temperature, pH, carbon source, oxidation-reduction potential and the presence of both oxyanions and metal cations. Although high levels of chromium(VI) are toxic to most microbes, several resistant bacterial species have been identified which could ultimately be employed in remediation strategies.

Chromium in soil is present mainly as insoluble oxide $\text{Cr}_2\text{O}_3 \cdot n\text{H}_2\text{O}$, and is not very mobile in soil. A leachability study was conducted to study the mobility of chromium in soil. Due to different pH values, a complicated adsorption process was observed and chromium moved only slightly in soil.

Chromium was not found in the leachate from soil, possibly because it formed complexes with organic matter. These results support previous data finding that chromium is not very mobile in soil. These results are supported by leachability investigation in which chromium mobility was studied for a period of 4 years in a sandy loam. The vertical migration pattern of chromium in this soil indicated that after an initial period of mobility, chromium forms insoluble complexes and little leaching is observed. Flooding of soils and the subsequent anaerobic decomposition of plant detritus matters may increase the mobilization of chromium(III) in soils due to formation of soluble complexes. This complexation may be facilitated by a lower soil pH. A smaller percentage of total chromium in soil exists as soluble chromium(VI) and chromium(III), which are more mobile in soil. The mobility of soluble chromium in soil will depend on the sorption characteristics of the soil. The relative retention of metals by soil is in the order of lead > antimony > copper > chromium > zinc > nickel > cobalt > cadmium. The sorption of chromium to soil depends primarily on the clay content of the soil and, to a lesser extent, on Fe_2O_3 and the organic content of soil. Chromium that is irreversibly sorbed onto soil, for example, in the interstitial lattice of goethite, FeOOH , will not be bioavailable to plants and animals under any condition. Organic matter in soil is expected to convert soluble chromate, chromium(VI), to insoluble chromium(III) oxide, Cr_2O_3 . Chromium in soil may be transported to the atmosphere as an aerosol.

Surface runoff from soil can transport both soluble and bulk precipitate of chromium to surface water. Soluble and unadsorbed chromium(VI) and chromium(III) complexes in soil may leach into groundwater. The leachability of chromium(VI) in the soil increases as the pH of the soil increases. On the other hand, lower pH present in acid rain may facilitate leaching of acid-soluble chromium(III) and chromium(VI) compounds in soil.

Chromium has a low mobility for translocation from roots to aboveground parts of plants. However, depending on the geographical areas where the plants are grown, the concentration of chromium in aerial parts of certain plants may differ by a factor of 2-3.

In the atmosphere, chromium(VI) may be reduced to chromium(III) at a significant rate by vanadium (V^{2+} , V^{3+} , and VO_2^+), Fe^{2+} , HSO_3^- , and As^{3+} . Conversely, chromium(III), if present as a salt other than Cr_2O_3 , may be oxidized to chromium(VI) in the atmosphere in the presence of at least 1% manganese oxide..

However, this reaction is unlikely under most environmental conditions. The estimated atmospheric half-life for chromium(VI) reduction to chromium(III) was reported in the range of 16 hours to about 5 days.

Cellulosic products, including cellulose ethers, generally have a low biodegradation rate and are generally of low toxicity to fish.

For formaldehyde:

Environmental fate:

Formaldehyde is ubiquitous in the environment as a contaminant of smoke and as photochemical smog.

In the atmosphere, formaldehyde both photolyses and reacts with reactive free radicals (primarily hydroxyl radicals); half-lives in the sunlit troposphere are 1.25 to 6 hours for photolysis, and 7.13-71.3 hours for reaction with hydroxyl radicals.

Reaction with nitrate radicals, insignificant during the day, may be an important removal process at night. Due to its solubility, formaldehyde will efficiently transfer to rain and surface water; one model predicts dry deposition and wet removal half-lives of 19 and 50 hours, respectively.

In water, formaldehyde will biodegrade to low concentrations within days; adsorption to sediment and volatilisation are not expected to be significant routes.

In soil, aqueous solutions of formaldehyde leach through the soil; at high concentrations adsorption to clay minerals may occur. Although biodegradable under both aerobic and anaerobic conditions the fate of formaldehyde in soil is unclear.

It does not bioconcentrate in the food chain.

Concentrated solutions containing formaldehyde are unstable, both oxidising slowly to form formic acid and polymerising. In the presence of air and moisture, polymerisation takes place readily in concentrated solutions at room temperature to form paraformaldehyde, a solid mixture of linear polyoxymethylene glycols containing 90-99% formaldehyde.

Drinking Water Standards:

hydrocarbon total: 10 ug/l (UK max.)

pesticide: 0.1 ug/l (UK max.)

formaldehyde: 900 ug/l (WHO guideline)

Air Quality Standards:

<0.1 mg/m³ as a 30 min. average, indoor air, non-industrial buildings (WHO guideline)

DO NOT discharge into sewer or waterways.

Persistence and degradability

Continued...

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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	<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. 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.
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SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

PORTLAND CEMENT IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

SILICA CRYSTALLINE - QUARTZ IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

Chemical Footprint Project - Chemicals of High Concern List

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

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

LIMESTONE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

GYPSUM IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

Continued...

Overcrete Complete Resurfacing Compound

CALCIUM OXIDE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)

CALCIUM ALUMINATE CEMENT IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

National Inventory Status

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (portland cement; silica crystalline - quartz; gypsum; calcium oxide; calcium aluminate cement)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (portland cement)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (portland cement; calcium aluminate cement)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (calcium aluminate cement)
Vietnam - NCI	Yes
Russia - ARIPS	No (calcium aluminate cement)
Legend:	<p>Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing (see specific ingredients in brackets)</p>

SECTION 16 OTHER INFORMATION

Revision Date	20/05/2020
Initial Date	19/05/2020

Other information

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

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

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit
IDLH: Immediately Dangerous to Life or Health Concentrations
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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