

Tradies Choice Masterflex Extra

SAS Australasia (Specialty Adhesives Systems)

Part Number: **Not Available**

Version No: **4.6**

Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

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

Product Identifier

Product name	Tradies Choice Masterflex Extra
Synonyms	Tile adhesive
Other means of identification	Not Available

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

Relevant identified uses	TC Masterflex Extra silica free, polymer modified, cement based tile adhesive designed for internal and external applications. It can be used for ceramic, porcelain and vitrified tiles. Use according to manufacturer's directions.
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Details of the manufacturer or supplier of the safety data sheet

Registered company name	SAS Australasia (Specialty Adhesives Systems)
Address	Unit A2/152 Miller Rd Chester Hill NSW 2162 Australia
Telephone	1300 202 894 (Mon-Fri, 9am-5pm)
Fax	Not Available
Website	https://sas-aa.com.au/
Email	techdept@sas-aa.com.au

Emergency telephone number

Association / Organisation	SAS Australasia (Specialty Adhesives Systems)
Emergency telephone number(s)	1300 202 894 (Mon-Fri, 9am-5pm)
Other emergency telephone number(s)	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Poisons Schedule	Not Applicable
Classification ^[1]	Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Germ Cell Mutagenicity Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 2
Legend:	1. Classification by vendor; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
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TC Masterflex Extra**Signal word** **Danger****Hazard statement(s)**

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

Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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SECTION 3 Composition / information on ingredients**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
65997-15-1	30-60	<u>portland cement</u>
471-34-1	30-60	<u>calcium carbonate</u>
544-17-2	<10	<u>calcium formate</u>
9004-65-3	<10	<u>hydroxypropyl methylcellulose</u>

Legend: 1. Classification by vendor; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available

SECTION 4 First aid measures**Description of first aid measures**

Eye Contact	If this product comes in contact with the eyes:
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	<ul style="list-style-type: none"> ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay.
Ingestion	<ul style="list-style-type: none"> ▶ Immediately give a glass of water. ▶ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

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

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

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

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

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

[Ellenhorn and Barceloux: Medical Toxicology]

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

[Ellenhorn and Barceloux: Medical Toxicology]

For acute or short-term repeated exposures to highly alkaline materials:

- ▶ Respiratory stress is uncommon but present occasionally because of soft tissue edema.
- ▶ Unless endotracheal intubation can be accomplished under direct vision, cricothyroidotomy or tracheotomy may be necessary.
- ▶ Oxygen is given as indicated.
- ▶ The presence of shock suggests perforation and mandates an intravenous line and fluid administration.
- ▶ Damage due to alkaline corrosives occurs by liquefaction necrosis whereby the saponification of fats and solubilisation of proteins allow deep penetration into the tissue.

Alkalis continue to cause damage after exposure.

INGESTION:

- ▶ Milk and water are the preferred diluents

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

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

* Catharsis and emesis are absolutely contra-indicated.

* Activated charcoal does not absorb alkali.

* Gastric lavage should not be used.

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Supportive care involves the following:

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

SKIN AND EYE:

- ▶ Injury should be irrigated for 20-30 minutes.
- Eye injuries require saline. [Ellenhorn & Barceloux: Medical Toxicology]

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

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

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

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	When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles. May emit poisonous fumes. May emit corrosive fumes. Heating calcium carbonate at high temperatures(825 C.) causes decomposition, releases carbon dioxide gas and leaves a residue of alkaline lime
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none"> ▶ Clean up all 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. ▶ Sweep up, shovel up or ▶ Vacuum up (consider explosion-proof machines designed to be grounded during storage and use). ▶ Place spilled material in clean, dry, sealable, labelled container.
Major Spills	<p>Moderate hazard.</p> <ul style="list-style-type: none"> ▶ CAUTION: Advise personnel in area. ▶ Alert Emergency Services and tell them location and nature of hazard. ▶ Control personal contact by wearing protective clothing. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Recover product wherever possible. ▶ IF DRY: Use dry clean up procedures and avoid generating dust. Collect residues and place in sealed plastic bags or other containers for disposal. IF WET: Vacuum/shovel up and place in labelled containers for disposal. ▶ ALWAYS: Wash area down with large amounts of water and prevent runoff into drains. ▶ If contamination of drains or waterways occurs, advise Emergency Services.

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

SECTION 7 Handling and storage

Precautions for safe handling

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

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Polyethylene or polypropylene container. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<p>For aluminas (aluminium oxide): 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.</p> <p>Aluminium oxide is an amphoteric substance, meaning it can react with both acids and bases, such as hydrofluoric acid and sodium hydroxide, acting as an acid with a base and a base with an acid, neutralising the other and producing a salt.</p> <p>Calcium carbonate:</p> <ul style="list-style-type: none"> ▶ is incompatible with acids, ammonium salts, fluorine, germanium, lead diacetate, magnesium, mercurous chloride, silicon, silver nitrate, titanium. <p>Contact with acid generates carbon dioxide gas, which may pressurise and then rupture closed containers</p> <p>Calcium oxide:</p> <ul style="list-style-type: none"> ▶ reacts violently with water, evolving high quantities of heat ▶ reacts violently, with possible ignition or explosion, with acids, anilinium perchlorate, bromine pentafluoride, chlorine trifluoride, fluorine, hydrogen fluoride, hydrazine, hydrogen sulfide, hydrogen trisulfide, isopropyl isocyanide dichloride, light metals, lithium, magnesium, powdered aluminium, phosphorus, potassium, sulfur trioxide ▶ increase the explosive sensitivity of azides, nitroalkanes (e.g. nitroethane, nitromethane, 1-nitropropane etc.) ▶ is incompatible with boric acid, boron trifluoride, carbon dioxide, ethanol, halogens (such as fluorine), metal halides, phosphorus pentoxide, selenium oxychloride, sulfur dioxide and many organic materials <p>Calcium sulfate:</p> <ul style="list-style-type: none"> ▶ reacts violently with reducing agents, acrolein, alcohols, chlorine trifluoride, diazomethane, ethers, fluorine, hydrazine, hydrazinium perchlorate, hydrogen peroxide, finely divided aluminium or magnesium, peroxyfuroic acid, red phosphorus, sodium acetylide ▶ sensitises most organic azides which are unstable shock- and heat- sensitive explosives ▶ may form explosive materials with 1,3-di(5-tetrazolyl)triazene ▶ is incompatible with glycidol, isopropyl chlorocarbonate, nitrosyl perchlorate, sodium borohydride ▶ is hygroscopic; reacts with water to form gypsum and Plaster of Paris <p>For iron oxide (ferric oxide):</p> <ul style="list-style-type: none"> ▶ Avoid storage with aluminium, calcium hypochlorite and ethylene oxide. ▶ Risk of explosion occurs following reaction with powdered aluminium, calcium silicide, ethylene oxide (polymerises), carbon monoxide, magnesium and perchlorates. ▶ Risk of ignition or formation of flammable gases or vapours occurs following reaction with carbides, for example caesium carbide, (produces heat), hydrogen sulfide, hydrogen peroxide (decomposes). ▶ An intimately powdered mixture with aluminium, usually ignited by magnesium ribbon, reacts with an intense exotherm to produce molten iron in the commercial "thermit" welding process ▶ WARNING: Avoid or control reaction with peroxides. All <i>transition metal</i> peroxides should be considered as potentially explosive. For example transition metal complexes of alkyl hydroperoxides may decompose explosively. ▶ The pi-complexes formed between chromium(0), vanadium(0) and other transition metals (haloarene-metal complexes) and mono- or poly-fluorobenzene show extreme sensitivity to heat and are explosive. ▶ Avoid reaction with borohydrides or cyanoborohydrides <p>Cellulose and its derivatives may react vigorously with calcium oxide, bleaching powder, perchlorates, perchloric acid, sodium chlorate, fluorine, nitric acid, sodium nitrate and sodium nitrite. May be incompatible with aminacrine hydrochloride, chlorocresol, mercuric chloride, phenol, resorcinol, tannic acid and silver nitrate.</p>

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



X — Must not be stored together

O — May be stored together with specific preventions

+ — May be stored together

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	portland cement	Portland cement	10 mg/m ³	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	calcium carbonate	Calcium carbonate	10 mg/m ³	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.

Ingredient	Original IDLH	Revised IDLH
portland cement	5,000 mg/m ³	Not Available
calcium carbonate	Not Available	Not Available
calcium formate	Not Available	Not Available
hydroxypropyl methylcellulose	Not Available	Not Available

MATERIAL DATA

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)

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

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

For calcium carbonate:

The TLV-TWA is thought to be protective against the significant risk of physical irritation associated with exposure.

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.

For aluminium oxide:

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

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

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

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

Exposure controls

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<p>Appropriate engineering controls</p>	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.</p> <ul style="list-style-type: none"> ▶ Local exhaust ventilation is required where solids are handled as powders or crystals; even when particulates are relatively large, a certain proportion will be powdered by mutual friction. ▶ If in spite of local exhaust an adverse concentration of the substance in air could occur, respiratory protection should be considered. <p>Such protection might consist of:</p> <p>(a): particle dust respirators, if necessary, combined with an absorption cartridge;</p> <p>(b): filter respirators with absorption cartridge or canister of the right type;</p> <p>(c): fresh-air hoods or masks.</p> <p>Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p> <table border="1" data-bbox="391 719 1492 880"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="391 936 1177 1115"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 4-10 m/s (800-2000 f/min) for extraction of crusher dusts generated 2 metres distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	Type of Contaminant:	Air Speed:	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use	4: Large hood or large air mass in motion	4: Small hood-local control only
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<p>Individual protection measures, such as personal protective equipment</p>																	
<p>Eye and face protection</p>	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ▶ 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]. 																
<p>Skin protection</p>	<p>See Hand protection below</p>																
<p>Hands/feet protection</p>	<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>																

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	<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"> ▶ Neoprene rubber gloves <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"> ▶ Overalls. ▶ P.V.C apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit.

Respiratory protection

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	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)

- 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.

Class P2 particulate filters are used for protection against mechanically and thermally generated particulates or both.

P2 is a respiratory filter rating under various international standards, Filters at least 94% of airborne particles

Suitable for:

- Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.

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- 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

SECTION 9 Physical and chemical properties**Information on basic physical and chemical properties**

Appearance	White alkaline powder; soluble in water.		
Physical state	Divided Solid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Available
Flash point (°C)	Not Available	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	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 (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Heat of Combustion (kJ/g)	Not Available	Ignition Distance (cm)	Not Available
Flame Height (cm)	Not Available	Flame Duration (s)	Not Available
Enclosed Space Ignition Time Equivalent (s/m³)	Not Available	Enclosed Space Ignition Deflagration Density (g/m³)	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information**Information on toxicological effects**

a) Acute Toxicity	Based on available data, the classification criteria are not met.
b) Skin Irritation/Corrosion	There is sufficient evidence to classify this material as skin corrosive or irritating.
c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	There is sufficient evidence to classify this material as sensitising to skin or the respiratory system
e) Mutagenicity	There is sufficient evidence to classify this material as mutagenic
f) Carcinogenicity	Based on available data, the classification criteria are not met.
g) Reproductivity	Based on available data, the classification criteria are not met.

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h) STOT - Single Exposure	There is sufficient evidence to classify this material as toxic to specific organs through single exposure
i) STOT - Repeated Exposure	There is sufficient evidence to classify this material as toxic to specific organs through repeated exposure
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	<p>Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>Cellulose, after a single intratracheal dose (15 mg per animal) brought about fibrosing granulomatous bronchioloalveolitis and an increase of IgA production in the bronchioalveolar lavage. Fibrosing alveolitis showed moderate progression as a function of time. Injury of Type I pneumocytes and incomplete repair of Type II pneumocytes were detected. The damage of alveolar epithelium initiated and activated a series of processes that led to definite pulmonary alterations and pulmonary fibrosis leading to disintegration of the alveolo-capillary morphological functional unit.</p> <p>Tatrai, E. et al: Journal of Applied Toxicology; 16(2) 129-135 (1996)</p> <p>Some health effects associated with wood, cotton, flax, jute and hemp particles or fibres are not attributable to cellulose content but to other substances and/or impurities.</p> <p>Inhalation may result in chrome ulcers or sores of nasal mucosa and lung damage.</p> <p>Persons with impaired respiratory function, airway diseases and conditions such as emphysema or chronic bronchitis, may incur further disability if excessive concentrations of particulate are inhaled.</p> <p>If prior damage to the circulatory or nervous systems has occurred or if kidney damage has been sustained, proper screenings should be conducted on individuals who may be exposed to further risk if handling and use of the material result in excessive exposures.</p> <p>Effects on lungs are significantly enhanced in the presence of respirable particles. Overexposure to respirable dust may produce wheezing, coughing and breathing difficulties leading to or symptomatic of impaired respiratory function.</p> <p>Inhalation of dusts, generated by the material during the course of normal handling, may be damaging to the health of the individual.</p>
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Ingestion	<p>Large doses of cellulose may be administered orally as non-nutritive bulk. Doses of up to 30 g/day can be tolerated as bulk laxative. Extremely large oral doses may produce gastrointestinal disturbances.</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>The material has NOT been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.</p> <p>Accidental ingestion of the material may be damaging to the health of the individual.</p>
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Skin Contact	<p>The material may accentuate any pre-existing dermatitis condition</p> <p>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.</p> <p>Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus.</p> <p>Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles.</p> <p>Four students received severe hand burns whilst making moulds of their hands with dental plaster substituted for Plaster of Paris. The dental plaster known as "Stone" was a special form of calcium sulfate hemihydrate containing alpha-hemihydrate crystals that provide high compression strength to the moulds. Beta-hemihydrate (normal Plaster of Paris) does not cause skin burns in similar circumstances.</p> <p>Handling wet cement can cause dermatitis. Cement when wet is quite alkaline and this alkali action on the skin contributes strongly to cement contact dermatitis since it may cause drying and defatting of the skin which is followed by hardening, cracking, lesions developing, possible infections of lesions and penetration by soluble salts.</p> <p>Skin contact may result in severe irritation particularly to broken skin. Ulceration known as "chrome ulcers" may develop. 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> <p>The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either</p> <ul style="list-style-type: none"> ▸ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or ▸ produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. <p>Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p>
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Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
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Chronic	<p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure.</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>
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Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.

Substances 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.

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.

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.

Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.

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.

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminum oxide during abrasives production.

Very fine Al₂O₃ powder was not fibrogenic in rats, guinea pigs, or hamsters when inhaled for 6 to 12 months and sacrificed at periods up to 12 months following the last exposure.

When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C.

The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis (aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity.

Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction.

There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveoli (sub 5 um) are able to produce pathogenic effects in the lungs.

Red blood cells and rabbit alveolar macrophages exposed to calcium silicate insulation materials in vitro showed haemolysis in one study but not in another. Both studies showed the substance to be more cytotoxic than titanium dioxide but less toxic than asbestos.

In a small cohort mortality study of workers in a wollastonite quarry, the observed number of deaths from all cancers combined and lung cancer were lower than expected. Wollastonite is a calcium inosilicate mineral (CaSiO₃). In some cases, small amounts of iron (Fe), and manganese (Mn), and lesser amounts of magnesium (Mg) substitute for calcium (Ca) in the mineral formulae (e.g., rhodonite).

In an inhalation study in rats no increase in tumour incidence was observed but the number of fibres with lengths exceeding 5 um and a diameter of less than 3 um was relatively low. Four grades of wollastonite of different fibre size were tested for carcinogenicity in one experiment in rats by intrapleural implantation. There was no information on the purity of the four samples used. A slight increase in the incidence of pleural sarcomas was observed with three grades, all of which contained fibres greater than 4 um in length and less than 0.5 um in diameter.

In two studies by intraperitoneal injection in rats using wollastonite with median fibre lengths of 8.1 um and 5.6 um respectively, no intra-abdominal tumours were found.

Evidence from wollastonite miners suggests that occupational exposure can cause impaired respiratory function and pneumoconiosis. However animal studies have demonstrated that wollastonite fibres have low biopersistence and induce a transient inflammatory response compared to various forms of asbestos. A two-year inhalation study in rats at one dose showed no significant inflammation or fibrosis.

Cement contact dermatitis (CCD) may occur when contact shows an allergic response, which may progress to sensitisation.

Sensitisation is due to soluble chromates (chromate compounds) present in trace amounts in some cements and cement products. Soluble chromates readily penetrate intact skin. Cement dermatitis can be characterised by fissures, eczematous rash, dystrophic nails, and dry skin; acute contact with highly alkaline mixtures may cause localised necrosis.

Cement eczema may be due to chromium in feed stocks or contamination from materials of construction used in processing the cement. Sensitisation to chromium may be the leading cause of nickel and cobalt sensitivity and the high alkalinity of cement is an important factor in cement dermatoses [ILO].

Repeated, prolonged severe inhalation exposure may cause pulmonary oedema and rarely, pulmonary fibrosis. Workers may also suffer from dust-induced bronchitis with chronic bronchitis reported in 17% of a group occupationally exposed to high dust levels.

Respiratory symptoms and ventilatory function were studied in a group of 591 male Portland cement workers employed in four Taiwanese cement plants, with at least 5 years of exposure (1). This group had a significantly lowered mean forced vital capacity (FCV), forced expiratory volume at 1 s (FEV₁) and forced expiratory flows after exhalation of 50% and 75% of the vital capacity

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(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

The cellulose derivatives pass essentially unchanged through the gastrointestinal tract following oral administration to rats, dogs and man. Acute, subchronic, chronic toxicity, reproductive and developmental toxicity, genotoxicity and carcinogenicity studies of cellulose derivatives indicated that they are practically non-toxic when administered by oral, intraperitoneal, subcutaneous or dermal routes. While no clinical inhalation studies have been conducted, long term exposure to the dusts of cellulose ethers in manufacturing operations has not led to any significant adverse effects. Ocular and dermal irritation studies indicate that the cellulose derivatives are, at most, minimally irritating and are not dermal sensitizers. Clinical studies confirm these results.

Amended Safety Assessment of Cellulose and Related Polymers as used in Cosmetics: Final Report of the Cosmetic Ingredient Review (CIR) Expert Panel: March 2009

Inhalation studies indicate that cellulose fibres may be fibrogenic; this finding continues to be the subject of extensive research.

Cellulose is not considered an inert substance because :

- in rats, it causes granulomatous fibrosing alveolitis at the end of the third month after exposure,
- in rats there was an increase in the secretion of plasminogen activator and interleukin 1 as well as the release of lactate dehydrogenase from macrophages, in a manner similar to asbestos,
- there were increases in the incidence of obstructive lung diseases and bronchial asthma in humans at work and in the residential environment where exposure to cellulose was common,
- the substance may induce free radical production in human leucocytes.

Byssinosis is an occupational disease of the lungs caused by inhalation of cotton dust or dusts from other vegetable fibres such as flax, hemp, or sisal. Byssinosis is a chronic, asthma-like narrowing of the airways. Also called brown lung disease, byssinosis occurs almost exclusively in people who work with unprocessed cotton.

Cotton dust disease, "byssinosis", is well known among cotton mill workers. Cotton dust consists largely of cellulose fibre.

Exposure to two components of the total dust, the "respirable" and "medium" fraction correlated significantly with the prevalence of respiratory symptoms. Inhalation exposure to a concentration of 0.3 to 0.4 mg/m³ of "fly-free" dust results in a 20% occurrence of byssinosis. "Fly-free" dust is the sum of respirable and medium-length fibres. At 0.46 mg/m³, Grade II byssinosis occurs. A byssinosis (all grades) prevalence of 20%, at 0.3 mg/m³ occurs when the fibre length is less than 15 µm (aerodynamic equivalent diameter). Byssinosis is not caused by mechanical irritation but by reactions caused by pharmacologically active substances producing oedema or contraction of the smooth musculature of the airways. The causative agent is suspected to be an endotoxin, in turn, thought to be a cell wall component of bacteria found in cotton. Symptoms of byssinosis include chest tightness, wheezing and dyspnoea. Symptoms usually appear after an absence from work and may subside after 2-days of exposure. As the disease progresses, symptoms may persist for longer periods until they are constant. The individual may eventually exhibit chronic bronchitis and emphysema. Increased physical exertion may produce shortness of breath.

Studies indicate that diets containing large amounts of non-absorbable polysaccharides, such as cellulose, might decrease absorption of calcium, magnesium, zinc and phosphorus.

Polysaccharides are polymeric carbohydrates that consist of monosaccharide units, which are connected together with glycosidic bonds. Due to the structural variation of different monosaccharides as well as the innumerable ways that these building blocks link with each other, polysaccharides can be considered as structurally complex biomacromolecules. Polysaccharides originating from plants (e.g., starch and guar gum), microbes (e.g., xanthan), algae (e.g., alginates and carrageenans) and animals (e.g., glycogen and chitin) are frequently used in food. Starch, a high molar mass compound consisting of (1->4)-linked alpha-D-glucopyranosyl units, is an important energy nutrient that is abundant in common foods, such as cereals and root crops. Although many other food polysaccharides are not digested in the upper gastrointestinal tract of humans, they often serve functions other than being components giving nutritional value. For example, plant cell-wall polysaccharides, such as arabinoxylans and beta-glucan, exist in cereal-based foods, and "plant gums" are used as thickeners, emulsifiers, emulsion stabilizers, gelling agents and encapsulating agents. These non-digestible polysaccharides are important for health because they are considered as dietary fibre, which promote colon health, regulate post-prandial blood glucose levels and reduce serum cholesterol levels.

Despite the fact that nature provides various sources of polysaccharides, and that scientific research on their exploitation as food materials is increasingly active, a relatively low number of polysaccharides are authorized for use as food ingredients. For example, in the European Union (EU) and in Switzerland, among the permitted food additives (identified by an E number) only a small percentage are polysaccharide-based (native or structurally modified). The difference between other food ingredients and food additives is mainly the quantity used in any given product. Food ingredients can be consumed alone as food (e.g., starch), whereas food additives (e.g., carboxymethyl cellulose) are used in small quantities (usually less than 2%) relative to the total food composition but they, nonetheless, play an important role in the food products. Regarding food additive use in Europe, the European Food Safety Authority (EFSA) has an expert Panel on Food Additives and Nutrient Sources Added to Food (ANS), which evaluates the safety of food additives. Similarly, if new ingredients are released into the market, EFSA's Panel on Dietetic Products, Nutrition and Allergies (NDA) has the responsibility of evaluating the safety of Novel Food ingredients

The vast majority of polysaccharides used as food ingredients are plant-based. In addition to the cellulosic polysaccharides, other types of food-grade ingredients or additives, such as, vanillin aroma, glycerol esters of wood rosin (E445), xylitol (E967) and sterols/stanols, are derived from wood. The main components of wood are polysaccharides: cellulose (40–50 wt%) and hemicelluloses (20–35%), while lignin comprises 15–30% of wood mass.

The material contains a substantial proportion of a polymer considered to be of low concern (PLC). The trend towards production of lower molecular weight polymers (thus reducing the required level of solvent use and creating a more "environmentally-friendly" material) has brought with it the need to define PLCs as those

having molecular weights of between 1000 and 10000 and containing less than 10% of the molecules with molecular weight below 500 and less than 25% of the molecules with a molecular weight below 1000. These may contain unlimited low concern functional groups or moderate concern reactive functional groups with a combined functional group equivalent weight (FGEW, a concept developed by the US EPA describing whether the reactive functional group is sufficiently diluted by polymeric material) of a 1000 or more (provided no high concern groups are present) or high concern reactive functional groups with a FGEW of 5000 or more (FGEW includes moderate concern groups if present).

having molecular weights exceeding 10000 (without restriction on reactive groups).

inhalation of polymers with molecular weights > 70,000 Da has been linked with irreversible lung damage due to lung overloading and impaired clearance of particles from the lung, particularly following repeated exposure. If the polymer is inhaled at low levels and/or infrequently, it is assumed that it will be cleared from the lungs.

Reactive functional groups are in turn classified as being of low, moderate or high concern Classification of the polymer as a PLC, in accordance with established criteria, does not mean that hazards will not be associated with the polymer (during its import, manufacture, use, storage, handling or disposal). The polymer may, for example, contain a large number of particles in

TC Masterflex Extra

the respirable range, a hazard which may need to be assessed in the health and safety risk assessment. Similarly a polymer with low concern reactive may be released into the environment in large quantities and produce an environmental hazard.

Whilst it is generally accepted that polymers with a molecular weight exceeding 1000 are unlikely to pass through biological membranes, oligomers with lower molecular weight and specifically, those with a molecular weight below 500, may. Estimations based on a "highly" dispersed polymer population (polydispersity = 10) suggests that the molecular weight of the polymer carrying a reactive group of high concern must be 5000 to be considered a PLC; similarly a polymer of approximate molecular weight 1000 could contain no more than one reactive group of moderate concern (for two moderate concern groups, the molecular weight would be about 2500).

Pure calcium carbonate does not produce pneumoconiosis probably being eliminated from the lungs slowly by solution.

As mined, unsterilised particulates can carry bacteria into the air passages and lungs, producing infection and bronchitis.

High blood concentrations of calcium ion may give rise to vasodilation and depress cardiac function leading to hypotension and syncope. Calcium ions enhance the effects of digitalis on the heart and may precipitate digitalis intoxication. Calcium salts also reduce the absorption of tetracyclines

In neonates calcification of soft-tissue has been observed following therapeutic administration.

Some studies show that large quantities of calcium intake can cause hypercalcaemia, which can in turn lead to renal failure. Renal failure can occur within hours or days or, alternatively, settles gradually, evolving over several years until it reaches terminal stages. Similarly, acute renal failure can also develop into chronic forms of the disease.

Hypercalcaemia conditions can be associated with normal or reduced calcium serum levels, as the body tends to maintain a balanced metabolism of the mineral, known as the compensation phase. When there is a slight increase in the concentration of ions in the blood, calcium excretion markedly increases, while intestinal absorption decreases. After kidney damage has set in, a loss of calcium may occur, thereby decreasing the serum concentration.

Serum protein levels may decrease as a result of proteinuria in cases of renal complications. Proteinuria is an indicator of kidney disease and represents an independent risk factor for the progression of such a condition. Increased serum creatinine levels may represent an important parameter, given that kidney diseases are associated with increased serum creatinine levels. When renal pathology occurs, a progressive loss of glomerular filtration begins, resulting in increased plasma creatinine concentrations.

During the course of kidney failure, discrete, but constant, increments in plasma creatinine levels occur.

Renal disease with albuminuria may also be the cause of hypoalbuminemia in patients with liver disease. In cases of established liver damage, increased calcium urinary excretion may occur. Therefore, a similar increase may cause the decline in serum calcium levels in the current study.

Overexposure to the breathable dust may cause coughing, wheezing, difficulty in breathing and impaired lung function. Chronic symptoms may include decreased vital lung capacity and chest infections. Repeated exposures in the workplace to high levels of fine-divided dusts may produce a condition known as pneumoconiosis, which is the lodgement of any inhaled dusts in the lung, irrespective of the effect. This is particularly true when a significant number of particles less than 0.5 microns (1/50000 inch) are present. Lung shadows are seen in the X-ray. Symptoms of pneumoconiosis may include a progressive dry cough, shortness of breath on exertion, increased chest expansion, weakness and weight loss. As the disease progresses, the cough produces stringy phlegm, vital capacity decreases further, and shortness of breath becomes more severe. Other signs or symptoms include changed breath sounds, reduced oxygen uptake during exercise, emphysema and rarely, pneumothorax (air in the lung cavity).

Removing workers from the possibility of further exposure to dust generally stops the progress of lung abnormalities. When there is high potential for worker exposure, examinations at regular period with emphasis on lung function should be performed.

Inhaling dust over an extended number of years may cause pneumoconiosis, which is the accumulation of dusts in the lungs and the subsequent tissue reaction. This may or may not be reversible.

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.

Chronic excessive iron exposure has been associated with haemosiderosis and consequent possible damage to the liver and pancreas. Haemosiderin is a golden-brown insoluble protein produced by phagocytic digestion of haematin (an iron-based pigment). Haemosiderin is found in most tissues, especially in the liver, in the form of granules. Other sites of haemosiderin deposition include the pancreas and skin. A related condition, haemochromatosis, which involves a disorder of metabolism of these deposits, may produce cirrhosis of the liver, diabetes, and bronze pigmentation of the skin - heart failure may eventually occur.

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Such exposure may also produce conjunctivitis, choroiditis, retinitis (both inflammatory conditions involving the eye) and siderosis of tissues if iron remains in these tissues. Siderosis is a form of pneumoconiosis produced by iron dusts. Siderosis also includes discoloration of organs, excess circulating iron and degeneration of the retina, lens and uvea as a result of the deposition of intraocular iron. Siderosis might also involve the lungs - involvement rarely develops before ten years of regular exposure. Often there is an accompanying inflammatory reaction of the bronchi. Permanent scarring of the lungs does not normally occur.

High levels of iron may raise the risk of cancer. This concern stems from the theory that iron causes oxidative damage to tissues and organs by generating highly reactive chemicals, called free radicals, which subsequently react with DNA. Cells may be disrupted and may become cancerous. People whose genetic disposition prevents them from keeping tight control over iron (e.g. those with the inherited disorder, haemochromatosis) may be at increased risk.

Iron overload in men may lead to diabetes, arthritis, liver cancer, heart irregularities and problems with other organs as iron builds up.

[K. Schmidt, New Scientist, No. 1919 pp.11-12, 2nd April, 1994]

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

TC Masterflex Extra	TOXICITY	IRRITATION
	Not Available	Not Available
portland cement	TOXICITY	IRRITATION
	Not Available	Not Available
calcium carbonate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (Rodent - rabbit): 750ug/24H - Severe
	Inhalation (Rat) LC50: >3 mg/4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Skin (Rodent - rabbit): 500mg/24H - Moderate
		Skin: no adverse effect observed (not irritating) ^[1]
calcium formate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (Rodent - rabbit): 100mg/24H - Moderate
	Inhalation (Rat) LC50: >0.67 mg/4h ^[1]	Eye: adverse effect observed (irritating) ^[1]
	Oral (Mouse) LD50: 1920 mg/kg ^[2]	Skin: no adverse effect observed (not irritating) ^[1]
hydroxypropyl methylcellulose	TOXICITY	IRRITATION
	Oral (Rat) LD50: >10000 mg/kg ^[2]	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

TC MASTERFLEX EXTRA	Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.
PORTLAND CEMENT	No significant acute toxicological data identified in literature search.
CALCIUM CARBONATE	No evidence of carcinogenic properties. No evidence of mutagenic or teratogenic effects. The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
CALCIUM FORMATE	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
HYDROXYPROPYL METHYLCELLULOSE	Intraperitoneal (rat) 5200 mg/kg
TC MASTERFLEX EXTRA & PORTLAND CEMENT & CALCIUM CARBONATE & CALCIUM FORMATE	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.

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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.

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

	Endpoint	Test Duration (hr)	Species	Value	Source
TC Masterflex Extra	Not Available	Not Available	Not Available	Not Available	Not Available
portland cement	Not Available	Not Available	Not Available	Not Available	Not Available
calcium carbonate	EC50	72h	Algae or other aquatic plants	>14mg/l	2
	NOEC(ECx)	1h	Fish	4-320mg/l	4
	LC50	96h	Fish	>165200mg/L	4
calcium formate	EC50	96h	Algae or other aquatic plants	584000mg/L	2
	EC50	72h	Algae or other aquatic plants	570mg/l	2
	EC50	48h	Crustacea	>1000mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	63mg/l	2
	ErC50	72h	Algae or other aquatic plants	570mg/l	2
	LC50	96h	Fish	>=1000mg/l	1
hydroxypropyl methylcellulose	Not Available	Not Available	Not Available	Not Available	Not Available
Legend:	<i>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</i>				

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.

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:

TC Masterflex Extra**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
	invertebrate	long-term	1.0	fathead minnow

* from Environmental Health Criteria 61: WHO Publication.

Toxicity in Microorganisms:

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

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

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.

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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

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

DO NOT discharge into sewer or waterways.

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
hydroxypropyl methylcellulose	LOW (LogKOW = -5.3)

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. <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. In most instances the supplier of the material should be consulted.</p> <ul style="list-style-type: none"> ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority.
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SECTION 14 Transport information

Labels Required

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Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

14.7. Maritime transport in bulk according to IMO instruments

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

Not Applicable

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

Product name	Group
portland cement	Not Available
calcium carbonate	Not Available
calcium formate	Not Available
hydroxypropyl methylcellulose	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
portland cement	Not Available
calcium carbonate	Not Available
calcium formate	Not Available
hydroxypropyl methylcellulose	Not Available

SECTION 15 Regulatory information

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

portland cement is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

calcium carbonate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

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

calcium formate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

hydroxypropyl methylcellulose is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

Australian Inventory of Industrial Chemicals (AIIC)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (portland cement; calcium formate; hydroxypropyl methylcellulose)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (hydroxypropyl methylcellulose)

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National Inventory	Status
Japan - ENCS	No (portland cement)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (portland cement)
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	<i>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.</i>

SECTION 16 Other information

Revision Date	13/02/2025
Initial Date	13/02/2025

Other information**Ingredients with multiple cas numbers**

Name	CAS No
calcium carbonate	471-34-1, 13397-26-7, 15634-14-7, 1317-65-3, 72608-12-9, 878759-26-3, 63660-97-9, 459411-10-0, 198352-33-9, 146358-95-4, 1357-85-3
hydroxypropyl methylcellulose	9004-65-3, 8063-82-9

Classification of the preparation and its individual components has drawn on official and authoritative sources 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
- ▶ DNEL: Derived No-Effect Level
- ▶ PNEC: Predicted no-effect concentration
- ▶ MARPOL: International Convention for the Prevention of Pollution from Ships
- ▶ IMSBC: International Maritime Solid Bulk Cargoes Code
- ▶ IGC: International Gas Carrier Code
- ▶ IBC: International Bulk Chemical Code

- ▶ AIIC: 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

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- 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