



Castor Crete SL Bags

ICP Construction Inc.

Version No: 3.7

Safety Data Sheet according to OSHA HazCom Standard (2012) requirements

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L.GHS.USA.EN

SECTION 1 Identification

Product Identifier

Product name	Castor Crete SL Bags
Synonyms	Not Available
Other means of identification	Not Available

Recommended use of the chemical and restrictions on use

Relevant identified uses	Use according to manufacturer's instructions
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Name, address, and telephone number of the chemical manufacturer, importer, or other responsible party

Registered company name	ICP Construction Inc.
Address	150 Dascomb Road Andover, MA 01810 United States
Telephone	1-866-667-5119 1-978-623-9987
Fax	Not Available
Website	www.icpgroup.com
Email	sds@icpgroup.com

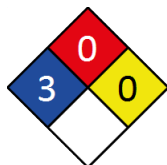
Emergency phone number

Association / Organisation	ChemTel
Emergency telephone numbers	1-800-255-3924
Other emergency telephone numbers	1-813-248-0585

SECTION 2 Hazard(s) identification

Classification of the substance or mixture

NFPA 704 diamond



Note: The hazard category numbers found in GHS classification in section 2 of this SDSs are NOT to be used to fill in the NFPA 704 diamond. Blue = Health Red = Fire Yellow = Reactivity White = Special (Oxidizer or water reactive substances)

Classification	Specific Target Organ Toxicity - Repeated Exposure Category 2, Serious Eye Damage/Eye Irritation Category 1, Carcinogenicity Category 1A, Sensitisation (Skin) Category 1, Germ Cell Mutagenicity Category 2, Skin Corrosion/Irritation Category 1A
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Label elements

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

H373	May cause damage to organs through prolonged or repeated exposure.
H350	May cause cancer.

Castor Crete SL Bags

H317	May cause an allergic skin reaction.
H341	Suspected of causing genetic defects.
H314	Causes severe skin burns and eye damage.

Hazard(s) not otherwise classified

Not Applicable

Precautionary statement(s) Prevention

P202	Do not handle until all safety precautions have been read and understood.
P260	Do not breathe mist/vapours/spray.
P264 P265	Wash thoroughly after handling. Do not touch eyes.
P272	Contaminated work clothing should not be allowed out of the workplace
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Precautionary statement(s) Response

P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P310	Immediately call a POISON CENTER or doctor/physician.
P303+P361+P353	IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower.
P333+P313	IF Skin irritation or rash occurs: Get medical advice/attention.
P308+P313	If exposed or concerned: Get medical advice/attention
P314	Get medical advice/attention if you feel unwell
P363 P305+P354+P338 P317	Wash contaminated clothing before reuse. IF IN EYES: Immediately rinse with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing. Get emergency medical help.

Precautionary statement(s) Storage

P405	Store locked up.
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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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Not Applicable

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
1305-62-0	1-5	<u>calcium hydroxide</u>
14808-60-7	45-70	<u>silica crystalline - quartz</u>
7429-90-5	7-13	<u>aluminium</u>
1305-78-8	1-5	<u>calcium oxide</u>
65997-15-1	10-30	<u>portland cement</u>

The specific chemical identity and/or exact percentage (concentration) of composition has been withheld as a trade secret.

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. ▶ DO NOT attempt to remove particles attached to or embedded in eye . ▶ Lay victim down, on stretcher if available and pad BOTH eyes, make sure dressing does not press on the injured eye by placing thick pads under dressing, above and below the eye. ▶ Seek urgent medical assistance, or transport to hospital.
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.

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Castor Crete SL Bags

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

Most important symptoms and effects, both acute and delayed

See Section 11

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

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, asterix, 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 Fire-fighting measures**Extinguishing media**

Metal dust fires need to be smothered with sand, inert dry powders.

DO NOT USE WATER, CO2 or FOAM.

- Use DRY sand, graphite powder, dry sodium chloride based extinguishers, G-1 or Met L-X to smother fire.
- Confining or smothering material is preferable to applying water as chemical reaction may produce flammable and explosive hydrogen gas.
- Chemical reaction with CO2 may produce flammable and explosive methane.
- If impossible to extinguish, withdraw, protect surroundings and allow fire to burn itself out.
- DO NOT** use halogenated fire extinguishing agents.

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.
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Castor Crete SL Bags

Special protective equipment and precautions for fire-fighters

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 in the event of a fire. Prevent, by any means available, spillage from entering drains or water courses. Use fire fighting procedures suitable for surrounding area. DO NOT approach containers suspected to be hot. Cool fire exposed containers with water spray from a protected location. If safe to do so, remove containers from path of fire. Equipment should be thoroughly decontaminated after use. When silica dust is dispersed in air, firefighters should wear inhalation protection as hazardous substances from the fire may be adsorbed on the silica particles. When heated to extreme temperatures, (>1700 deg.C) amorphous silica can fuse.
Fire/Explosion Hazard	<ul style="list-style-type: none"> DO NOT disturb burning dust. Explosion may result if dust is stirred into a cloud, by providing oxygen to a large surface of hot metal. DO NOT use water or foam as generation of explosive hydrogen may result. <p>With the exception of the metals that burn in contact with air or water (for example, sodium), masses of combustible metals do not represent unusual fire risks because they have the ability to conduct heat away from hot spots so efficiently that the heat of combustion cannot be maintained - this means that it will require a lot of heat to ignite a mass of combustible metal. Generally, metal fire risks exist when sawdust, machine shavings and other metal 'fines' are present.</p> <p>Metal powders, while generally regarded as non-combustible:</p> <ul style="list-style-type: none"> May burn when metal is finely divided and energy input is high. May react explosively with water. May be ignited by friction, heat, sparks or flame. May REIGNITE after fire is extinguished. Will burn with intense heat. <p>Note:</p> <ul style="list-style-type: none"> Metal dust fires are slow moving but intense and difficult to extinguish. Containers may explode on heating. Dusts or fumes may form explosive mixtures with air. Gases generated in fire may be poisonous, corrosive or irritating. Hot or burning metals may react violently upon contact with other materials, such as oxidising agents and extinguishing agents used on fires involving ordinary combustibles or flammable liquids. Temperatures produced by burning metals can be higher than temperatures generated by burning flammable liquids Some metals can continue to burn in carbon dioxide, nitrogen, water, or steam atmospheres in which ordinary combustibles or flammable liquids would be incapable of burning. <p>Decomposition may produce toxic fumes of: silicon dioxide (SiO₂) metal oxides</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 corrosive fumes.</p> <ul style="list-style-type: none"> Particle size, coating and dispersion in air determine reactivity of aluminium Bulk aluminium is not combustible but at high temperatures, molten aluminium can be ignited and burn. Molten aluminium may react violently if it comes into contact with water. Aluminium is rapidly oxidised by water at 180 C Atomised aluminium dusts are potentially explosive. Electric sparks may ignite the dust cloud even in atmospheres containing low oxygen (7%). In air the dust may be ignited in contact with hot surfaces or flame where temperatures exceed 640 deg C.

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none"> 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"> Do not use compressed air to remove metal dusts from floors, beams or equipment Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation. Use non-sparking handling equipment, tools and natural bristle brushes. Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations Cover and reseal partially empty containers. Do not allow chips, fines or dusts to contact water, particularly in enclosed areas. <p>If molten:</p> <ul style="list-style-type: none"> Contain the flow using dry sand or salt flux as a dam. All tooling (e.g., shovels or hand tools) and containers which come in contact with molten metal must be preheated or specially coated, rust free and approved for such use. Allow the spill to cool before remelting scrap.

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

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Castor Crete SL Bags

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	<p>For molten metals:</p> <ul style="list-style-type: none"> · Molten metal and water can be an explosive combination. The risk is greatest when there is sufficient molten metal to entrap or seal off water. Water and other forms of contamination on or contained in scrap or remelt ingot are known to have caused explosions in melting operations. While the products may have minimal surface roughness and internal voids, there remains the possibility of moisture contamination or entrapment. If confined, even a few drops can lead to violent explosions. · All tooling, containers, molds and ladles, which come in contact with molten metal must be preheated or specially coated, rust free and approved for such use. · Any surfaces that may contact molten metal (e.g. concrete) should be specially coated · Drops of molten metal in water (e.g. from plasma arc cutting), while not normally an explosion hazard, can generate enough flammable hydrogen gas to present an explosion hazard. Vigorous circulation of the water and removal of the particles minimise the hazard. <p>During melting operations, the following minimum guidelines should be observed:</p> <ul style="list-style-type: none"> · Inspect all materials prior to furnace charging and completely remove surface contamination such as water, ice, snow, deposits of grease and oil or other surface contamination resulting from weather exposure, shipment, or storage. · Store materials in dry, heated areas with any cracks or cavities pointed downwards. · Preheat and dry large objects adequately before charging in to a furnace containing molten metal. This is typically done by the use of a drying oven or homogenising furnace. The dry cycle should bring the metal temperature of the coldest item of the batch to 200 degree C (400 deg F) and then hold at that temperature for 6 hours. ▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ DO NOT enter confined spaces until atmosphere has been checked. ▸ DO NOT allow material to contact humans, exposed food or food utensils. ▸ Avoid contact with incompatible materials. ▸ When handling, DO NOT eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	<ul style="list-style-type: none"> ▸ 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 bunded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams). ▸ Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▸ Lined metal can, lined metal pail/ can. ▸ Plastic pail. ▸ Polyliner drum. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks. ▸ Bulk bags: Reinforced bags required for dense materials.
Storage incompatibility	<p>The material is described as an electropositive metal.</p> <p>The activity or electromotive series of metals is a listing of the metals in decreasing order of their reactivity with hydrogen-ion sources such as water and acids. In the reaction with a hydrogen-ion source, the metal is oxidised to a metal ion, and the hydrogen ion is reduced to H₂. The ordering of the activity series can be related to the standard reduction potential of a metal cation. The more positive the standard reduction potential of the cation, the more difficult it is to oxidise the metal to a hydrated metal cation and the later that metal falls in the series</p> <p>Three notable groups comprise the series</p> <ul style="list-style-type: none"> ▸ very electropositive metals ▸ electropositive metals ▸ electronegative metals <p>Electropositive metals have electronegativities that fall between 1.4 and 1.9. Cations of these metals generally have standard reduction potentials between 0.0 and -1.6 V</p> <p>They:</p> <ul style="list-style-type: none"> ▸ do not react very readily with water to release hydrogen ▸ react with H⁺ (acids) <p>Electropositive metals do not burn in air as readily as do very electropositive metals. The surfaces of these metals will tarnish in the presence of oxygen forming a protective oxide coating. This coating protects the bulk of the metal against further oxidation (the metal is passivated).</p> <p>Reaction is reduced in the massive form (sheet, rod, or drop), compared with finely divided forms. The less active metals will not burn in air but:</p> <ul style="list-style-type: none"> ▸ can react exothermically with oxidising acids to form noxious gases. ▸ catalyse polymerisation and other reactions, particularly when finely divided ▸ react with halogenated hydrocarbons (for example, copper dissolves when heated in carbon tetrachloride), sometimes forming explosive compounds. ▸ Elemental metals may react with azo/diazo compounds to form explosive products <ul style="list-style-type: none"> ▸ Finely divided metal powders develop pyrophoricity when a critical specific surface area is exceeded; this is ascribed to high heat of oxide formation on exposure to air. ▸ Safe handling is possible in relatively low concentrations of oxygen in an inert gas ▸ Several pyrophoric metals, stored in glass bottles have ignited when the container is broken on impact. Storage of these materials moist and

Castor Crete SL Bags

- in metal containers is recommended.
- ▶ The reaction residues from various metal syntheses (involving vacuum evaporation and co-deposition with a ligand) are often pyrophoric

If the surface of the metal is in contact with both oxygen and water, corrosion can occur. In corrosion, the metal acts as an anode and is oxidised.

Many metals may incandesce, react violently, ignite or react explosively upon addition of concentrated nitric acid. Some electropositive metals do not react with nitric acid because they are passivated.

<http://www.wou.edu/las/physci/ch412/activity.htm>

For aluminas (aluminium oxide):

Incompatible with hot chlorinated rubber.

In the presence of chlorine trifluoride may react violently and ignite.

-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.

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

Calcium oxide:

- ▶ reacts violently with water, evolving high quantities of heat

- ▶ reacts violently, with possible ignition or explosion, with acids, anilinium perchlorate, bromine pentafluoride, chlorine trifluoride, fluorine, hydrogen fluoride, hydrazine, hydrogen sulfide, hydrogen trisulfide, isopropyl isocyanide dichloride, light metals, lithium, magnesium, powdered aluminium, phosphorus, potassium, sulfur trioxide

- ▶ increase the explosive sensitivity of azides, nitroalkanes (e.g. nitroethane, nitromethane, 1-nitropropane etc.)

- ▶ is incompatible with boric acid, boron trifluoride, carbon dioxide, ethanol, halogens (such as fluorine), metal halides, phosphorus pentoxide, selenium oxychloride, sulfur dioxide and many organic materials

Calcium sulfate:

- ▶ reacts violently with reducing agents, acrolein, alcohols, chlorine trifluoride, diazomethane, ethers, fluorine, hydrazine, hydrazinium perchlorate, hydrogen peroxide, finely divided aluminium or magnesium, peroxyfuroic acid, red phosphorus, sodium 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

For iron oxide (ferric oxide):

- ▶ Avoid storage with aluminium, calcium hypochlorite and ethylene oxide.

- ▶ Risk of explosion occurs following reaction with powdered aluminium, calcium silicide, ethylene oxide (polymerises), carbon monoxide, magnesium and perchlorates.

- ▶ Risk of ignition or formation of flammable gases or vapours occurs following reaction with carbides, for example caesium carbide, (produces heat), hydrogen sulfide, hydrogen peroxide (decomposes).

- ▶ An intimately powdered mixture with aluminium, usually ignited by magnesium ribbon, reacts with an intense exotherm to produce molten iron in the commercial "thermit" welding process

Silicas:

- ▶ react with hydrofluoric acid to produce silicon tetrafluoride gas

- ▶ react with xenon hexafluoride to produce explosive xenon trioxide

- ▶ reacts exothermically with oxygen difluoride, and explosively with chlorine trifluoride (these halogenated materials are not commonplace industrial materials) and other fluorine-containing compounds

- ▶ may react with fluorine, chlorates

- ▶ are incompatible with strong oxidisers, manganese trioxide, chlorine trioxide, strong alkalis, metal oxides, concentrated orthophosphoric acid, vinyl acetate

- ▶ may react vigorously when heated with alkali carbonates.

- ▶ WARNING: Avoid or control reaction with peroxides. All *transition metal* peroxides should be considered as potentially explosive. For example transition metal complexes of alkyl hydroperoxides may decompose explosively.

- ▶ The pi-complexes formed between chromium(0), vanadium(0) and other transition metals (haloarene-metal complexes) and mono- or poly-fluorobenzene show extreme sensitivity to heat and are explosive.

- ▶ Avoid reaction with borohydrides or cyanoborohydrides

- ▶ Metals and their oxides or salts may react violently with chlorine trifluoride and bromine trifluoride.

- ▶ These trifluorides are hypergolic oxidisers. They ignite on contact (without external source of heat or ignition) with recognised fuels - contact with these materials, following an ambient or slightly elevated temperature, is often violent and may produce ignition.

- ▶ The state of subdivision may affect the results.

- ▶ Many metals may incandesce, react violently, ignite or react explosively upon addition of concentrated nitric acid.

- ▶ Avoid contact with copper, aluminium and their alloys.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US OSHA Permissible Exposure Limits (PELs) Table Z-3	calcium hydroxide	Inert or Nuisance Dust: Respirable fraction	5 mg/m ³ / 15 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	calcium hydroxide	Inert or Nuisance Dust: Total Dust	15 mg/m ³ / 50 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	calcium hydroxide	Calcium hydroxide- Total dust	15 mg/m ³	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	calcium hydroxide	Calcium hydroxide- Respirable fraction	5 mg/m ³	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	calcium hydroxide	Calcium hydroxide	5 mg/m ³	Not Available	Not Available	Not Available

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Castor Crete SL Bags

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US ACGIH Threshold Limit Values (TLV)	calcium hydroxide	Calcium hydroxide	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	silica crystalline - quartz	Silica: Crystalline: Quartz (Respirable)	10 (%SiO ₂ +2) mg/m3 / 250 (%SiO ₂ +5) mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	silica crystalline - quartz	Silica, crystalline (as respirable dust)	0.05 mg/m3	Not Available	Not Available	Ca; See Appendix A
US ACGIH Threshold Limit Values (TLV)	silica crystalline - quartz	Silica, crystalline - α-quartz and cristobalite (Respirable particulate matter)	0.025 mg/m3	Not Available	Not Available	A2
US OSHA Permissible Exposure Limits (PELs) Table Z-3	aluminium	Inert or Nuisance Dust: Total Dust	15 mg/m3 / 50 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	aluminium	Inert or Nuisance Dust: Respirable fraction	5 mg/m3 / 15 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	aluminium	Aluminum Metal (as Al)- Respirable fraction	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	aluminium	Aluminum Metal (as Al)- Total dust	15 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	aluminium	Aluminum - total	10 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	aluminium	Aluminum - respirable	5 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	aluminium	Aluminum (pyro powders and welding fumes, as Al)	5 mg/m3	Not Available	Not Available	Not Available
US ACGIH Threshold Limit Values (TLV)	aluminium	Aluminum metal and insoluble compounds (Respirable particulate matter)	1 mg/m3	Not Available	Not Available	A4
US OSHA Permissible Exposure Limits (PELs) Table Z-1	calcium oxide	Calcium oxide	5 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	calcium oxide	Calcium oxide	2 mg/m3	Not Available	Not Available	Not Available
US ACGIH Threshold Limit Values (TLV)	calcium oxide	Calcium oxide	2 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	portland cement	Silicates (less than 1% crystalline silica): Portland cement	50 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	portland cement	Portland cement- Respirable fraction	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	portland cement	Portland cement- Total dust	15 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	portland cement	Portland cement - total	10 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	portland cement	Portland cement - respirable	5 mg/m3	Not Available	Not Available	Not Available
US ACGIH Threshold Limit Values (TLV)	portland cement	Portland cement (Respirable particulate matter)	1 mg/m3	Not Available	Not Available	A4

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
calcium hydroxide	15 mg/m3	240 mg/m3	1,500 mg/m3
silica crystalline - quartz	0.075 mg/m3	33 mg/m3	200 mg/m3
calcium oxide	6 mg/m3	110 mg/m3	660 mg/m3

Ingredient	Original IDLH	Revised IDLH
calcium hydroxide	Not Available	Not Available
silica crystalline - quartz	25 mg/m3 / 50 mg/m3	Not Available
aluminium	Not Available	Not Available
calcium oxide	25 mg/m3	Not Available
portland cement	5,000 mg/m3	Not Available

MATERIAL DATA

WARNING: For inhalation exposure **ONLY:**

This substance has been classified by the ACGIH as A2 Suspected Human Carcinogen. for chrome(VI) containing substances:

Some jurisdictions require that health surveillance be carried on workers occupationally exposed to inorganic chromium. Such surveillance should emphasise

- demography, occupational and medical history and health advice
- physical examination with emphasis on the respiratory system and skin
- weekly skin inspection of hands and forearms by a "responsible person"

An induction threshold for chromium (VI) allergy is difficult to define, but from experience in the construction industry and among cement workers it is well known that levels of 10-20 mg/kg soluble chromium (VI) in the cement has caused sensitisation with a prevalence of about 4-5% of the exposed population.

Minimum elicitation thresholds (MET10%) which will elicit an allergic response in 10% of already sensitised individuals are found to be in the range of 0.02 to 0.9 ug/cm²/ 2 days in different studies (Annex XV Report - Proposal for a restriction: Chromium (VI) compounds - Jan 2012)

<http://echa.europa.eu/documents/10162/4d88d444-4b8b-48ab-9c11-6e74819e047c>

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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 hydroxide:

In the absence of reports of adverse effects from exposure and the recognised lesser alkalinity of the alkaline earths compared with the the alkali hydroxides the relatively high value of TLV-TWA is recommended. This value corresponds in total alkalinity to 5 mg/m³ of sodium hydroxide or 2.5 times the TLV-TWA of sodium hydroxide.

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 and pyrophoric grades of aluminium:

Twenty seven year experience with aluminium oxide dust (particle size 96% 1.2 µm) without adverse effects either systemically or on the lung, and at a calculated concentration equivalent to 2 mg/m³ over an 8-hour shift has lead to the current recommendation of the TLV-TWA.

The limit should also apply to aluminium pyro powders whose toxicity is reportedly greater than aluminium dusts and should be protective against lung changes.

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

The recommended TLV is thought to reduce the likelihood of respiratory irritation and skin irritation from exposure to aerosols and mists of soluble iron salts.


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.

Because the margin of safety of the quartz TLV is not known with certainty and given the associated link between silicosis and lung cancer it is recommended that quartz concentrations be maintained as far below the TLV as prudent practices will allow.

Exposure to respirable crystalline silicas (RCS) represents a significant hazard to workers, particularly those employed in the construction industry where respirable dusts of cement and concrete are common. Cutting, grinding and other high speed processes, involving their finished products, may further result in dusty atmospheres. Bricks are also a potential source of RCSs under such circumstances.

It is estimated that half of the occupations, involved in construction work, are exposed to levels of RCSs, higher than the current allowable limits. Beaudry et al: Journal of Occupational and Environmental Hygiene 10: 71-77; 2013

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.</p> <p>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 unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure. ▶ Chemical goggles whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted. ▶ Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face

Castor Crete SL Bags

	<p>protection.</p> <ul style="list-style-type: none"> ▶ Alternatively a gas mask may replace splash goggles and face shields. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	<ul style="list-style-type: none"> ▶ Elbow length PVC gloves <p>NOTE:</p> <ul style="list-style-type: none"> ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>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.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> · 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. <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>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.</p> <p>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.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> · 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 <p>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.</p> <ul style="list-style-type: none"> ▶ Protective gloves eg. Leather gloves or gloves with Leather facing <p>Experience indicates that the following polymers are suitable as glove materials for protection against undissolved, dry solids, where abrasive particles are not present.</p> <ul style="list-style-type: none"> ▶ polychloroprene. ▶ nitrile rubber. ▶ butyl rubber. ▶ fluorocautchouc. ▶ polyvinyl chloride. <p>Gloves should be examined for wear and/ or degradation constantly.</p>
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent] ▶ Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent] ▶ Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely. ▶ Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. ▶ Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▶ Overalls. ▶ P.V.C apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit.

Castor Crete SL Bags

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	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
PVA	C
VITON	C

* 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.

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

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

* - Negative pressure demand ** - Continuous flow

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

If inhalation risk above the TLV exists, wear approved dust respirator.

Use respirators with protection factors appropriate for the exposure level.

- Up to 5 X TLV, use valveless mask type; up to 10 X TLV, use 1/2 mask dust respirator
- Up to 50 X TLV, use full face dust respirator or demand type C air supplied respirator
- Up to 500 X TLV, use powered air-purifying dust respirator or a Type C pressure demand supplied-air respirator
- Over 500 X TLV wear full-face self-contained breathing apparatus with positive pressure mode or a combination respirator with a Type C positive pressure supplied-air full-face respirator and an auxiliary self-contained breathing apparatus operated in pressure demand or other positive pressure mode
- Respirators may be necessary when engineering and administrative controls do not adequately prevent exposures.
- The decision to use respiratory protection should be based on professional judgment that takes into account toxicity information, exposure measurement data, and frequency and likelihood of the worker's exposure - ensure users are not subject to high thermal loads which may result in heat stress or distress due to personal protective equipment (powered, positive flow, full face apparatus may be an option).
- Published occupational exposure limits, where they exist, will assist in determining the adequacy of the selected respiratory protection. These may be government mandated or vendor recommended.
- Certified respirators will be useful for protecting workers from inhalation of particulates when properly selected and fit tested as part of a complete respiratory protection program.
- Where protection from nuisance levels of dusts are desired, use type N95 (US) or type P1 (EN143) dust masks. Use respirators and components tested and approved under appropriate government standards such as NIOSH (US) or CEN (EU)
- Use approved positive flow mask if significant quantities of dust becomes airborne.
- Try to avoid creating dust conditions.

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

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

Filtration rate: Filters at least 99.95% of airborne particles

Suitable for:

- Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.
 - Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.
 - Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS
 - Highly toxic particles e.g. Organophosphate Insecticides, Radionuclides, Asbestos
- Note: P3 Rating can only be achieved when used with a Full Face Respirator or Powered Air-Purifying Respirator (PAPR). If used with any other respirator, it will only provide filtration protection up to a P2 rating.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	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

Continued...

Castor Crete SL Bags

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
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (Not Available%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	Product is considered stable and 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>Inhalation of alkaline corrosives may produce irritation of the respiratory tract with coughing, choking, pain and mucous membrane damage. Pulmonary oedema may develop in more severe cases; this may be immediate or in most cases following a latent period of 5-72 hours. Symptoms may include a tightness in the chest, dyspnoea, frothy sputum, cyanosis and dizziness. Findings may include hypotension, a weak and rapid pulse and moist rales.</p> <p>Inhalation may result in chrome ulcers or sores of nasal mucosa and lung damage.</p> <p>The material has NOT been classified by EC Directives or other classification systems as "harmful by inhalation" nor has it been designated as "irritating to the respiratory system". This is because of the lack of corroborating animal or human evidence. In the absence of such evidence, care should be taken nevertheless to ensure exposure is kept to a minimum and that suitable control measures be used, in an occupational setting to control dusts and fumes.</p> <p>Not normally a hazard due to non-volatile nature of product</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> <p>Acute silicosis occurs under conditions of extremely high silica dust exposure particularly when the particle size of the dust is small. It differs greatly from classical silicosis both clinically and pathologically. The disease is rapidly progressive with diffuse pulmonary involvement developing only months after the initial exposure and causing deaths within 1 to 2 years. It is often complicated by an associated tuberculosis. The lungs of victims contain no classical silicotic nodules or only a few, microscopic abortive nodules, whereas the air spaces are diffusively filled and distended with silica-containing, lipoprotein paste in which degenerating and necrotic macrophages are sometimes discernible - the condition is sometimes described as alveolar lipoproteinosis. The uptake of silica particles by macrophages and lysosomal incorporation, is followed by rupture of the lysosomal membrane and release of lysosomal enzymes into cytoplasm of the macrophage. This causes the macrophage to be digested by its own enzymes and after lysis the free silica is released to be ingested by other macrophages thus continuing initiate collagen formation in the lung tissue producing the characteristic nodule found in classical (chronic) silicosis.</p> <p>Effects on lungs are significantly enhanced in the presence of respirable particles. Overexposure to respirable dust may produce wheezing, coughing and breathing difficulties leading to or symptomatic of impaired respiratory function.</p>
Ingestion	<p>Ingestion of alkaline corrosives may produce immediate pain, and circumoral burns. Mucous membrane corrosive damage is characterised by a white appearance and soapy feel; this may then become brown, oedematous and ulcerated. Profuse salivation with an inability to swallow or speak may also result. Even where there is limited or no evidence of chemical burns, both the oesophagus and stomach may experience a burning pain; vomiting and diarrhoea may follow. The vomitus may be thick and may be slimy (mucous) and may eventually contain blood and shreds of mucosa. Epiglottal oedema may result in respiratory distress and asphyxia. Marked hypotension is symptomatic of shock; a weak and rapid pulse, shallow respiration and clammy skin may also be evident. Circulatory collapse may occur and, if uncorrected, may produce renal failure. Severe exposures may result in oesophageal or gastric perforation accompanied by mediastinitis, substernal pain, peritonitis, abdominal rigidity and fever. Although oesophageal, gastric or pyloric stricture may be evident initially, these may occur after weeks or even months and years. Death may be quick and results from asphyxia, circulatory collapse or aspiration of even minute amounts. Death may also be delayed as a result of perforation, pneumonia or the effects of stricture formation.</p> <p>Chromate salts are corrosive because of their oxidising potency and produce tissue injury similar to acid burns. Ingestion may produce violent gastroenteritis, severe circulatory collapse and toxic nephritis. Peripheral vascular shock may also ensue.</p> <p>Acute toxic responses to aluminium are confined to the more soluble forms.</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>

Castor Crete SL Bags

	Not normally a hazard due to the physical form of product. The material is a physical irritant to the gastro-intestinal tract
Skin Contact	<p>The material can produce severe chemical burns following direct contact with the skin.</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>Skin contact may result in severe irritation particularly to broken skin. Ulceration known as "chrome ulcers" may develop. Chrome ulcers and skin cancer are significantly related.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
Eye	<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. Direct contact with alkaline corrosives may produce pain and burns. Oedema, destruction of the epithelium, corneal opacification and iritis may occur. In less severe cases these symptoms tend to resolve. In severe injuries the full extent of the damage may not be immediately apparent with late complications comprising a persistent oedema, vascularisation and corneal scarring, permanent opacity, staphyloma, cataract, symblepharon and loss of sight.</p>
Chronic	<p>Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis.</p> <p>Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure.</p> <p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.</p> <p>Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers</p> <p>Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.</p> <p>Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.</p> <p>On the basis of epidemiological data, the material is regarded as carcinogenic to humans. There is sufficient data to establish a causal association between human exposure to the material and the development of cancer.</p> <p>Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>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 insilicate 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, no intra-abdominal tumours were found.</p> <p>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</p> <p>Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress</p>

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and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic. Because aluminium competes with calcium for absorption, increased amounts of dietary aluminium may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies, aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseous tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory. These salts' estrogen-like effects have led to their classification as a metalloestrogen. Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.

After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus. Aluminium may persist for a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans.

At high levels of exposure, some aluminium compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminium compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminium potassium sulphate at high levels in the diet.

Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer's disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produce lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, testes, embryotoxicity, and the developing nervous system of 52, 75, 100, and 50 mg aluminium/kg bw/day, respectively. Similarly, the lowest no-observed-adverse-effect levels (NOAELs) for effects on these endpoints were reported at 30, 27, 100, and for effects on the developing nervous system, between 10 and 42 mg aluminium/kg bw per day, respectively.

Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 mg/l aluminium compared with communities where the aluminium level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminium exposure to brain disease. Aluminium concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramid-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations.

Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of "tau" a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminium hyperphosphorylates tau in vitro. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade. Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA).

Aluminium enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminium include baking powder, antacids and aluminium products used for general food preparation and storage (over 12 months, aluminium levels in soft drink packed in aluminium cans rose from 0.05 to 0.9 mg/l). [Walton, J and Bryson-Taylor, D. - *Chemistry in Australia*, August 1995] 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 (FVC), forced expiratory volume at 1 s (FEV1) and forced expiratory flows after exhalation of 50% and 75% of the vital capacity (FEF50, FEF75). The data suggests that occupational exposure to Portland cement dust may lead to a higher incidence of chronic respiratory symptoms and a reduction of ventilatory capacity.

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

Chronic symptoms produced by crystalline silicas included decreased vital lung capacity and chest infections. Lengthy exposure may cause silicosis a disabling form of pneumoconiosis which may lead to fibrosis, a scarring of the lining of the air sacs in the lung.

The form and severity in which silicosis manifests itself depends in part on the type and extent of exposure to silica dusts: chronic, accelerated and acute forms are all recognized. In later stages the critical condition may become disabling and potentially fatal. Restrictive and/or obstructive lung function changes may result from chronic exposure. A risk associated with silicosis is development of pulmonary tuberculosis (silico-tuberculosis). Respiratory insufficiencies due to massive fibrosis and reduced pulmonary function, possibly with accompanying heart failure, are other potential causes of death due to silicosis.

Not all individuals with silicosis will exhibit symptoms (signs) of the disease. However, silicosis can be progressive, and symptoms may potentially appear years after exposures have ceased. Symptoms of silicosis may include (but are not limited to): Shortness of breath; difficulty breathing with or without exertion; coughing; diminished work capacity; diminished chest expansion; reduction of lung volume; heart enlargement and/or failure.

Respirable dust containing newly broken particles has been shown to be more hazardous to animals in laboratory tests than respirable dust containing older silica particles of similar size. Respirable silica particles which had aged for sixty days or more showed less lung injury in animals than equal exposures of respirable dust containing newly broken pieces of silica. There are reports in the literature indicating that crystalline silica exposure may be associated with adverse health effects involving the kidney, scleroderma (thickening of the skin caused by swelling and thickening of fibrous tissue) and other autoimmune and immunity-related disorders. Several studies of persons with silicosis or silica exposure also indicate or suggest increased risk of developing lung cancer, a risk that may increase with the duration of exposure. Many of these studies of silicosis do not account for lung cancer confounders, especially smoking.

Symptoms may appear 8 to 18 months after initial exposure. Smoking increases this risk. Classic silicosis is a chronic disease characterised by the formation of scattered, rounded or stellate silica-containing nodules of scar tissue in the lungs ranging from microscopic to 1.0 cm or more.

The nodules isolate the inhaled silica particles and protect the surrounding normal and functioning tissue from continuing injury. Simple silicosis (in which the nodules are less than 1.0 cm in diameter) is generally asymptomatic but may be slowly progressive even in the absence of continuing exposure. Simple silicosis can develop in complicated silicoses (in which nodules are greater than 1.0 cm in diameter) and can produce disabilities including an associated tuberculous infection (which 50 years ago accounted for 75% of the deaths among silicotic workers). Crystalline silica deposited in the lungs causes epithelial and macrophage injury and activation. Crystalline silica translocates to the interstitium

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and the regional lymph nodes and cause the recruitment of inflammatory cells in a dose dependent manner. In humans, a large fraction of crystalline silica persists in the lungs. The question of potential carcinogenicity associated with chronic inhalation of crystalline silica remains equivocal with some studies supporting the proposition and others finding no significant association. The results of recent epidemiological studies suggest that lung cancer risk is elevated only in those patients with overt silicosis. A relatively large number of epidemiological studies have been undertaken and in some, increased risk gradients have been observed in relation to dose surrogates - cumulative exposure, duration of exposure, the presence of radiographically defined silicosis, and peak intensity exposure. Chronic inhalation in rats by single or repeated intratracheal instillation produced a significant increase in the incidences of adenocarcinomas and squamous cell carcinomas of the lung. Lifetime inhalation of crystalline silica (87% alpha-quartz) at 1 mg/m³ (74% respirable) by rats, produced an increase in animals with keratinising cystic squamous cell tumours, adenomas, adenocarcinomas, adenosquamous cell carcinomas, squamous cell carcinoma and nodular bronchiolar alveolar hyperplasia accompanied by extensive subpleural and peribronchiolar fibrosis, increased pulmonary collagen content, focal lipoproteinosis and macrophage infiltration. Thoracic and abdominal malignant lymphomas developed in rats after single intrapleural and intraperitoneal injection of suspensions of several types of quartz.

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

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

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

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.

Castor Crete SL Bags	TOXICITY	IRRITATION
	Not Available	Not Available
calcium hydroxide	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 10 mg - SEVERE
	Inhalation(Rat) LC50; >3 mg/l4h ^[1]	Eye: adverse effect observed (irritating) ^[1]
silica crystalline - quartz	TOXICITY	IRRITATION
	Oral (Rat) LD50; 500 mg/kg ^[2]	Not Available
aluminium	TOXICITY	IRRITATION
	Inhalation(Rat) LC50; >2.3 mg/l4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50; >2000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
calcium oxide	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: adverse effect observed (irreversible damage) ^[1]
	Inhalation(Rat) LC50; >3 mg/l4h ^[1]	Skin: adverse effect observed (irritating) ^[1]
portland cement	TOXICITY	IRRITATION
	Not Available	Not Available
Legend:	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. * Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

Castor Crete SL Bags	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
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studies.

For aluminium compounds:

Aluminium present in food and drinking water is poorly absorbed through the gastrointestinal tract. The bioavailability of aluminium is dependent on the form in which it is ingested and the presence of dietary constituents with which the metal cation can complex. Ligands in food can have a marked effect on absorption of aluminium, as they can either enhance uptake by forming absorbable (usually water soluble) complexes (e.g., with carboxylic acids such as citric and lactic), or reduce it by forming insoluble compounds (e.g., with phosphate or dissolved silicate).

Considering the available human and animal data it is likely that the oral absorption of aluminium can vary 10-fold based on chemical form alone. Although bioavailability appears to generally parallel water solubility, insufficient data are available to directly extrapolate from solubility in water to bioavailability.

For oral intake from food, the European Food Safety Authority (EFSA) has derived a tolerable weekly intake (TWI) of 1 milligram (mg) of aluminium per kilogram of bodyweight. In its health assessment, the EFSA states a medium bioavailability of 0.1 % for all aluminium compounds which are ingested with food. This corresponds to a systemically available tolerable daily dose of 0.143 microgrammes (µg) per kilogramme (kg) of body weight. This means that for an adult weighing 60 kg, a systemically available dose of 8.6 µg per day is considered safe.

Based on a neuro-developmental toxicity study of aluminium citrate administered via drinking water to rats, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) established a Provisional Tolerable Weekly Intake (PTWI) of 2 mg/kg bw (expressed as aluminium) for all aluminium compounds in food, including food additives. The Committee on Toxicity of chemicals in food, consumer products and the environment (COT) considers that the derivation of this PTWI was sound and that it should be used in assessing potential risks from dietary exposure to aluminium.

The Federal Institute for Risk Assessment (BfR) of Germany has assessed the estimated aluminium absorption from antiperspirants. For this purpose, the data, derived from experimental studies, on dermal absorption of aluminium from antiperspirants for healthy and damaged skin was used as a basis. At about 10.5 µg, the calculated systemic intake values for healthy skin are above the 8.6 µg per day that are considered safe for an adult weighing 60 kg. If aluminium -containing antiperspirants are used on a daily basis, the tolerable weekly intake determined by the EFSA is therefore exceeded. The values for damaged skin, for example injuries from shaving, are many times higher. This means that in case of daily use of an aluminium-containing antiperspirant alone, the TWI may be completely exhausted. In addition, further aluminium absorption sources such as food, cooking utensils and other cosmetic products must be taken into account.

Systemic toxicity after repeated exposure

No studies were located regarding dermal effects in animals following intermediate or chronic-duration dermal exposure to various forms of aluminium.

When orally administered to rats, aluminium compounds (including aluminium nitrate, aluminium sulfate and potassium aluminium sulfate) have produced various effects, including decreased gain in body weight and mild histopathological changes in the spleen, kidney and liver of rats (104 mg Al/kg bw/day) and dogs (88-93 mg Al/kg bw/day) during subchronic oral exposure. Effects on nerve cells, testes, bone and stomach have been reported at higher doses. Severity of effects increased with dose.

The main toxic effects of aluminium that have been observed in experimental animals are neurotoxicity and nephrotoxicity. Neurotoxicity has also been described in patients dialysed with water containing high concentrations of aluminium, but epidemiological data on possible adverse effects in humans at lower exposures are inconsistent.

Reproductive and developmental toxicity:

Studies of reproductive toxicity in male mice (intraperitoneal or subcutaneous administration of aluminium nitrate or chloride) and rabbits (administration of aluminium chloride by gavage) have demonstrated the ability of aluminium to cause testicular toxicity, decreased sperm quality in mice and rabbits and reduced fertility in mice. No reproductive toxicity was seen in females given aluminium nitrate by gavage or dissolved in drinking water. Multi-generation reproductive studies in which aluminium sulfate and aluminium ammonium sulfate were administered to rats in drinking water, showed no evidence of reproductive toxicity.

High doses of aluminium compounds given by gavage have induced signs of embryotoxicity in mice and rats in particular, reduced fetal body weight or pup weight at birth and delayed ossification. Developmental toxicity studies in which aluminium chloride was administered by gavage to pregnant rats showed evidence of foetotoxicity, but it was unclear whether the findings were secondary to maternal toxicity. A twelve-month neuro-development with aluminium citrate administered via the drinking water to Sprague-Dawley rats, was conducted according to Good Laboratory Practice (GLP). Aluminium citrate was selected for the study since it is the most soluble and bioavailable aluminium salt. Pregnant rats were exposed to aluminium citrate from gestational day 6 through lactation, and then the offspring were exposed post-weaning until postnatal day 364. An extensive functional observational battery of tests was performed at various times. Evidence of aluminium toxicity was demonstrated in the high (300 mg/kg bw/day of aluminium) and to a lesser extent, the mid-dose groups (100 mg/kg bw/day of aluminium). In the high-dose group, the main effect was renal damage, resulting in high mortality in the male offspring. No major neurological pathology or neurobehavioural effects were observed, other than in the neuromuscular subdomain (reduced grip strength and increased foot splay). Thus, the lowest observed adverse effect level (LOAEL) was 100 mg/kg bw/day and the no observed adverse effect level (NOAEL) was 30 mg/kg bw/day. Bioavailability of aluminium chloride, sulfate and nitrate and aluminium hydroxide was much lower than that of aluminium citrate. This study was used by JECFA as key study to derive the PTWI.

Genotoxicity

Aluminium compounds were non-mutagenic in bacterial and mammalian cell systems, but some produced DNA damage and effects on chromosome integrity and segregation in vitro. Clastogenic effects were also observed in vivo when aluminium sulfate was administered at high doses by gavage or by the intraperitoneal route. Several indirect mechanisms have been proposed to explain the variety of genotoxic effects elicited by aluminium salts in experimental systems. Cross-linking of DNA with chromosomal proteins, interaction with microtubule assembly and mitotic spindle functioning, induction of oxidative damage, damage of lysosomal membranes with liberation of DNAase, have been suggested to explain the induction of structural chromosomal aberrations, sister chromatid exchanges, chromosome loss and formation of oxidized bases in experimental systems. The EFSA Panel noted that these indirect mechanisms of genotoxicity, occurring at relatively high levels of exposure, are unlikely to be of relevance for humans exposed to aluminium via the diet. Aluminium compounds do not cause gene mutations in either bacteria or mammalian cells. Exposure to aluminium compounds does result in both structural and numerical chromosome aberrations both in in-vitro and in-vivo mutagenicity tests. DNA damage is probably the result of indirect mechanisms. The DNA damage was observed only at high exposure levels.

Carcinogenicity.

The available epidemiological studies provide limited evidence that certain exposures in the aluminium production industry are carcinogenic to humans, giving rise to cancer of the lung and bladder. However, the aluminium exposure was confounded by exposure to other agents including polycyclic aromatic hydrocarbons, aromatic amines, nitro compounds and asbestos. There is no evidence of increased cancer risk in non-occupationally exposed persons.

Neurodegenerative diseases.

Following the observation that high levels of aluminium in dialysis fluid could cause a form of dementia in dialysis patients, a number of studies were carried out to determine if aluminium could cause dementia or cognitive impairment as a consequence of environmental exposure over long periods. Aluminium was identified, along with other elements, in the amyloid plaques that are one of the diagnostic lesions in the brain for Alzheimer disease, a common form of senile and pre-senile dementia. Some of the epidemiology studies suggest the possibility of an association of Alzheimer disease with aluminium in water, but other studies do not confirm this association. All studies lack information on ingestion of aluminium from food and how concentrations of aluminium in food affect the association between aluminium in water and Alzheimer disease."

There are suggestions that persons with some genetic variants may absorb more aluminium than others, but there is a need for more analytical research to determine whether aluminium from various sources has a significant causal association with Alzheimer disease and other neurodegenerative diseases. Aluminium is a neurotoxicant in experimental animals. However, most of the animal studies performed have several limitations and therefore cannot be used for quantitative risk assessment.

Contact sensitivity:

It has been suggested that the body burden of aluminium may be linked to different diseases. Macrophagic myofasciitis and chronic fatigue syndrome can be caused by aluminium-containing adjuvants in vaccines. Macrophagic myofasciitis (MMF) has been described as a disease in adults presenting with ascending myalgia and severe fatigue following exposure to aluminium hydroxide-containing vaccines. The corresponding histological findings include aluminium-containing macrophages infiltrating muscle tissue at the injection site. The hypothesis is that the long-lasting granuloma triggers the development of the systemic syndrome.

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	Aluminium acts not only as an adjuvant, stimulating the immune system either to fend off infections or to tolerate antigens, it also acts as a sensitiser causing contact allergy and allergic contact dermatitis. In general, metal allergies are very common and aluminium is considered to be a weak allergen. A metal must be ionised to be able to act as a contact allergen, then it has to undergo haptenisation to be immunogenic and to initiate an immune response. Once inside the skin, the metal ions must bind to proteins to become immunologically reactive. The most important routes of exposure and sensitisation to aluminium are through aluminium-containing vaccines. One Swedish study showed a statistically significant association between contact allergy to aluminium and persistent itching nodules in children treated with allergen-specific immunotherapy (ASIT). Nodules were overrepresented in patients with contact allergy to aluminium. Other routes of sensitisation reported in the literature are the prolonged use of aluminium-containing antiperspirants, topical medication, and tattooing of the skin with aluminium-containing pigments. Most of the patients experienced eczematous reactions whereas tattooing caused granulomas. Even though aluminium is used extensively in industry, only a low number of cases of occupational skin sensitisation to aluminium have been reported. Systemic allergic contact dermatitis in the form of flare-up reactions after re-exposure to aluminium has been documented: pruritic nodules at present and previous injection sites, eczema at the site of vaccination as well as at typically atopic localisations after vaccination with aluminium-containing vaccines and/or patch testing with aluminium, and also after use of aluminium-containing toothpaste.
CALCIUM HYDROXIDE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
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>
Castor Crete SL Bags & 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.
Castor Crete SL Bags & CALCIUM HYDROXIDE & CALCIUM OXIDE & PORTLAND CEMENT	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
ALUMINIUM & PORTLAND 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

Castor Crete SL Bags	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
calcium hydroxide	Endpoint	Test Duration (hr)	Species	Value	Source
	EC10(ECx)	72h	Algae or other aquatic plants	>14mg/l	2
	LC50	96h	Fish	33.9mg/l	2
	EC50	72h	Algae or other aquatic plants	>14mg/l	2
	EC50	48h	Crustacea	49.1mg/l	2
silica crystalline - quartz	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
aluminium	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	48h	Crustacea	>100mg/l	1

Continued...

Castor Crete SL Bags

	LC50	96h	Fish	0.078-0.108mg/l	2
	EC50	72h	Algae or other aquatic plants	0.2mg/l	2
	EC50	48h	Crustacea	1.5mg/l	2
	EC50	96h	Algae or other aquatic plants	0.024mg/l	2
calcium oxide	Endpoint	Test Duration (hr)	Species	Value	Source
	EC10(ECx)	72h	Algae or other aquatic plants	>14mg/l	2
	LC50	96h	Fish	50.6mg/l	2
	EC50	72h	Algae or other aquatic plants	>14mg/l	2
	EC50	48h	Crustacea	49.1mg/l	2
portland cement	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
Legend:	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 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				

For Metal:

Atmospheric Fate: Metal-containing inorganic substances generally have negligible vapour pressure and are not expected to partition to air.

Environmental Fate: Environmental processes, such as oxidation, the presence of acids or bases and microbiological processes, may transform insoluble metals to more soluble ionic forms. Environmental processes may enhance bioavailability and may also be important in changing solubilities.

Aquatic/Terrestrial Fate: When released to dry soil, most metals will exhibit limited mobility and remain in the upper layer; some will leach locally into ground water and/ or surface water ecosystems when soaked by rain or melt ice. A metal ion is considered infinitely persistent because it cannot degrade further. Once released to surface waters and moist soils their fate depends on solubility and dissociation in water. A significant proportion of dissolved/ sorbed metals will end up in sediments through the settling of suspended particles. The remaining metal ions can then be taken up by aquatic organisms. Ionic species may bind to dissolved ligands or sorb to solid particles in water.

Ecotoxicity: Even though many metals show few toxic effects at physiological pH levels, transformation may introduce new or magnified effects.

For Silica:

Environmental Fate: Most documentation on the fate of silica in the environment concerns dissolved silica, in the aquatic environment, regardless of origin, (man-made or natural), or structure, (crystalline or amorphous).

Terrestrial Fate: Silicon makes up 25.7% of the Earth's crust, by weight, and is the second most abundant element, being exceeded only by oxygen. Silicon is not found free in nature, but occurs chiefly as the oxide and as silicates. Once released into the environment, no distinction can be made between the initial forms of silica.

Aquatic Fate: At normal environmental pH, dissolved silica exists exclusively as monosilicic acid. At pH 9.4, amorphous silica is highly soluble in water. Crystalline silica, in the form of quartz, has low solubility in water. Silicic acid plays an important role in the biological/geological/chemical cycle of silicon, especially in the ocean. Marine organisms such as diatoms, silicoflagellates and radiolarians use silicic acid in their skeletal structures and their skeletal remains leave silica in sea sediment

Ecotoxicity: Silicon is important to plant and animal life and is practically non-toxic to fish including zebrafish, and Daphnia magna water fleas.

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 to 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 be 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).

Continued...

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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 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 CrO₄²⁻ and HCrO₄⁻. 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 Cr₂O₃. nH₂O, 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 Fe₂O₃ 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, Cr₂O₃. 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 (V²⁺, V³⁺, and VO₂⁺), Fe²⁺, HSO₃⁻, and As³⁺. Conversely, chromium(III), if present as a salt other than Cr₂O₃, 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

For aluminium and its compounds and salts:

Despite its prevalence in the environment, no known form of life uses aluminium salts metabolically. In keeping with its pervasiveness, aluminium is well tolerated by plants and animals. Owing to their prevalence, potential beneficial (or otherwise) biological roles of aluminium compounds are of continuing interest.

Environmental fate:

Aluminium occurs in the environment in the form of silicates, oxides and hydroxides, combined with other elements such as sodium, fluorine and arsenic complexes with organic matter.

Acidification of soils releases aluminium as a transportable solution. Mobilisation of aluminium by acid rain results in aluminium becoming available for plant uptake.

As an element, aluminium cannot be degraded in the environment, but may undergo various precipitation or ligand exchange reactions. Aluminium in compounds has only one oxidation state (+3), and would not undergo oxidation-reduction reactions under environmental conditions. Aluminium can be complexed by various ligands present in the environment (e.g., fulvic and humic acids). The solubility of aluminium in the environment will depend on the ligands present and the pH.

The trivalent aluminium ion is surrounded by six water molecules in solution. The hydrated aluminium ion, [Al(H₂O)₆]³⁺, undergoes hydrolysis, in which a stepwise deprotonation of the coordinated water ligands forms bound hydroxide ligands (e.g., [Al(H₂O)₅(OH)]²⁺, [Al(H₂O)₄(OH)₂]⁺). The speciation of aluminium in water is pH dependent. The hydrated trivalent aluminium ion is the predominant form at pH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are Al(OH)₂⁺ and Al(OH)₂⁺, while the solid Al(OH)₃ is most prevalent between pH 5.2 and 8.8. The soluble species Al(OH)₄⁻ is the predominant species above pH 9, and is the only species present above pH 10. Polymeric aluminium hydroxides appear between pH 4.7 and 10.5, and increase in size until they are transformed into colloidal particles of amorphous Al(OH)₃, which crystallise to gibbsite in acid waters. Polymerisation is affected by the presence of dissolved silica; when enough silica is present, aluminium is precipitated as poorly crystallised clay mineral species.

Hydroxyaluminum compounds are considered amphoteric (e.g., they can act as both acids and bases in solution). Because of this property, aluminum hydroxides can act as buffers and resist pH changes within the narrow pH range of 4-5.

Monomeric aluminum compounds, typified by aluminum fluoride, chloride, and sulfate, are considered reactive or labile compounds, whereas polymeric aluminum species react much more slowly in the environment. Aluminium has a stronger attraction for fluoride in an acidic environment compared to other inorganic ligand.

The adsorption of aluminium onto clay surfaces can be a significant factor in controlling aluminium mobility in the environment, and these adsorption reactions, measured in one study at pH 3.0-4.1, have been observed to be very rapid. However, clays may act either as a sink or a source for soluble aluminium depending on the degree of aluminium saturation on the clay surface.

Within the pH range of 5-6, aluminium complexes with phosphate and is removed from solution. Because phosphate is a necessary nutrient in ecological systems, this immobilization of both aluminium and phosphate may result in depleted nutrient states in surface water.

Plant species and cultivars of the same species differ considerably in their ability to take up and translocate aluminium to above-ground parts. Tea leaves may contain very high concentrations of aluminium, >5,000 mg/kg in old leaves. Other plants that may contain high levels of aluminium include Lycopodium (Lycopodiaceae), a few ferns, Symlocos (Symlocaceae), and Orites (Proteaceae). Aluminium is often taken up and concentrated in root tissue. In sub-alpine ecosystems, the large root biomass of the Douglas fir, *Abies amabilis*, takes up aluminium and immobilizes it, preventing large accumulation in above-ground tissue. It is unclear to what extent aluminium is taken up into root food crops and leafy vegetables. An uptake factor (concentration of aluminium in the plant/concentration of aluminium in soil) of 0.004 for leafy vegetables and 0.00065 for fruits and tubers has been reported, but the pH and plant species from which these uptake factors were derived are unclear. Based upon these values, however, it is clear that aluminium is not taken up in plants from soil, but is instead biodiluted.

Aluminium concentrations in rainbow trout from an alum-treated lake, an untreated lake, and a hatchery were highest in gill tissue and lowest in muscle. Aluminium residue analyses in brook trout have shown that whole-body aluminium content decreases as the fish advance from larvae to juveniles. These results imply that the aging larvae begin to decrease their rate of aluminium uptake, to eliminate aluminium at a rate that exceeds uptake, or to maintain approximately the same amount of aluminium while the body mass increases. The decline

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in whole-body aluminum residues in juvenile brook trout may be related to growth and dilution by edible muscle tissue that accumulated less aluminum than did the other tissues. The greatest fraction of the gill-associated aluminum was not sorbed to the gill tissue, but to the gill mucus. It is thought that mucus appears to retard aluminum transport from solution to the membrane surface, thus delaying the acute biological response of the fish. It has been reported that concentrations of aluminum in whole-body tissue of the Atlantic salmon exposed to high concentrations of aluminum ranging from 3 ug/g (for fish exposed to 33 ug/L) to 96 ug/g (for fish exposed to 264 ug/L) at pH 5.5. After 60 days of exposure, BCFs ranged from 76 to 190 and were directly related to the aluminum exposure concentration. In acidic waters (pH 4.6-5.3) with low concentrations of calcium (0.5-1.5 mg Ca/L), labile aluminum between 25 and 75 ug/L is toxic. Because aluminum is toxic to many aquatic species, it is not bioaccumulated to a significant degree (BCF <300) in most fish and shellfish; therefore, consumption of contaminated fish does not appear to be a significant source of aluminum exposure in humans. Bioconcentration of aluminum has also been reported for several aquatic invertebrate species. BCF values ranging from 0.13 to 0.5 in the whole-body were reported for the snail. Bioconcentration of aluminum has also been reported for aquatic insects.

Ecotoxicity:**Freshwater species pH >6.5**

Fish: Acute LC50 (48-96 h) 5 spp: 0.6 (*Salmo salar*) - 106 mg/L; Chronic NOEC (8-28 d): 7 spp, NOEC, 0.034-7.1 mg/L. The lowest measured chronic figure was an 8-d LC50 of 0.17 mg/L for *Micropterus* sp.

Amphibian: Acute LC50 (4 d): *Bufo americanus*, 0.86-1.66 mg/L; Chronic LC50 (8-d) 2.28 mg/L

Crustaceans LC50 (48 h): 1 sp 2.3-36 9 mg/L; Chronic NOEC (7-28 d) 3 spp, 0.136-1.72 mg/L

Algae EC50 (96 h): population growth, 0.46-0.57 mg/L; 2 spp, chronic NOEC, 0.8-2.0 mg/L

Freshwater species pH <6.5 (all between pH 4.5 and 6.0)

Fish LC50 (24-96 h): 4 spp, 0.015 (*S. trutta*) - 4.2 mg/L; chronic data on *Salmo trutta*, LC50 (21-42 d) 0.015- 0.105 mg/L

Amphibians LC50 (4-5 d): 2 spp, 0.540-2.670 mg/L (absolute range 0.40-5.2 mg/L)

Alga: 1 sp NOEC growth 2.0 mg/L

Among freshwater aquatic plants, single-celled plants are generally the most sensitive to aluminium. Fish are generally more sensitive to aluminium than aquatic invertebrates.

Aluminium is a gill toxicant to fish, causing both ionoregulatory and respiratory effects.

The bioavailability and toxicity of aluminium is generally greatest in acid solutions. Aluminium in acid habitats has been observed to be toxic to fish and phytoplankton. Aluminium is generally more toxic over the pH range 4.4-5.4, with a maximum toxicity occurring around pH 5.0-5.2. The inorganic single unit aluminium species $Al(OH)_2^+$ is thought to be the most toxic. Under very acid conditions, the toxic effects of the high H^+ concentration appear to be more important than the effects of low concentrations of aluminium; at approximately neutral pH values, the toxicity of aluminium is greatly reduced. The solubility of aluminium is also enhanced under alkaline conditions, due to its amphoteric character, and some researchers found that the acute toxicity of aluminium increased from pH 7 to pH 9. However, the opposite relationship was found in other studies. The uptake and toxicity of aluminium in freshwater organisms generally decreases with increasing water hardness under acidic, neutral and alkaline conditions. Complexing agents such as fluoride, citrate and humic substances reduce the availability of aluminium to organisms, resulting in lower toxicity. Silicon can also reduce aluminium toxicity to fish.

Drinking Water Standards:

aluminium: 200 ug/l (UK max.)

200 ug/l (WHO guideline)

chloride: 400 mg/l (UK max.)

250 mg/l (WHO guideline)

fluoride: 1.5 mg/l (UK max.)

1.5 mg/l (WHO guideline)

nitrate: 50 mg/l (UK max.)

50 mg/l (WHO guideline)

sulfate: 250 mg/l (UK max.)

Soil Guideline: none available.

Air Quality Standards: none available.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
	No Data available for all ingredients	No Data available for all ingredients

Bioaccumulative potential

Ingredient	Bioaccumulation
	No Data available for all ingredients

Mobility in soil

Ingredient	Mobility
	No Data available for all ingredients

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. Recycle wherever possible or consult manufacturer for recycling options. Consult State Land Waste Management Authority for disposal. Bury residue in an authorised landfill. Recycle containers if possible, or dispose of in an authorised landfill.
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SECTION 14 Transport information**Labels Required**

Marine Pollutant	NO
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Land transport (DOT): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS**Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS****Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS****Transport in bulk according to Annex II of MARPOL and the IBC code**

Not Applicable

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

Product name	Group
calcium hydroxide	Not Available
silica crystalline - quartz	Not Available
aluminium	Not Available
calcium oxide	Not Available
portland cement	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
calcium hydroxide	Not Available
silica crystalline - quartz	Not Available
aluminium	Not Available
calcium oxide	Not Available
portland cement	Not Available

SECTION 15 Regulatory information**Safety, health and environmental regulations / legislation specific for the substance or mixture****calcium hydroxide is found on the following regulatory lists**

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

US - Alaska Air Quality Control - Concentrations Triggering an Air Quality Episode for Air Pollutants Other Than PM-2.5

US - Massachusetts - Right To Know Listed Chemicals

US ACGIH Threshold Limit Values (TLV)

US DOE Temporary Emergency Exposure Limits (TEELs)

US NIOSH Recommended Exposure Limits (RELs)

US OSHA Permissible Exposure Limits (PELs) Table Z-1

US OSHA Permissible Exposure Limits (PELs) Table Z-3

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

US TSCA Chemical Substance Inventory - Interim List of Active Substances

silica crystalline - quartz is found on the following regulatory lists

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

US - California Proposition 65 - Carcinogens

US - California Safe Drinking Water and Toxic Enforcement Act of 1986 - Proposition 65 List

US - Massachusetts - Right To Know Listed Chemicals

US ACGIH Threshold Limit Values (TLV)

US ACGIH Threshold Limit Values (TLV) - Carcinogens

US DOE Temporary Emergency Exposure Limits (TEELs)

US National Toxicology Program (NTP) 15th Report Part A Known to be Human Carcinogens

US NIOSH Carcinogen List

US NIOSH Recommended Exposure Limits (RELs)

US OSHA Carcinogens Listing

US OSHA Permissible Exposure Limits (PELs) Table Z-3

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

US TSCA Chemical Substance Inventory - Interim List of Active Substances

aluminium is found on the following regulatory lists

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

US - Alaska Air Quality Control - Concentrations Triggering an Air Quality Episode for Air Pollutants Other Than PM-2.5

US - Massachusetts - Right To Know Listed Chemicals

US ACGIH Threshold Limit Values (TLV)

US ACGIH Threshold Limit Values (TLV) - Carcinogens

US ATSDR Minimal Risk Levels for Hazardous Substances (MRLs)

US Department of Homeland Security (DHS) - Chemical Facility Anti-Terrorism Standards (CFATS) - Chemicals of Interest

US EPCRA Section 313 Chemical List

US NIOSH Recommended Exposure Limits (RELs)

US OSHA Permissible Exposure Limits (PELs) Table Z-1

US OSHA Permissible Exposure Limits (PELs) Table Z-3

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

US TSCA Chemical Substance Inventory - Interim List of Active Substances

calcium oxide is found on the following regulatory lists

US - Massachusetts - Right To Know Listed Chemicals

US ACGIH Threshold Limit Values (TLV)

US DOE Temporary Emergency Exposure Limits (TEELs)

US NIOSH Recommended Exposure Limits (RELs)

US OSHA Permissible Exposure Limits (PELs) Table Z-1

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

US TSCA Chemical Substance Inventory - Interim List of Active Substances

portland cement is found on the following regulatory lists

US - Massachusetts - Right To Know Listed Chemicals

US ACGIH Threshold Limit Values (TLV)

US ACGIH Threshold Limit Values (TLV) - Carcinogens

US NIOSH Recommended Exposure Limits (RELs)

US OSHA Permissible Exposure Limits (PELs) Table Z-1

US OSHA Permissible Exposure Limits (PELs) Table Z-3

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

US TSCA Chemical Substance Inventory - Interim List of Active Substances

Federal Regulations

Continued...

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Superfund Amendments and Reauthorization Act of 1986 (SARA)

Section 311/312 hazard categories


Flammable (Gases, Aerosols, Liquids, or Solids)	No
Gas under pressure	No
Explosive	No
Self-heating	No
Pyrophoric (Liquid or Solid)	No
Pyrophoric Gas	No
Corrosive to metal	No
Oxidizer (Liquid, Solid or Gas)	No
Organic Peroxide	No
Self-reactive	No
In contact with water emits flammable gas	No
Combustible Dust	No
Carcinogenicity	Yes
Acute toxicity (any route of exposure)	No
Reproductive toxicity	No
Skin Corrosion or Irritation	Yes
Respiratory or Skin Sensitization	Yes
Serious eye damage or eye irritation	Yes
Specific target organ toxicity (single or repeated exposure)	Yes
Aspiration Hazard	No
Germ cell mutagenicity	Yes
Simple Asphyxiant	No
Hazards Not Otherwise Classified	No

US. EPA CERCLA Hazardous Substances and Reportable Quantities (40 CFR 302.4)

None Reported

State Regulations

US. California Proposition 65

 **WARNING:** This product can expose you to chemicals including **silica crystalline - quartz**, which is known to the State of California to cause cancer. For more information, go to www.P65Warnings.ca.gov.

National Inventory Status

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

SECTION 16 Other information

Revision Date	05/02/2022
Initial Date	08/16/2021

CONTACT POINT

PLEASE NOTE THAT TITANIUM DIOXIDE IS NOT PRESENT IN CLEAR OR NEUTRAL BASES

SDS Version Summary

Continued...

Castor Crete SL Bags

Version	Date of Update	Sections Updated
2.7	05/02/2022	Ingredients

Other information

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

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

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit.
IDLH: Immediately Dangerous to Life or Health Concentrations
ES: Exposure Standard
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index
AIIIC: Australian Inventory of Industrial Chemicals
DSL: Domestic Substances List
NDSL: Non-Domestic Substances List
IECSC: Inventory of Existing Chemical Substance in China
EINECS: European INventory of Existing Commercial chemical Substances
ELINCS: European List of Notified Chemical Substances
NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory
KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals
PICCS: Philippine Inventory of Chemicals and Chemical Substances
TSCA: Toxic Substances Control Act
TCSI: Taiwan Chemical Substance Inventory
INSQ: Inventario Nacional de Sustancias Químicas
NCI: National Chemical Inventory
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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