WELDING ACCESSORIES - Model:WZ8

Welding Guns of Australia Pty Ltd

Chemwatch Hazard Alert Code: 3

Chemwatch: **7918-84**
Version No: **2.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Issue Date: **26/11/2024**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	WELDING ACCESSORIES - Model:WZ8
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	TOXIC SOLID, INORGANIC, N.O.S. (contains arsenic and copper)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Use according to manufacturer's directions.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**Welding Guns of Australia Pty Ltd**
Address	112 Christina Road Villawood NSW 2163 Australia
Telephone	+61 2 9780 4200
Fax	+61 2 9780 4244
Website	www.unimig.com.au
Email	orders@unimig.com.au

Emergency telephone number

Association / Organisation	**Welding Guns of Australia Pty Ltd**	**CHEMWATCH EMERGENCY RESPONSE (24/7)**
Emergency telephone number(s)	+61 2 9780 4200	+61 1800 951 288
Other emergency telephone number(s)	Not Available	+61 3 9573 3188

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Acute Toxicity (Inhalation) Category 2, Germ Cell Mutagenicity Category 1A, Carcinogenicity Category 1B, Reproductive Toxicity Category 1B, Reproductive Toxicity Effects on or via Lactation, Specific Target Organ Toxicity - Repeated Exposure Category 2, Hazardous to the Aquatic Environment Acute Hazard Category 3, Hazardous to the Aquatic Environment Long-Term Hazard Category 2
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI***

Label elements

Hazard pictogram(s)	

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Signal word	**Danger**

Hazard statement(s)

H302	Harmful if swallowed.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H318	Causes serious eye damage.
H330	Fatal if inhaled.
H340	May cause genetic defects.
H350	May cause cancer.
H360Df	May damage the unborn child. Suspected of damaging fertility.
H362	May cause harm to breast-fed children.
H373	May cause damage to organs through prolonged or repeated exposure.
H402	Harmful to aquatic life.
H411	Toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume.
P263	Avoid contact during pregnancy and while nursing.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P270	Do not eat, drink or smoke when using this product.
P264	Wash all exposed external body areas thoroughly after handling.
P273	Avoid release to the environment.
P284	[In case of inadequate ventilation] wear respiratory protection.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
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 and soap.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P330	Rinse mouth.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

Continued...

CAS No	%[weight]	Name
7440-33-7	>90	tungsten
7439-89-6	<1.14	iron
7440-02-0	<1.14	nickel
7429-90-5	<1.14	aluminium
7440-70-2	<1.14	calcium
7439-95-4	<1.14	magnesium
7631-86-9	<1.14	silica amorphous
7439-98-7	<1.14	molybdenum
7440-44-0	<1.14	coke powder
7723-14-0.	<1.14	phosphorus, black
7440-50-8	<1.14	copper
7439-96-5	<1.14	manganese
7440-47-3	<1.14	chromium
7440-69-9	<1.14	bismuth
7440-43-9	<1.14	cadmium
7440-09-7	<1.14	potassium
7440-23-5	<1.14	sodium
7440-38-2	<1.14	arsenic
7440-36-0	<1.14	antimony
7439-92-1	<1.14	lead
7440-31-5	<1.14	tin
64417-98-7	0.8	zirconium(IV) oxide - yttria stabilised

Legend:	***1. Classified by Chemwatch; 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: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. ▸ **DO NOT** attempt to remove particles attached to or embedded in eye . ▸ Lay victim down, on stretcher if available and pad **BOTH** eyes, make sure dressing does not press on the injured eye by placing thick pads under dressing, above and below the eye. ▸ Seek urgent medical assistance, or transport to hospital.
Skin Contact	If skin or hair contact occurs: ▸ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▸ Quickly remove all contaminated clothing, including footwear. ▸ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▸ Transport to hospital, or doctor. For thermal burns: ▸ Decontaminate area around burn. ▸ Consider the use of cold packs and topical antibiotics. For first-degree burns (affecting top layer of skin) ▸ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides. ▸ Use compresses if running water is not available. ▸ Cover with sterile non-adhesive bandage or clean cloth. ▸ Do NOT apply butter or ointments; this may cause infection. ▸ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur. For second-degree burns (affecting top two layers of skin) ▸ Cool the burn by immerse in cold running water for 10-15 minutes. ▸ Use compresses if running water is not available. ▸ Do NOT apply ice as this may lower body temperature and cause further damage. ▸ Do NOT break blisters or apply butter or ointments; this may cause infection. ▸ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape. To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort): ▸ Lay the person flat. ▸ Elevate feet about 12 inches. ▸ Elevate burn area above heart level, if possible. ▸ Cover the person with coat or blanket.

Continued...

	▸ Seek medical assistance. For third-degree burns Seek immediate medical or emergency assistance. In the mean time: ▸ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound. ▸ Separate burned toes and fingers with dry, sterile dressings. ▸ Do not soak burn in water or apply ointments or butter; this may cause infection. ▸ To prevent shock see above. ▸ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. ▸ Have a person with a facial burn sit up. ▸ Check pulse and breathing to monitor for shock until emergency help arrives.
Inhalation	▸ 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	▸ Give a slurry of activated charcoal in water to drink. **NEVER** GIVE AN UNCONSCIOUS PATIENT WATER TO DRINK. ▸ At least 3 tablespoons in a glass of water should be given. ▸ Although induction of vomiting may be recommended (**IN CONSCIOUS PERSONS ONLY**), such a first aid measure is dissuaded due to the risk of aspiration of stomach contents. (i) It is better to take the patient to a doctor who can decide on the necessity and method of emptying the stomach. (ii) Special circumstances may however exist; these include non-availability of charcoal and the ready availability of the doctor. **NOTE:** If vomiting is induced, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. **NOTE:** Wear protective gloves when inducing vomiting. ▸ REFER FOR MEDICAL ATTENTION WITHOUT DELAY. ▸ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. ▸ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist. ▸ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS. (ICSC20305/20307)

Indication of any immediate medical attention and special treatment needed

For acute or short term repeated exposures to arsenic, soluble compounds: Treat as per arsenic poisoning.

- Acute skin lesions such as contact dermatitis usually do not require other treatment than removal from exposure.
- If more severe symptoms of the respiratory system, the skin or the gastro-intestinal tract occur, British Anti-Lewisite (BAL, dimercaprol) may be given. Prompt administration in such cases is vital; to obtain maximum benefit such treatment should be administered within 4 hours of poisoning.
- In addition, general treatment such as prevention of further absorption from the gastro-intestinal tract are mandatory.
- General supportive therapy such as maintenance of respiration and circulation, maintenance of water and electrolyte balance and control of nervous system effects, as well as elimination of absorbed poison through dialysis and exchange transfusion, may be used if feasible.
- Dimercaprol is given by deep intramuscular injection as a 5% solution in peanut oil (or a 10% solution with benzyl-benzoate in vegetable oil). It is usually given in a dose of 3 mg/kg, 4-hourly, for the first two days, or twice daily for up to seven days. [ILO Encyclopedia]
- BAL Therapy is effective for haematological manifestations of chronic arsenic poisoning but not for neurological symptoms. Watch for side effects (e.g. urticaria, burning sensation in the lips, mouth and throat, fever, conjunctivitis etc).
- Some relief results from administration of diphenhydramine (Benadryl) (1.5 mg/kg intramuscularly or by mouth every 6 hour). [Ellenhorn and Barceloux: Medical Toxicology]

BIOLOGICAL EXPOSURE INDEX - BEI (Notice of Intent to Establish)

BEIs represent the levels of determinants which are most likely to be observed in specimens collected from a healthy worker who has been exposed to chemicals to the same extent as a worker with inhalation exposure to the

Exposure Standard (ES or TLV):

Determinant	Index	Sampling Time	Comments
Inorganic arsenic metabolites in urine	35 ug/gm creatinine	End of workweek	B

B: Background levels occur in specimens collected from subjects **NOT** exposed Consult specific documentation.

for copper intoxication:

- Unless extensive vomiting has occurred empty the stomach by lavage with water, milk, sodium bicarbonate solution or a 0.1% solution of potassium ferrocyanide (the resulting copper ferrocyanide is insoluble).
- Administer egg white and other demulcents.
- Maintain electrolyte and fluid balances.
- Morphine or meperidine (Demerol) may be necessary for control of pain.
- If symptoms persist or intensify (especially circulatory collapse or cerebral disturbances, try BAL intramuscularly or penicillamine in accordance with the supplier's recommendations.
- Treat shock vigorously with blood transfusions and perhaps vasopressor amines.
- If intravascular haemolysis becomes evident protect the kidneys by maintaining a diuresis with mannitol and perhaps by alkalinising the urine with sodium bicarbonate.

Continued...

- It is unlikely that methylene blue would be effective against the occassional methaemoglobinemia and it might exacerbate the subsequent haemolytic episode.
- Institute measures for impending renal and hepatic failure.

[GOSSELIN, SMITH & HODGE: Commercial Toxicology of Commercial Products]

- A role for activated charcoals for emesis is, as yet, unproven.
- In severe poisoning CaNa2EDTA has been proposed.

[ELLENHORN & BARCELOUX: Medical Toxicology]

* Preplacement and periodic medical examinations are essential for workers exposed to arsenic on a regular basis. Preplacement physical examinations should give particular attention to allergic and chronic skin lesions, eye disease, psoriasis, chronic eczematous dermatitis, hyperpigmentation of the skin, keratosis and warts, baseline weight, baseline blood and haemoglobin counts, baseline urinary arsenic determinations.
Annual physical examinations should give attention to general health, weight, skin condition, and any evidence of excessive exposure or absorption of arsenic

Copper, magnesium, aluminium, antimony, iron, manganese, nickel, zinc (and their compounds) in welding, brazing, galvanising or smelting operations all give rise to thermally produced particulates of smaller dimension than may be produced if the metals are divided mechanically. Where insufficient ventilation or respiratory protection is available these particulates may produce "metal fume fever" in workers from an acute or long term exposure.

- Onset occurs in 4-6 hours generally on the evening following exposure. Tolerance develops in workers but may be lost over the weekend. (Monday Morning Fever)
- Pulmonary function tests may indicate reduced lung volumes, small airway obstruction and decreased carbon monoxide diffusing capacity but these abnormalities resolve after several months.
- Although mildly elevated urinary levels of heavy metal may occur they do not correlate with clinical effects.
- The general approach to treatment is recognition of the disease, supportive care and prevention of exposure.
- Seriously symptomatic patients should receive chest x-rays, have arterial blood gases determined and be observed for the development of tracheobronchitis and pulmonary edema.

[Ellenhorn and Barceloux: Medical Toxicology]
For acute and short term repeated exposures:
Treat as for arsenic poisoning. Prompt administration is essential.

- General treatment for prevention of further absorption is essential. British anti-lewisite (BAL, dimercaprol, 2,3 dimercaptopropanol) may be given as a deep intramuscular injection, as 5% in oil, or as 10% in benzoyl benzoate - vegetable oil. Dose may be 3 mg/kg at 4 hours for first two days, then at 6 hours for third day, then once or twice daily.
- Supportive therapy is maintenance of water and electrolyte balance and control of CNS effects.
- Elimination of absorbed poison by dialysis and exchange transfusion may be required.

Both dimercaptol and calcium disodium edetate are said to be effective in acute experimental tungsten poisonings.

Both dermal and oral toxicity of manganese salts is low because of limited solubility of manganese. No known permanent pulmonary sequelae develop after acute manganese exposure. Treatment is supportive.

[***Ellenhorn and Barceloux: Medical Toxicology***]

In clinical trials with miners exposed to manganese-containing dusts, L-dopa relieved extrapyramidal symptoms of both hypo kinetic and dystonic patients. For short periods of time symptoms could also be controlled with scopolamine and amphetamine. BAL and calcium EDTA prove ineffective.

[***Gosselin et al: Clinical Toxicology of Commercial Products.***]

- Chelation with British Anti-Lewisite (BAL) for serious antimony exposures should be employed.
- Dialyse as needed. The role of exchange diffusion is not clear.
- Be sure to monitor for dysrhythmias.

[Ellenhorn and Barceloux: Medical Toxicology]
Severe bismuth intoxication may be treated with dimercaptol (BAL in oil). Induction of acidosis by administration of ammonium chloride has been claimed to promote mobilisation of bismuth from tissue depots and increase the rate of urinary excretion.
[Martindale:The Extra Pharmacopoeia]
In mouse models D-penicillamine (Cuprimine M.S. & D) is a useful chelating agent. [Ellenhorn & Barceloux: Medical Toxicology]

- Gastric acids solubilise lead and its salts and lead absorption occurs in the small bowel.
- Particles of less than 1 um diameter are substantially absorbed by the alveoli following inhalation.
- Lead is distributed to the red blood cells and has a half-life of 35 days. It is subsequently redistributed to soft tissue & bone-stores or eliminated. The kidney accounts for 75% of daily lead loss; integumentary and alimentary losses account for the remainder.
- Neurasthenic symptoms are the most common symptoms of intoxication. Lead toxicity produces a classic motor neuropathy. Acute encephalopathy appears infrequently in adults. Diazepam is the best drug for seizures.
- Whole-blood lead is the best measure of recent exposure; free erythrocyte protoporphyrin (FEP) provides the best screening for chronic exposure. Obvious clinical symptoms occur in adults when whole-blood lead exceeds 80 ug/dL.
- British Anti-Lewisite is an effective antidote and enhances faecal and urinary excretion of lead. The onset of action of BAL is about 30 minutes and most of the chelated metal complex is excreted in 4-6 hours, primarily in the bile. Adverse reaction appears in up to 50% of patients given BAL in doses exceeding 5 mg/kg. CaNa2EDTA has also been used alone or in concert with BAL as an antidote. D-penicillamine is the usual oral agent for mobilisation of bone lead; its use in the treatment of lead poisoning remains investigational. 2,3-dimercapto-1-propanesulfonic acid (DMPS) and dimercaptosuccinic acid (DMSA) are water soluble analogues of BAL and their effectiveness is undergoing review. As a rule, stop BAL if lead decreases below 50 ug/dL; stop CaNa2EDTA if blood lead decreases below 40 ug/dL or urinary lead drops below 2 mg/24hrs.

[Ellenhorn & Barceloux: Medical Toxicology]

BIOLOGICAL EXPOSURE INDEX - BEI

These represent the determinants observed in specimens collected from a healthy worker who has been exposed at the Exposure Standard (ES or TLV):

Continued...

Determinant	Index	Sampling Time	Comments
1. Lead in blood	30 ug/100 ml	Not Critical	
2. Lead in urine	150 ug/gm creatinine	Not Critical	B
3. Zinc protoporphyrin in blood	250 ug/100 ml erythrocytes OR 100 ug/100 ml blood	After 1 month exposure	B

B: Background levels occur in specimens collected from subjects **NOT** exposed.

SECTION 5 Firefighting measures

Extinguishing media

Metal dust fires need to be smothered with sand, inert dry powders.
DO NOT USE WATER, CO2 or FOAM.

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	▸ Reacts with acids producing flammable / explosive hydrogen (H2) gas None known.

Advice for firefighters

Fire Fighting	▸ When silica dust is dispersed in air, firefighters should wear inhalation protection as hazardous substances from the fire may be adsorbed on the silica particles. ▸ When heated to extreme temperatures, (>1700 deg.C) amorphous silica can fuse. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ 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	▸ **DO NOT** disturb burning dust. Explosion may result if dust is stirred into a cloud, by providing oxygen to a large surface of hot metal. ▸ **DO NOT** use water or foam as generation of explosive hydrogen may result. With the exception of the metals that burn in contact with air or water (for example, sodium), masses of combustible metals do not represent unusual fire risks because they have the ability to conduct heat away from hot spots so efficiently that the heat of combustion cannot be maintained - this means that it will require a lot of heat to ignite a mass of combustible metal. Generally, metal fire risks exist when sawdust, machine shavings and other metal 'fines' are present. Metal powders, while generally regarded as non-combustible: ▸ May burn when metal is finely divided and energy input is high. ▸ May react explosively with water. ▸ May be ignited by friction, heat, sparks or flame. ▸ May **REIGNITE** after fire is extinguished. ▸ Will burn with intense heat. Note: ▸ Metal dust fires are slow moving but intense and difficult to extinguish. ▸ Containers may explode on heating. ▸ Dusts or fumes may form explosive mixtures with air. ▸ Gases generated in fire may be poisonous, corrosive or irritating. ▸ Hot or burning metals may react violently upon contact with other materials, such as oxidising agents and extinguishing agents used on fires involving ordinary combustibles or flammable liquids. ▸ Temperatures produced by burning metals can be higher than temperatures generated by burning flammable liquids ▸ Some metals can continue to burn in carbon dioxide, nitrogen, water, or steam atmospheres in which ordinary combustibles or flammable liquids would be incapable of burning. ▸ When silica dust is dispersed in air, firefighters should wear inhalation protection as hazardous substances from the fire may be adsorbed on the silica particles. ▸ When heated to extreme temperatures, (>1700 deg.C) amorphous silica can fuse. Decomposition may produce toxic fumes of: silicon dioxide (SiO2) metal oxides arsenic compounds tungsten 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.
HAZCHEM	2X

SECTION 6 Accidental release measures

Continued...

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	Environmental hazard - contain spillage. ▸ Clean up waste regularly and abnormal spills immediately. ▸ Avoid breathing dust and contact with skin and eyes. ▸ Wear protective clothing, gloves, safety glasses and dust respirator. ▸ Use dry clean up procedures and avoid generating dust. ▸ Vacuum up or sweep up. **NOTE:** Vacuum cleaner must be fitted with an exhaust micro filter (H-Class HEPA type) (consider explosion-proof machines designed to be grounded during storage and use). H-Class HEPA filtered industrial vacuum cleaners should NOT be used on wet materials or surfaces. ▸ Dampen with water to prevent dusting before sweeping. ▸ Place in suitable containers for disposal.
Major Spills	Environmental hazard - contain spillage. ▸ Clean up all spills immediately. ▸ Avoid 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 or ▸ Vacuum up (consider explosion-proof machines designed to be grounded during storage and use). ▸ Place in clean drum then flush area with water.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	For molten metals: · Molten metal and water can be an explosive combination. The risk is greatest when there is sufficient molten metal to entrap or seal off water. Water and other forms of contamination on or contained in scrap or remelt ingot are known to have caused explosions in melting operations. While the products may have minimal surface roughness and internal voids, there remains the possibility of moisture contamination or entrapment. If confined, even a few drops can lead to violent explosions. · All tooling, containers, molds and ladles, which come in contact with molten metal must be preheated or specially coated, rust free and approved for such use. · Any surfaces that may contact molten metal (e.g. concrete) should be specially coated · Drops of molten metal in water (e.g. from plasma arc cutting), while not normally an explosion hazard, can generate enough flammable hydrogen gas to present an explosion hazard. Vigorous circulation of the water and removal of the particles minimise the hazard. During melting operations, the following minimum guidelines should be observed: · Inspect all materials prior to furnace charging and completely remove surface contamination such as water, ice, snow, deposits of grease and oil or other surface contamination resulting from weather exposure, shipment, or storage. · Store materials in dry, heated areas with any cracks or cavities pointed downwards. · Preheat and dry large objects adequately before charging in to a furnace containing molten metal. This is typically done by the use of a drying oven or homogenising furnace. The dry cycle should bring the metal temperature of the coldest item of the batch to 200 degree C (400 deg F) and then hold at that temperature for 6 hours. ▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ **DO NOT** allow material to contact humans, exposed food or food utensils. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ No smoking, naked lights or ignition sources.

Continued...

	▸ Store in a cool, dry, well-ventilated area. ▸ 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.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ **CARE**: Packing of high density product in light weight metal or plastic packages may result in container collapse with product release ▸ Heavy gauge metal packages / Heavy gauge metal drums ▸ Lined metal can, lined metal pail/ can. ▸ Plastic pail. ▸ Polyliner drum. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks. For low viscosity materials ▸ Drums and jerricans must be of the non-removable head type. ▸ Where a can is to be used as an inner package, the can must have a screwed enclosure. For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.): ▸ Removable head packaging; ▸ Cans with friction closures and ▸ low pressure tubes and cartridges may be used. - Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages *. - In addition, where inner packagings are glass and contain liquids of packing group I and II there must be sufficient inert absorbent to absorb any spillage *. - * unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	▸ Avoid strong acids, bases.

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	nickel	Nickel, metal	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	nickel	Nickel, powder	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium	Aluminium (welding fumes) (as Al)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium	Aluminium, pyro powders (as Al)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium	Aluminium (metal dust)	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Precipitated silica	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Silica gel	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Precipitated silica	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica gel	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Diatomaceous earth (uncalcined)	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Fumed silica (respirable dust)	2 mg/m3	Not Available	Not Available	Not Available

Continued...

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Fume (thermally generated)(respirable dust)	2 mg/m3	Not Available	Not Available	(e) Containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Fumed silica (respirable dust)	2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Diatomaceous earth (uncalcined)	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	phosphorus, black	Phosphorus (yellow)	0.1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	copper	Copper, dusts & mists (as Cu)	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	copper	Copper (fume)	0.2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	manganese	Manganese, fume (as Mn)	1 mg/m3	3 mg/m3	Not Available	Not Available
Australia Exposure Standards	chromium	Chromium (metal)	0.5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	cadmium	Cadmium and compounds (as Cd)	0.01 mg/m3	Not Available	Not Available	(g) Some compounds in these groups are classified as carcinogenic or as sensitisers. Check individual classification details on the safety data sheet for information on classification.
Australia Exposure Standards	arsenic	Arsenic & soluble compounds (as As)	0.05 mg/m3	Not Available	Not Available	(g) Some compounds in these groups are classified as carcinogenic or as sensitisers. Check individual classification details on the safety data sheet for information on classification.
Australia Exposure Standards	antimony	Antimony & compounds (as Sb)	0.5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	lead	Lead, inorganic dusts & fumes (as Pb)	0.05 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	tin	Tin, metal	2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	zirconium(IV) oxide - yttria stabilised	Yttrium, metal & compounds (as Y)	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	zirconium(IV) oxide - yttria stabilised	Zirconium compounds (as Zr)	5 mg/m3	10 mg/m3	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
tungsten	Not Available	Not Available
iron	Not Available	Not Available
nickel	10 mg/m3	Not Available
aluminium	Not Available	Not Available
calcium	Not Available	Not Available
magnesium	Not Available	Not Available
silica amorphous	3,000 mg/m3	Not Available
molybdenum	Not Available	Not Available
coke powder	Not Available	Not Available
phosphorus, black	5 mg/m3	Not Available
copper	100 mg/m3	Not Available
manganese	500 mg/m3	Not Available
chromium	250 mg/m3	Not Available
bismuth	Not Available	Not Available
cadmium	9 mg/m3	Not Available
potassium	Not Available	Not Available
sodium	Not Available	Not Available
arsenic	5 mg/m3	Not Available
antimony	Not Available	Not Available

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Ingredient	Original IDLH	Revised IDLH
lead	Not Available	Not Available
tin	Not Available	Not Available
zirconium(IV) oxide - yttria stabilised	500 mg/m3 / 25 mg/m3	Not Available

MATERIAL DATA

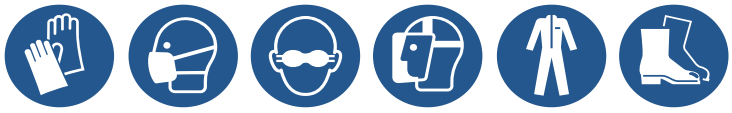
Exposure controls

Appropriate engineering controls

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

- Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area.
- Work should be undertaken in an isolated system such as a "glove-box" . Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system.
- Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within.
- Open-vessel systems are prohibited.
- Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation.
- Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system.
- For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood.
- Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas).
- Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air.
- Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed.

Individual protection measures, such as personal protective equipment



Eye and face protection

- 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].

Skin protection

See Hand protection below

Hands/feet protection

- Wear chemical protective gloves, e.g. PVC.
- Wear safety footwear or safety gumboots, e.g. Rubber

NOTE:

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

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
· frequency and duration of contact,
· chemical resistance of glove material,
· glove thickness and

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	· dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. ▸ Protective gloves eg. Leather gloves or gloves with Leather facing
Body protection	See Other protection below
Other protection	▸ Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent] ▸ Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent] ▸ Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely. ▸ Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. ▸ Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. · During repair or maintenance activities the potential exists for exposures to toxic metal particulate in excess of the occupational standards. Under these circumstances, protecting workers can require the use of specific work practices or procedures involving the combined use of ventilation, wet and vacuum cleaning methods, respiratory protection, decontamination, special protective clothing, and when necessary, restricted work zones. · Protective over-garments or work clothing must be worn by persons who may become contaminated with particulate during activities such as machining, furnace rebuilding, air cleaning equipment filter changes, maintenance, furnace tending, etc. Contaminated work clothing and over-garments must be managed in a controlled manner to prevent secondary exposure to workers of third parties, to prevent the spread of particulate to other areas, and to prevent particulate from being taken home by workers. · Personnel who handle and work with molten metal should utilise primary protective clothing like polycarbonate face shields, fire resistant tapper's jackets, neck shades (snoods), leggings, spats and similar equipment to prevent burn injuries. In addition to primary protection, secondary or day-to-day work clothing that is fire resistant and sheds metal splash is recommended for use with molten metal. Synthetic materials should never be worn even as secondary clothing (undergarments).

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

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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.
Where significant concentrations of the material are likely to enter the breathing zone, a Class P3 respirator may be required.
Class P3 particulate filters are used for protection against highly toxic or highly irritant particulates.
Filtration rate: Filters at least 99.95% of airborne particles
Suitable for:
· Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.
· Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.
· Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS
· Highly toxic particles e.g. Organophosphate Insecticides, Radionuclides, Asbestos
Note: P3 Rating can only be achieved when used with a Full Face Respirator or Powered Air-Purifying Respirator (PAPR). If used with any other respirator, it will only provide filtration protection up to a P2 rating.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Silver coloured solid with no odour; insoluble in water.

Physical state	Solid	**Relative density (Water = 1)**	19.3
Odour	No Odour	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Applicable
pH (as supplied)	Not Applicable	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	3410	**Viscosity (cSt)**	Not Applicable
Initial boiling point and boiling range (°C)	5900	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Applicable	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Applicable	**Surface Tension (dyn/cm or mN/m)**	Not Applicable
Lower Explosive Limit (%)	Not Applicable	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Applicable	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (1%)**	Not Applicable
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	▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7

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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	There is sufficient evidence to classify this material as acutely toxic.
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	There is sufficient evidence to classify this material as carcinogenic
g) Reproductivity	There is sufficient evidence to classify this material as toxic to reproductivity
h) STOT - Single Exposure	Based on available data, the classification criteria are not met.
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	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. Inhalation of dusts, generated by the material during the course of normal handling, may produce severe damage to the health of the individual. Relatively small amounts absorbed from the lungs may prove fatal. Persons with impaired respiratory function, airway diseases and conditions such as emphysema or chronic bronchitis, may incur further disability if excessive concentrations of particulate are inhaled. If prior damage to the circulatory or nervous systems has occurred or if kidney damage has been sustained, proper screenings should be conducted on individuals who may be exposed to further risk if handling and use of the material result in excessive exposures.
Ingestion	Toxic effects may result from the accidental ingestion of the material; animal experiments indicate that ingestion of less than 40 gram may be fatal or may produce serious damage to the health of the individual.
Skin Contact	The material produces mild skin irritation; evidence exists, or practical experience predicts, that the material either ▸ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or ▸ produces significant, but mild, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus. Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. 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. 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 than can cuase 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

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respiratory sensitisers
Wherever it is reasonably practicable, exposure to substances that can cuase 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.
On the basis of epidemiological data, the material is regarded as carcinogenic to humans. There is sufficient data to establish a causal association between human exposure to the material and the development of cancer.
There is sufficient evidence to provide a strong presumption that human exposure to the material may produce heritable genetic damage.
There is sufficient evidence to provide a strong presumption that human exposure to the material may result in the development of heritable genetic damage, generally on the basis of
- appropriate animal studies,
- other relevant information
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.
There is sufficient evidence to establish a causal relationship between human exposure to the material and impaired fertility
There is sufficient evidence to establish a causal relationship between human exposure to the material and subsequent developmental toxic effects in the off-spring.

There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: - clear evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects.
There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of:
- clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.

Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.
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 alveolii (sub 5 um) are able to produce pathogenic effects in the lungs.
The synthetic, amorphous silicas are believed to represent a very greatly reduced silicosis hazard compared to crystalline silicas and are considered to be nuisance dusts.
When heated to high temperature and a long time, amorphous silica can produce crystalline silica on cooling. Inhalation of dusts containing crystalline silicas may lead to silicosis, a disabling pulmonary fibrosis that may take years to develop. Discrepancies between various studies showing that fibrosis associated with chronic exposure to amorphous silica and those that do not may be explained by assuming that diatomaceous earth (a non-synthetic silica commonly used in industry) is either weakly fibrogenic or nonfibrogenic and that fibrosis is due to contamination by crystalline silica content
Symptoms of chronic arsenic poisoning, by inhalation, include weight loss, nausea and diarrhoea alternating with constipation, pigmentation and eruption of the skin, loss of hair, peripheral neuritis, blood disorders (anaemia), striations on fingernails and toenails. Long-term exposure can cause an ulcer or hole in the "bone"'dividing the inner nose. Hoarseness and sore eyes also occur. anaemia, liver and kidney injury, skin and lung cancer. Renal damage may develop after acute or chronic exposures. Prolonged inhalation of arsenical dusts are implicated in lung cancer.

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Subacute and chronic exposure to arsenic and its organic salts may produce anorexia, mild gastrointestinal disturbances (nausea and vomiting), low grade fever, persistent headache, pallor, weakness, catarrhal inflammation and neuropathies with severe crippling effects.. Stomatitis (oral lesions) and salivation are common. High or repeated exposure can cause nerve damage with "pins and needles"' burning, numbness, and later weakness (incoordination) of arms and legs, anaemia, liver and kidney injury, skin and lung cancer. Repeated exposure can also damage the liver, causing narrowing of the blood vessels, or interfere with bone marrow's ability to make red blood cells.
Skin afflictions are many and varied; erythema, eczema, pigmentation (arsenic melanosis), diffuse alopecia, keratosis (especially of the palms and soles), scaling and desquamation, brittle nails, white lines or bands on the nails (Mees lines), loss of hair and nails and localised subcutaneous oedema (especially of the eye-lids). Signs of renal damage may also develop. Liver enlargement (hepatomegaly) with jaundice (and sometimes pruritus) may develop into cirrhosis with accumulation of body fluids in the abdominal cavity (ascites). Nervous system effects involving the extremities (numbness, tingling, burning pain, weakness, incoordination) may also occur.
Arsenic exposure is linked with increase in diseases including ischaemia, cerebrovascular disease and carotid atherosclerosis. Several cytokines and growth factors involved in inflammation, have been identified in humans after prolonged exposure; these may heighten the risk of atherosclerosis (1). Individual variability in arsenic metabolism may determine susceptibility to arsenic disease. Genetic polymorphism (variability) for enzymes instrumental in arsenic metabolism (purine nucleoside phosphorylase and glutathione S-transferase omega 1-1) has been identified amongst individuals of European and Mexican Hispanic (indigenous) ancestry (2).
Many cases of skin cancer have been reported among people exposed to arsenic through medical treatment with inorganic trivalent arsenic compounds. In some instances skin cancers have occurred in combination with other cancers, such as liver angiosarcoma, intestinal and urinary bladder carcinomas and meningioma. Epidemiological studies of cancer after medical treatment have shown an excess of skin cancers but no clear association with other cancers has been shown.
Prolonged inhalation of arsenical dusts are implicated in lung cancer. Occupational exposure to inorganic arsenic, especially in mining and copper smelting, has consistently been associated with an increased risk of cancer. An almost tenfold increase in the incidence of lung cancer was found in workers most heavily exposed to arsenic and relatively clear dose-response relationships have been obtained with regard to cumulative
exposure. Other smelter worker population's have been shown to have consistent increases in lung cancer incidence, as well as increases of about 20% in the incidence of gastrointestinal cancer and of 30% for renal cancer and haematolymphatic malignancies
An association between environmental exposure to arsenic through drinking water and skin cancer has been observed and confirmed. Epidemiological studies in areas where drinking water contained 0.35-1.14 mg/l arsenic elevated risks for cancers of the bladder, kidney, skin, liver, lung and colon in both men and women. Taiwanese exposed to naturally occurring arsenic in well-water have experienced skin cancer. Inorganic and organic arsenics are established carcinogens in man. Chronic human exposure to non-overtly toxic doses of arsenic is associated with carcinogenicity, although the mechanism is not fully understood. Arsenic does not directly cause DNA damage or mutations. Data indicates that nontoxic doses of arsenite can interact with glucocorticoid receptor (GR) complexes and selectively inhibit GR-mediated transcription which is associated with altered nuclear function (3).
(1) Wu et al; Environmental Health Perspectives, 111, 1429-1438, 2003
(2) Yu et al; Environmental Health Perspectives, 111, 1421-1427, 2003
(3) Kaltreider et al; Environmental Health Perspectives, 109, pp 245-251, 2001

Excessive exposure to lead can affect the blood, the nervous system, heart, endocrine organs and the immune system and the digestive system. The synthesis of haemoglobin is inhibited and can result in anaemia. If left untreated, neuromuscular dysfunction, possible paralysis and encephalopathy (brain tissue damage) may result. Other symptoms of overexposure include joint and muscle pain, weakness of the extensor muscles (frequently the hand and wrist), headache, dizziness, abdominal pain, diarrhoea, constipation, nausea, vomiting, blue line on the gums, insomnia and metallic taste. High body levels produce cerebrospinal pressure, brain damage with stupor leading to coma and, in some cases, death. Early symptoms of lead poisoning ("plumbism") include anorexia and loss of weight, constipation, apathy or irritability, occasional vomiting, fatigue, headache, weakness, and a metallic taste in the mouth. Advanced poisonings are characterised by intermittent vomiting, irritability, nervousness, myalgia of the arms and legs (often with wrist and foot drop). Severe poisonings may produce persistent vomiting, ataxia, stupor or lethargy, visual disturbances progressing to optic neuritis and atrophy, hyper- tension, papilloedema, cranial nerve paralysis, delirium, convulsions and coma. Neurological effects include mental retardation, seizures, cerebral palsy and marked muscular contractions that distort the spine, limbs, hips and sometimes the cranial inervated muscles (dystonia musculorum deformans). Industrial exposure has been associated with irreversible kidney damage.
Lead is a cumulative poison with adverse effects in pregnancy [NIOSHTIC]
Lead salts have been reported to cross the placenta and induce embryo- and foeto-mortality. They also may have a teratogenic effect (causing birth deformities) in certain animal species. Organometallic lead may not produce these effects. Adverse effects of lead on human reproduction, embryonic and foetal development and postnatal mental development have also been recorded. Foetal exposure to lead may result in birth defects, mental retardation, behavioural disorders and death during the first year of childhood. Paternal effects may include reduced sex drive, impotence, sterility and adverse effects on the sperm which in turn may increase the potential for increased birth defects. Maternal effects may include miscarriage and stillbirth in exposed women, or women whose husbands might be exposed, sterility or decreased fertility, and abnormal menses. Exposure by both parents to lead may exacerbate the reproductive effects.
Repeated exposure to synthetic amorphous silicas may produce skin dryness and cracking.
Available data confirm the absence of significant toxicity by oral and dermal routes of exposure.
Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted in a number of species, at airborne concentrations ranging from 0.5 mg/m3 to 150 mg/m3. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m3. When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m3. Differences in values may be due to particle size, and therefore the number of particles administered per unit dose. Generally, as particle size diminishes so does the NOAEL/ LOAEL. Exposure produced transient increases in lung inflammation, markers of cell injury and lung collagen content. There was no evidence of interstitial pulmonary fibrosis.
For copper and its compounds (typically copper chloride):
Acute toxicity: There are no reliable acute oral toxicity results available. Animal testing shows that skin in exposure to copper may lead to hardness of the skin, scar formation, exudation and reddish changes. Inflammation, irritation and injury of the skin were noted.
Repeat dose toxicity: Animal testing shows that very high levels of copper monochloride may cause anaemia.

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Genetic toxicity: Copper monochloride does not appear to cause mutations in vivo, although chromosomal aberrations were seen at very high concentrations in vitro.
Cancer-causing potential: There was insufficient information to evaluate the cancer-causing activity of copper monochloride.
Repeated or prolonged exposure may also damage the liver and may cause a decrease in the heart rate. Systemic poisoning may result from inhalation or chronic ingestion of manganese containing substances. Progressive and permanent disability can occur from chronic manganese poisoning if it is not treated, but it is not fatal.
Chronic exposure has been associated with two major effects; bronchitis/pneumonitis following inhalation of manganese dusts and "manganism", a neuropsychiatric disorder that may also arise from inhalation exposures. Chronic exposure to low levels may result in the accumulation of toxic concentrations in critical organs. The brain in particular appears to sustain cellular damage to the ganglion. Symptoms appear before any pathology is evident and may include a mask-like facial expression, spastic gait, tremors, slurred speech, sometimes dystonia (disordered muscle tone), fatigue, anorexia, asthenia (loss of strength and energy), apathy and the inability to concentrate. Insomnia may be an early finding. Chronic poisoning may occur over a 6-24 month period depending on exposure levels.
The onset of chronic manganese poisoning is insidious, with apathy, anorexia weakness, headache and spasms. Manganese psychosis follows with certain definitive features: unaccountable laughter, euphoria, impulsive acts, absentmindedness, mental confusion, aggressiveness and hallucinations. The final stage is characterised by speech difficulties, muscular twitching, spastic gait and other nervous system effects. Symptoms resemble those of Parkinson's disease. Rat studies indicate the gradual accumulation of brain manganese to produce lesions mimicking those found in Parkinsonism. If the disease is diagnosed whilst still in the early stages and the patient is removed from exposure, the course may be reversed.
Inhalation of manganese fumes may cause 'metal fume fever' characterised by flu-like symptoms: fever, chill, nausea, weakness and body aches. Manganese dust is no longer believed to be a causative factor in pneumonia. If there is any relationship at all, it appears to be as an aggravating factor to a preexisting condition.
Prolonged or repeated eye contact may result in conjunctivitis.
Manganese is an essential trace element in all living organisms with the level of tissue manganese remaining remarkably constant throughout life.
Persons, exposed for long periods to molybdenum oxides, suffer from anaemia. Animals exposed to certain insoluble molybdenum compounds show anorexia, diarrhoea, weight loss, listlessness, and liver and kidney damage. Molybdenum disturbs bone metabolism, giving rise to lameness, bone joint abnormalities, osteoporosis and high serum phosphatase levels.Cattle, rabbits, and chicks on high dietary levels of molybdenum exhibited deformities of joints of the extremities. Low molybdenum intake has been attributed to the high incidence of oesophageal cancer in South Africa among the Bantu of Transkei, in China and in Russia.
Chronic exposure of workers in Russian molybdenum-copper plant resulted in a fall in the albumin/globulin ratio owing to a rise in globulins (particularly alpha-globulins) which is interpreted as evidence of liver dysfunction with hyperbilirubinaemia. Hepatotoxic effects are also found in animals given molybdenum salts with a rise in alpha-globulin levels, hypoalbuminaemia and increased serum bilirubin reported. Other reported biochemical effects include an early depletive effect on tissue nicotinamide nucleotides, hyperaminoaciduria, reduction in red blood cell life-span and hyper-thyroidism. Industrial exposure to some insoluble molybdenum compounds is thought to have resulted in an increased incidence of weakness, fatigue, anorexia, headache and joint and muscular pain. Under the conditions of a 2-year inhalation study* there was equivocal evidence of carcinogenic activity of molybdenum trioxide in male rats, male mice and female mice based on a marginally significant positive trend of alveolar/bronchiolar adenoma or carcinoma. There was no evidence of carcinogenic activity in female rats exposed to 10, 30 and 100 mg/m3. Exposure of male and female rats to molybdenum trioxide by inhalation resulted in increased incidences of chronic alveolar inflammation, hyaline degeneration of the olfactory epithelium (females), hyaline degeneration of the respiratory epithelium and squamous metaplasia of the epiglottis. Exposure of female and male mice to molybdenum trioxide resulted in an increased incidence of metaplasia of the alveolar epithelium, histiocyte cellular inflammation (males), hyaline degeneration of the respiratory epithelium, hyaline degeneration of the olfactory epithelium (females), squamous metaplasia of the epiglottis, and hyperplasia of the larynx.
Guinea pigs exposed to molybdenum trioxide dust for 1 hour daily at 250 mg/m3 showed extreme irritation. Symptoms include loss of appetite, weight loss, diarrhoea, muscular incoordination and loss of hair. Of the 51 animals exposed, 26 died after the tenth exposure. Exposure to freshly generated MoO_2 fume under about the same exposure conditions proved unexpectedly less toxic, with only 8.3% mortality compared with 51% mortality with the dust, and no mortality when the exposure level was reduced to about one-third (57 mg Mo/m3). Explanation for this unexpected finding was felt to reside in the more rapid solution and elimination of the large surface area fume particle. [Patty's]
Exposure of male and female rats to molybdenum trioxide resulted in the development of respiratory system lesions. In the lung, the incidence and severity of chronic alveolar inflammation increased with increasing exposure concentration in male and female rats. In some male rats, exposure to the material resulted in alveolar/ bronchiolar adenomas or carcinomas. Lesions in the nose (hyaline degeneration) and larynx (squamous metaplasia) were considered to be a non-specific defensive or adaptive response to chronic inhalation exposure. Inhalation exposure of mice to molybdenum trioxide was associated with the development of lung neoplasms and an increased incidence of alveolar/ bronchiolar carcinoma or adenoma in both sexes. Chronic inflammatory lesions were not present in the lungs. Lesions of the nose and larynx were similar to those observed in rats.
Molybdenum trioxide was not mutagenic in any of five strains of Salmonella typhimurium and did not induce sister chromatid exchanges or chromosomal aberrations in cultured Chinese hamster ovary cells in vitro. All tests were conducted with or without S9 metabolic activation enzymes.
Pneumoconiosis has been described in experimental animals exposed sub-chronically to molybdenum trioxide.
The mechanism of molybdenum trioxide action in lung carcinogenicity is not known; the material is not mutagenic. Non-neoplastic lesions of the nose and larynx of rats and in the nose, larynx and lungs of mice were apparently due to the development of a more durable epithelium in response to chronic exposure.
The US Department of Health and Human Services (1) concluded that there was equivocal evidence of carcinogenic activity in male F344/N rats based on a marginally significant positive trend of alveolar/ bronchiolar adenoma or carcinoma; that there was no evidence of carcinogenic activity in female F344/N rats; that there was some evidence of carcinogenic activity in male B6C3F1 mice and that there was evidence of carcinogenic activity in female B6C3F1 mice
National Toxicology Program: Technical Report Series 462, April 1997
Yttrium is a member of the so-called heavy-group (the yttriums) of the rare earths (or lanthanoids). No occupational diseases or cases of poisoning in workers producing rare earth elements have been described.
Lanthanoids entering the human body due to exposure to various industrial processes can affect metabolic processes. Trivalent lanthanoid ions, especially lanthanum 3+ and gadolinium 3+, can interfere with calcium channels in human and animal cells. Lanthanoids can also alter or even inhibit the action of various enzymes. Lanthanoid ions found in neurons can regulate synaptic

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transmission, as well as block some receptors (for example, glutamate receptors). Lanthanoids target the liver causing fatty liver degeneration and a decrease in liver glycogen and blood glucose levels.
Lanthanoids because of their high density can produce significant abnormalities in a chest X-ray. Lanthanoids are generally not fibrogenic and lesions typically have little or no clinical importance. Occasional cases of suspected pneumoconiosis have however been reported. The toxicity of all elements in the yttrium group has been investigated in workers and animals alike. Effects on peripheral blood including a decrease haemoglobin and erythrocyte content and changes in the leucocyte formula have been recorded. Studies show that yttrium is deposited in the skeleton within only a few hours of intake. Animal lungs show productive inflammation and a tendency to develop nodular or diffuse sclerosis following administration by intratracheal injection. The main risks to workers involved in production of rare earths are due to dust inhalation.

Based on the available toxicity data, the rare earth chlorides appear to have moderate acute and chronic toxicity. However these substances cause severe eye irritation and severe irritation in abraded skin. They are poorly absorbed by the gastrointestinal tract and by unbroken skin but slight liver damage has been demonstrated in subchronic oral toxicity studies at high doses. The literature indicates that chronic inhalation exposure to the rare earth chlorides may cause pneumoconiosis in humans. There are no indications of carcinogenicity in the rare earth chlorides. Mutagenicity studies on these substances have mixed results, but are predominantly negative.

* IUPAC currently recommends the name lanthanoid rather than lanthanide, as the suffix "-ide" generally indicates negative ions whereas the suffix "-oid" indicates similarity to one of the members of the containing family of elements. In the older literature, the name "lanthanon" was often used.
Repeated or prolonged exposure to antimony and its compounds may produce stomatitis, dry throat, metallic taste, gingivitis, septal and laryngeal perforation, laryngitis, headache, dyspnea, indigestion, nausea, vomiting, diarrhoea, anorexia, anaemia, weight loss, pain and chest tightness, sleeplessness, muscular pain and weakness, dizziness, pharyngitis,tracheitis, bronchitis, pneumonitis, benign pneumoconiosis (with obstructive lung disease and emphysema) and haematological disorders.
Degenerative changes of the liver and kidney may occur. Symptoms can be variable, and may including fatigue, myopathy (muscle aches and inflammation), hypotension, angina and immune dysregulation and hypertrophy of splenic follicles.
Antimony's deposition in body tissues and its detrimental effects depend upon the oxidation state of the element. The trivalent antimony compounds are cardiotoxic. Trivalent antimony affects liver functions, impairs enzymes, and may interfere with sulfur chemistry. If antimony impairs phosphofructokinase (PFK), then purine metabolism may be disrupted, resulting in elevated blood and/or urine levels of hypoxanthine, uric acid and possibly ammonia. Pentavalent antimony deposits in bone, kidney, and in organs of the endocrine system.
Chronic exposure to antimony compounds may result in itchiness, papules and pustules around sweat and sebaceous glands ("antimony spots"), but rarely around the face, and dermatitis.
Collapse and sudden death due to anaphylactic-type reactions have occurred. Therapeutic doses given intravenously cause nausea, vomiting, cough and abdominal pain and diarrhoea. Other side-effects include anorexia, chest, muscle and joint pains, pruritus, skin rashes, dizziness and oedema. Renal and hepatic damage occur rarely and haemolytic anaemia has been reported. Continuous treatment with small doses of antimony may give rise to subacute poisoning similar to chronic arsenic poisoning.
Smelter workers often show skin rashes on the forearms and thighs resembling chicken pox pustules.
Workers exposed to inorganic antimony compounds show a benign pneumoconiosis and obstructive lung disease - these are probably non-specific. Woman appear to more susceptible to systemic effects following exposure. Antimony crosses the placenta, is present in amniotic fluids, and is excreted in breast milk. There are suggestions that exposure may produce an increased incidence of spontaneous late abortions, premature births, and gynecological problems among female antimony smelter workers. An excess of deaths from lung cancer has been reported in smelter workers with more than 7 years exposure to relatively high levels of dust and fume. Animal studies demonstrate that the dust may produce pathological changes in cardiac muscle and may produce an interstitial pneumonitis and endogenous pneumonia. One animal study has also suggested that inhalation of the dust by rats induced a significantly increased incidence of carcinogenic tumours of the lungs and thorax.
Increased chromosome defects occur when human lymphocytes are incubated with a soluble antimony salt.
The inhalation data suggests that the myocardium is a target of antimony toxicity. It is possible that antimony affects circulating glucose by interfering with enzymes of the glycogenolysis and gluconeogenesis pathways. The mechanism of action of antimony remains unclear. However, some studies suggest that antimony combines with sulfhydryl groups including those in several enzymes important for tissue respiration
Symptoms of chronic bismuth toxicity in humans consist of decreased appetite, weakness, rheumatic pain, diarrhoea, fever, foul breath, gingivitis and dermatitis. A blue line of the gums, the "bismuth line", may persist for years after exposure has ceased. Jaundice and conjunctival haemorrhage are rare, but have been reported. Bismuth neuropathy (kidney damage), with proteinuria may occur. The kidney is the site of highest concentration with liver concentrations being significantly lower. Renal failure may be reversible if treated early but anuria and death have occurred.
Metallic dusts generated by the industrial process give rise to a number of potential health problems. The larger particles, above 5 micron, are nose and throat irritants. Smaller particles however, may cause lung deterioration. Particles of less than 1.5 micron can be trapped in the lungs and, dependent on the nature of the particle, may give rise to further serious health consequences.
Metals are widely distributed in the environment and are not biodegradable. Biologically, many metals are essential to living systems and are involved in a variety of cellular, physiological, and structural functions. They often are cofactors of enzymes, and play a role in transcriptional control, muscle contraction, nerve transmission, blood clotting, and oxygen transport and delivery.
Although all metals are potentially toxic at some level, some are highly toxic at relatively low levels. Moreover, in some cases the same metal can be essential at low levels and toxic at higher levels, or it may be toxic via one route of entry but not another.
Toxic effects of some metals are associated with disruption of functions of essential metals. Metals may have a range of effects, including cancer, neurotoxicity, immunotoxicity, cardiotoxicity, reproductive toxicity, teratogenicity, and genotoxicity. Biological half lives of metals vary greatly, from hours to years. Furthermore, the half life of a given metal varies in different tissues. Lead has a half life of 14 days in soft tissues and 20 years in bone.
In considering how to evaluate the toxicity of metals of potential concern, a number of aspects of metal toxicity should be kept in mind:
Different species vary in their responses to different metals; in some cases, humans are more sensitive than rodents. Thus, there is a need for broad-based testing of metals;

- The route of exposure may affect the dose and site where the metal concentrates, and thus the observed toxic effects;
- Metal-metal interactions can reduce or enhance toxicity; biotransformation can reduce or enhance toxicity;
- It is difficult to predict the toxicity of one metal based on the adverse effects of another; in trying to evaluate the toxicity of one particular metal compound, predictions based on similar compounds of the same metal may be valid.

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WELDING ACCESSORIES - Model:WZ8	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
tungsten	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (Rodent - rabbit): 500mg/24H - Mild
	Oral (Rat) LD50: >2000 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
		Eye: no adverse effect observed (not irritating)[1]
		Skin (Rodent - rabbit): 500mg/24H - Mild
		Skin: no adverse effect observed (not irritating)[1]
iron	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 98600 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
nickel	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 5000 mg/kg[2]	Not Available
aluminium	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: >2.3 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
calcium	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2500 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
magnesium	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: >2.1 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	
silica amorphous	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (Rodent - rabbit): 25mg/24H - Mild
	Inhalation (Rat) LC50: >0.09<0.84 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >1000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
molybdenum	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: >1.93 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	
coke powder	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >2000 mg/kg[1]	Not Available
phosphorus, black	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: ~100 mg/kg[2]	Not Available
	Inhalation (Rat) LC50: >5.75 mg/l4h[1]	
	Oral (Rat) LD50: >2000 mg/kg[1]	
copper	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: 0.733 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]

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	Oral (Mouse) LD50; 0.7 mg/kg[2]	
manganese	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: >5.14 mg/l4h[1]	Eye (Rodent - rabbit): 500mg/24H - Mild
	Oral (Rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
		Skin (Rodent - rabbit): 500mg/24H - Mild
		Skin: no adverse effect observed (not irritating)[1]
chromium	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: >5.41 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >5000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
bismuth	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 5000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
cadmium	**TOXICITY**	**IRRITATION**
	Inhalation(Rabbit) LC50; 0.028 mg/L4h[1]	Eye: adverse effect observed (irritating)[1]
	Oral (Rat) LD50: 225 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
potassium	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
sodium	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
arsenic	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2400 mg/kg[1]	Eye: adverse effect observed (irreversible damage)[1]
	Oral (Mouse) LD50; 144 mg/kg[1]	Skin: adverse effect observed (irritating)[1]
antimony	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >8000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: >5.2 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: 7000 mg/kg[2]	
lead	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: >5.05 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	
tin	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: >4.75 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	
zirconium(IV) oxide - yttria stabilised	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: >4.3 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

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***

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TUNGSTEN	Substance has been investigated as a reproductive effector in female rodents- Oral TDLo 1.16 mg/kg Tungsten toxicology has been investigated principally by administration of sodium tungstate. Subcutaneous administration produces severe rectal temperature drops in rats. Parenteral doses are almost completely eliminated in the urine within 12 hours. Parenteral, but not oral, routes show an enlargement of adrenals and kidneys. Tungsten powder but not tungsten carbide is toxic by intraperitoneal administration - fatalities occur within 24 hours with survivors showing minor gross changes in liver and spleen.
NICKEL	Oral (rat) TDLo: 500 mg/kg/5D-I Inhalation (rat) TCLo: 0.1 mg/m3/24H/17W-C **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [***National Toxicology Program: U.S. Dep. of Health & Human Services 2002***]
CALCIUM	The solid may react violently on contact with wet skin tissue, i.e. eyes, mouth, causing chemical and thermal burns. The acute effects include burns, ulceration, or tissue death, severe eye damage (corneal burns or opacification), and probable blindness. Inhalation of dust or fumes (especially from a fire involving calcium) will cause shortness of breath, nausea, headache, nose and respiratory tract irritation and in extreme, pneumonitis
SILICA AMORPHOUS	Reports indicate high/prolonged exposures to amorphous silicas induced lung fibrosis in experimental animals; in some experiments these effects were reversible. [PATTYS] For silica amorphous: Derived No Adverse Effects Level (NOAEL) in the range of 1000 mg/kg/d. In humans, synthetic amorphous silica (SAS) is essentially non-toxic by mouth, skin or eyes, and by inhalation. Epidemiology studies show little evidence of adverse health effects due to SAS. Repeated exposure (without personal protection) may cause mechanical irritation of the eye and drying/cracking of the skin. When experimental animals inhale synthetic amorphous silica (SAS) dust, it dissolves in the lung fluid and is rapidly eliminated. If swallowed, the vast majority of SAS is excreted in the faeces and there is little accumulation in the body. Following absorption across the gut, SAS is eliminated via urine without modification in animals and humans. SAS is not expected to be broken down (metabolised) in mammals. After ingestion, there is limited accumulation of SAS in body tissues and rapid elimination occurs. Intestinal absorption has not been calculated, but appears to be insignificant in animals and humans. SASs injected subcutaneously are subjected to rapid dissolution and removal. There is no indication of metabolism of SAS in animals or humans based on chemical structure and available data. In contrast to crystalline silica, SAS is soluble in physiological media and the soluble chemical species that are formed are eliminated via the urinary tract without modification. Both the mammalian and environmental toxicology of SASs are significantly influenced by the physical and chemical properties, particularly those of solubility and particle size. SAS has no acute intrinsic toxicity by inhalation. Adverse effects, including suffocation, that have been reported were caused by the presence of high numbers of respirable particles generated to meet the required test atmosphere. These results are not representative of exposure to commercial SASs and should not be used for human risk assessment. Though repeated exposure of the skin may cause dryness and cracking, SAS is not a skin or eye irritant, and it is not a sensitiser. Repeated-dose and chronic toxicity studies confirm the absence of toxicity when SAS is swallowed or upon skin contact. Long-term inhalation of SAS caused some adverse effects in animals (increases in lung inflammation, cell injury and lung collagen content), all of which subsided after exposure. Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted with SAS in a number of species, at airborne concentrations ranging from 0.5 mg/m3 to 150 mg/m3. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m3. When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m3. The difference in values may be explained by different particle size, and therefore the number of particles administered per unit dose. In general, as particle size decreases so does the NOAEL/LOAEL. Neither inhalation nor oral administration caused neoplasms (tumours). SAS is not mutagenic in vitro. No genotoxicity was detected in in vivo assays. SAS does not impair development of the foetus. Fertility was not specifically studied, but the reproductive organs in long-term studies were not affected. For Synthetic Amorphous Silica (SAS) Repeated dose toxicity Oral (rat), 2 weeks to 6 months, no significant treatment-related adverse effects at doses of up to 8% silica in the diet. Inhalation (rat), 13 weeks, Lowest Observed Effect Level (LOEL) =1.3 mg/m3 based on mild reversible effects in the lungs. Inhalation (rat), 90 days, LOEL = 1 mg/m3 based on reversible effects in the lungs and effects in the nasal cavity. For silane treated synthetic amorphous silica: Repeated dose toxicity: oral (rat), 28-d, diet, no significant treatment-related adverse effects at the doses tested. There is no evidence of cancer or other long-term respiratory health effects (for example, silicosis) in workers employed in the manufacture of SAS. Respiratory symptoms in SAS workers have been shown to correlate with smoking but not with SAS exposure, while serial pulmonary function values and chest radiographs are not adversely affected by long-term exposure to SAS.
COKE POWDER	Substance has been investigated as a reproductive effector. TDLo: 167mg/kg
COPPER	WARNING: Inhalation of high concentrations of copper fume may cause "metal fume fever", an acute industrial disease of short duration. Symptoms are tiredness, influenza like respiratory tract irritation with fever. for copper and its compounds (typically copper chloride): **Acute toxicity:** There are no reliable acute oral toxicity results available. In an acute dermal toxicity study (OECD TG 402), one group of 5 male rats and 5 groups of 5 female rats received doses of 1000, 1500 and 2000 mg/kg bw via dermal application for 24 hours. The LD50 values of copper monochloride were 2,000 mg/kg bw or greater for male (no deaths observed) and 1,224 mg/kg bw for female. Four females died at both 1500 and 2000 mg/kg bw, and one at 1,000 mg/kg bw. Symptom of the hardness of skin, an exudation of hardness site, the formation of scar and reddish changes were observed on application sites in all treated animals. Skin inflammation and injury were also noted. In addition, a reddish or black urine was observed in females at 2,000, 1,500 and 1,000 mg/kg bw. Female rats appeared to be more sensitive than male based on mortality and clinical signs. No reliable skin/eye irritation studies were available. The acute dermal study with copper monochloride suggests that it has a potential to cause skin irritation. **Repeat dose toxicity:** In repeated dose toxicity study performed according to OECD TG 422, copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39 - 51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL value was 5 and 1.3 mg/kg bw/day for male and female rats, respectively. No deaths were observed in male rats. One treatment-related death was observed in female rats in the high dose group. Erythropoietic toxicity

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	(anaemia) was seen in both sexes at the 80 mg/kg bw/day. The frequency of squamous cell hyperplasia of the forestomach was increased in a dose-dependent manner in male and female rats at all treatment groups, and was statistically significant in males at doses of =20 mg/kg bw/day and in females at doses of =5 mg/kg bw/day doses. The observed effects are considered to be local, non-systemic effect on the forestomach which result from oral (gavage) administration of copper monochloride. **Genotoxicity:** An in vitro genotoxicity study with copper monochloride showed negative results in a bacterial reverse mutation test with Salmonella typhimurium strains (TA 98, TA 100, TA 1535, and TA 1537) with and without S9 mix at concentrations of up to 1,000 ug/plate. An in vitro test for chromosome aberration in Chinese hamster lung (CHL) cells showed that copper monochloride induced structural and numerical aberrations at the concentration of 50, 70 and 100 ug/mL without S9 mix. In the presence of the metabolic activation system, significant increases of structural aberrations were observed at 50 and 70 ug/mL and significant increases of numerical aberrations were observed at 70 ug/mL. In an in vivo mammalian erythrocyte micronucleus assay, all animals dosed (15 - 60 mg/kg bw) with copper monochloride exhibited similar PCE/(PCE+NCE) ratios and MNPCE frequencies compared to those of the negative control animals. Therefore copper monochloride is not an in vivo mutagen. **Carcinogenicity:** there was insufficient information to evaluate the carcinogenic activity of copper monochloride. Reproductive and developmental toxicity: In the combined repeated dose toxicity study with the reproduction/developmental toxicity screening test (OECD TG 422), copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39-51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL of copper monochloride for fertility toxicity was 80 mg/kg bw/day for the parental animals. No treatment-related effects were observed on the reproductive organs and the fertility parameters assessed. For developmental toxicity the NOAEL was 20 mg/kg bw/day. Three of 120 pups appeared to have icterus at birth; 4 of 120 pups appeared runted at the highest dose tested (80 mg/kg bw/day).
CHROMIUM	Gastrointestinal tumours, lymphoma, musculoskeletal tumours and tumours at site of application recorded. For chrome(III) and other valence states (except hexavalent): For inhalation exposure, all trivalent and other chromium compounds are treated as particulates, not gases. The mechanisms of chromium toxicity are very complex, and although many studies on chromium are available, there is a great deal of uncertainty about how chromium exerts its toxic influence. Much more is known about the mechanisms of hexavalent chromium toxicity than trivalent chromium toxicity. There is an abundance of information available on the carcinogenic potential of chromium compounds and on the genotoxicity and mutagenicity of chromium compounds in experimental systems. The consensus from various reviews and agencies is that evidence of carcinogenicity of elemental, divalent, or trivalent chromium compounds is lacking. Epidemiological studies of workers in a number of industries (chromate production, chromate pigment production and use, and chrome plating) conclude that while occupational exposure to hexavalent chromium compounds is associated with an increased risk of respiratory system cancers (primarily bronchogenic and nasal), results from occupational exposure studies to mixtures that were mainly elemental and trivalent (ferrochromium alloy worker) were inconclusive. Studies in leather tanners, who were exposed to trivalent chromium were consistently negative. In addition to the lack of direct evidence of carcinogenicity of trivalent or elemental chromium and its compounds, the genotoxic evidence is overwhelmingly negative. The lesser potency of trivalent chromium relative to hexavalent chromium is likely related to the higher redox potential of hexavalent chromium and its greater ability to enter cells. enter cells The general inability of trivalent chromium to traverse membranes and thus be absorbed or reach peripheral tissue in significant amounts is generally accepted as a probable explanation for the overall absence of systemic trivalent chromium toxicity. Elemental and divalent forms of chromium are not able to traverse membranes readily either. This is not to say that elemental, divalent, or trivalent chromium compounds cannot traverse membranes and reach peripheral tissue, the mechanism of absorption is simply less efficient in comparison to absorption of hexavalent chromium compounds. Hexavalent chromium compounds exist as tetrahedral chromate anions, resembling the forms of other natural anions like sulfate and phosphate which are permeable across nonselective membranes. Trivalent chromium forms octahedral complexes which cannot easily enter though these channels, instead being absorbed via passive diffusion and phagocytosis. Although trivalent chromium is less well absorbed than hexavalent chromium, workers exposed to trivalent compounds have had detectable levels of chromium in the urine at the end of a workday. Absorbed chromium is widely distributed throughout the body via the bloodstream, and can reach the foetus. Although there is ample in vivo evidence that hexavalent chromium is efficiently reduced to trivalent chromium in the gastrointestinal tract and can be reduced to the trivalent form by ascorbate and glutathione in the lungs, there is no evidence that trivalent chromium is converted to hexavalent chromium in biological systems. In general, trivalent chromium compounds are cleared rapidly from the blood and more slowly from the tissues. Although not fully characterized, the biologically active trivalent chromium molecule appears to be chromodulin, also referred to as (GTF). Chromodulin is an oligopeptide complex containing four chromic ions. Chromodulin may facilitate interactions of insulin with its receptor site, influencing protein, glucose, and lipid metabolism. Inorganic trivalent chromium compounds, which do not appear to have insulin-potentiating properties, are capable of being converted into biologically active forms by humans and animals Chromium can be a potent sensitiser in a small minority of humans, both from dermal and inhalation exposures. The most sensitive endpoint identified in animal studies of acute exposure to trivalent chromium appears to involve the respiratory system. Specifically, acute exposure to trivalent chromium is associated with impaired lung function and lung damage. Based on what is known about absorption of chromium in the human body, its potential mechanism of action in cells, and occupational data indicating that valence states other than hexavalent exhibit a relative lack of toxicity the toxicity of elemental and divalent chromium compounds is expected to be similar to or less than common trivalent forms. Tenth Annual Report on Carcinogens: Substance known to be Carcinogenic [***National Toxicology Program: U.S. Dep. of Health and Human Services 2002***]
ARSENIC	Tumorigenic - Carcinogenic by RTECS criteria. Arsenic compounds are classified by the European Union as toxic by inhalation and ingestion and toxic to aquatic life and long lasting in the environment. IARC classify arsenic in drinking water as a confirmed human carcinogen (IARC 1). The main inorganic forms of arsenic relevant for human exposures are pentavalent arsenic (also called arsenate, As(V), or As+5) and trivalent arsenic (also called arsenite, As(III), or As+3). These inorganic species undergoes a series of reduction and oxidative/methylation steps in human liver and other tissues to form tri- and pentavalent methylated metabolites of methylarsonite [MA(III)], methylarsonate [MA(V)], dimethylarsinite [DMA(III)], and dimethylarsinate [DMA(V)]. Some mammalian species also produce trimethylated metabolites, trimethylarsine oxide The distinction between inorganic and organic forms is important because it is generally accepted that the organic species are excreted more quickly from the body and generally considered less toxic, with a relative rank order of As(III) > As(V) >> MA(V),

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	DMA(V) >> arsenobetaine. However, the methylated trivalent metabolites, MA(III) and DMA(III), are significantly more toxic than their pentavalent counterpart and either As(III) or As(V) . In many cases, biomonitoring or environmental occurrence data are reported as total arsenic and do not distinguish between the different species. In those situations, understanding the relevant sources of arsenic is essential to evaluate potential arsenic related health effects, especially those related to inorganic arsenic exposure. **WARNING:** This substance has been classified by the IARC as Group 1: **CARCINOGENIC TO HUMANS**.
LEAD	WARNING: Lead is a cumulative poison and has the potential to cause abortion and intellectual impairment to unborn children of pregnant workers.
ZIRCONIUM(IV) OXIDE - YTTRIA STABILISED	Symptoms of acute lanthanide toxicity in rats are immediate defecation, writhing, ataxia (the inability to coordinate voluntary muscular movement), sedation, laboured respiration and reduced activity. Death is due mainly to respiratory and cardiac failure. The rare earths exhibit low toxicity following ingestion but may be toxic by the intraperitoneal route and mildly toxic when administered by the subcutaneous route.The production of skin and lung granulomas, following exposure, may also occur. for typical lanthanides: The symptoms of toxicity of the rare earth elements include writhing, ataxia, labored respiration, walking on the toes with arched back and sedation. The rare earth elements exhibit low toxicity by ingestion exposure. However, the intraperitoneal route may be highly toxic while the subcutaneous route is poison to moderately toxic. The production of skin and lung granulomas after exposure to them requires extensive protection to prevent such exposure. **Chronic Inhalation Toxicity:** An accumulation of insoluble lanthanide particles has been observed in the respiratory tract of humans following chronic occupational exposure and in rodents following chronic exposure to a similar lanthanide cerium oxide. Lymphoid hyperplasia in the bronchial lymph nodes was the critical inhalation health effect identified by the USEPA in a 2008 toxicological review of cerium oxide. **Developmental/Reproductive Toxicity:** Lanthanum carbonate, did not affect fertility or produce any harm to the fetus in a rat study. **Mutagenicity:** Cerium oxide, was negative in the Ames bacterial mutagenic test using bacterial strains TA135, TA1537, TA98, TA100, TA102, and WP2uvrA., and in the mouse in vivo micronucleus assay. **Carcinogenicity:** Lanthanum carbonate, was not carcinogenic in a two-year oral rat study. Not assessed by IARC, NTP, or USEPA.
TUNGSTEN & MANGANESE	The material may be irritating to the eye, with prolonged contact causing 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 epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
NICKEL & COPPER	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.
ALUMINIUM & CALCIUM & MOLYBDENUM & PHOSPHORUS, BLACK & CHROMIUM & POTASSIUM & SODIUM & TIN & ZIRCONIUM(IV) OXIDE - YTTRIA STABILISED	No significant acute toxicological data identified in literature search.
CALCIUM & POTASSIUM & SODIUM & ZIRCONIUM(IV) OXIDE - YTTRIA STABILISED	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.
SILICA AMORPHOUS & CHROMIUM	The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.

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***

Continued...

SECTION 12 Ecological information

Toxicity

WELDING ACCESSORIES - Model:WZ8

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

tungsten

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	7.35mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	0.812mg/l	2
EC50	48h	Crustacea	>163mg/l	2
LC50	96h	Fish	>181mg/l	2

iron

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	18mg/l	2
NOEC(ECx)	48h	Algae or other aquatic plants	0.1-4mg/l	4
EC50	48h	Crustacea	>100mg/l	2
LC50	96h	Fish	0.005-0.008mg/L	4

nickel

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.18mg/l	1
EC50	96h	Algae or other aquatic plants	0.174-0.311mg/L	4
EC50(ECx)	72h	Algae or other aquatic plants	0.18mg/l	1
EC50	48h	Crustacea	>100mg/l	1
LC50	96h	Fish	0.06mg/L	4

aluminium

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.017mg/L	2
EC50	96h	Algae or other aquatic plants	0.005mg/L	2
NOEC(ECx)	72h	Algae or other aquatic plants	>100mg/l	1
EC50	48h	Crustacea	0.736mg/L	2
LC50	96h	Fish	0.078-0.108mg/l	2

calcium

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	336h	Crustacea	32mg/l	2
EC50	48h	Crustacea	49.1mg/l	2

magnesium

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>12mg/l	2
EC50	96h	Algae or other aquatic plants	222.37mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	>=12mg/l	2
EC50	48h	Crustacea	344mg/l	2
LC50	96h	Fish	541mg/l	2

silica amorphous

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	14.1mg/l	2
EC50	96h	Algae or other aquatic plants	217.576mg/l	2
EC0(ECx)	24h	Crustacea	>=10000mg/l	1
EC50	48h	Crustacea	>86mg/l	2
LC50	96h	Fish	1033.016mg/l	2

molybdenum

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	26mg/l	2
NOEC(ECx)	48h	Algae or other aquatic plants	0.5-80mg/l	4
EC50	48h	Crustacea	130.9mg/l	2

Continued...

	LC50	96h	Fish	211mg/l	2

coke powder

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	48h	Crustacea	>10mg/l	2
EC50	48h	Crustacea	>10mg/l	2

phosphorus, black

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	~1.3mg/l	2
NOEC(ECx)	5784h	Fish	0.0004mg/l	1
EC50	48h	Crustacea	0.025-0.037mg/L	4
LC50	96h	Fish	0.001-0.004mg/L	4

copper

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.011-0.017mg/L	4
EC50	96h	Algae or other aquatic plants	0.03-0.058mg/l	4
NOEC(ECx)	48h	Fish	<0.001mg/L	4
EC50	48h	Crustacea	<0.001mg/L	4
LC50	96h	Fish	0.003mg/L	2

manganese

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	2.8mg/l	2
NOEC(ECx)	504h	Algae or other aquatic plants	0.05-3.7mg/l	4
EC50	48h	Crustacea	>1.6mg/l	2
LC50	96h	Fish	>3.6mg/l	2

chromium

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.026-0.208mg/L	4
EC50	96h	Algae or other aquatic plants	36mg/L	4
EC50(ECx)	48h	Crustacea	<0.001mg/l	2
EC50	48h	Crustacea	<0.001mg/l	2
LC50	96h	Fish	0.106mg/L	4

bismuth

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>1.26mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	1mg/l	2
EC50	48h	Crustacea	>1.26mg/l	2
ErC50	72h	Algae or other aquatic plants	>1.26mg/l	2
LC50	96h	Fish	>100mg/l	2

cadmium

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.018mg/L	2
EC50	96h	Algae or other aquatic plants	0.049-0.162mg/l	4
NOEC(ECx)	1104h	Fish	<0.001mg/L	2
EC50	48h	Crustacea	0.005-0.037mg/L	4
LC50	96h	Fish	4.2-6.9mg/l	Not Available

potassium

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

sodium

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	48h	Fish	11.955mg/L	4

Continued...

	EC50	48h	Crustacea	1640mg/L	4

arsenic					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	0.254mg/L	2
	EC50	96h	Algae or other aquatic plants	0.11-0.209mg/l	4
	EC10(ECx)	168h	Algae or other aquatic plants	0.005mg/L	2
	EC50	48h	Crustacea	0.016mg/L	2
	LC50	96h	Fish	2.8-4.2mg/l	Not Available

antimony					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	>2.4mg/l	2
	EC50	96h	Algae or other aquatic plants	0.61mg/l	2
	NOEC(ECx)	720h	Fish	>0.008mg/L	2
	EC50	48h	Crustacea	423.45mg/l	2
	LC50	96h	Fish	0.93mg/l	2

lead					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	0.021mg/L	2
	EC50	96h	Algae or other aquatic plants	0.282-0.864mg/l	4
	NOEC(ECx)	672h	Crustacea	<0.001mg/L	2
	EC50	48h	Crustacea	0.029mg/L	2
	LC50	96h	Fish	0.008mg/L	2

tin					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	>0.019mg/L	2
	NOEC(ECx)	168h	Crustacea	<0.005mg/L	2
	LC50	96h	Fish	>0.012mg/L	2

zirconium(IV) oxide - yttria stabilised					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	>0.042mg/L	2
	NOEC(ECx)	72h	Algae or other aquatic plants	0.004mg/L	2
	LC50	96h	Fish	>100mg/l	2

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

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
DO NOT discharge into sewer or waterways.

Persistence and degradability

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

Bioaccumulative potential

Ingredient	Bioaccumulation
iron	LOW (LogKOW = -0.77)
nickel	LOW (LogKOW = -0.57)
aluminium	LOW (LogKOW = 0.33)
calcium	LOW (LogKOW = -0.57)
magnesium	LOW (LogKOW = -0.57)
silica amorphous	LOW (LogKOW = 0.5294)
coke powder	LOW (LogKOW = 1.09)
phosphorus, black	HIGH (BCF = 2310000)
bismuth	LOW (LogKOW = 0.73)
cadmium	LOW (LogKOW = -0.07)
potassium	LOW (LogKOW = -0.77)

Continued...

Ingredient	Bioaccumulation
sodium	LOW (LogKOW = -0.77)
arsenic	LOW (LogKOW = 0.68)
lead	LOW (LogKOW = 0.73)
tin	LOW (LogKOW = 1.29)

Mobility in soil

Ingredient	Mobility
silica amorphous	LOW (Log KOC = 23.74)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Management Authority for disposal. ▸ Bury residue in an authorised landfill. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	2X

Land transport (ADG)

14.1. **UN number or ID number**	3288
14.2. **UN proper shipping name**	TOXIC SOLID, INORGANIC, N.O.S. (contains arsenic and copper)
14.3. **Transport hazard class(es)**	Class: 6.1 Subsidiary Hazard: Not Applicable
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: 223 274 Limited quantity: 5 kg

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	3288
14.2. **UN proper shipping name**	Toxic solid, inorganic, n.o.s. * (contains arsenic and copper)

Continued...

14.3. **Transport hazard class(es)**	ICAO/IATA Class: 6.1 ICAO / IATA Subsidiary Hazard: Not Applicable ERG Code: 6L
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: A3 A5 Cargo Only Packing Instructions: 677 Cargo Only Maximum Qty / Pack: 200 kg Passenger and Cargo Packing Instructions: 670 Passenger and Cargo Maximum Qty / Pack: 100 kg Passenger and Cargo Limited Quantity Packing Instructions: Y645 Passenger and Cargo Limited Maximum Qty / Pack: 10 kg

Sea transport (IMDG-Code / GGVSee)

14.1. **UN number**	3288
14.2. **UN proper shipping name**	TOXIC SOLID, INORGANIC, N.O.S. (contains arsenic and copper)
14.3. **Transport hazard class(es)**	IMDG Class: 6.1 IMDG Subsidiary Hazard: Not Applicable
14.4. **Packing group**	III
14.5 **Environmental hazard**	Marine Pollutant
14.6. **Special precautions for user**	EMS Number: F-A , S-A Special provisions: 223 274 Limited Quantities: 5 kg

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
tungsten	Not Available
iron	Not Available
nickel	Not Available
aluminium	Not Available
calcium	Not Available
magnesium	Not Available
silica amorphous	Not Available
molybdenum	Not Available
coke powder	Not Available
phosphorus, black	Not Available
copper	Not Available
manganese	Not Available
chromium	Not Available
bismuth	Not Available
cadmium	Not Available
potassium	Not Available
sodium	Not Available
arsenic	Not Available
antimony	Not Available
lead	Not Available

Continued...

Product name	Group
tin	Not Available
zirconium(IV) oxide - yttria stabilised	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
tungsten	Not Available
iron	Not Available
nickel	Not Available
aluminium	Not Available
calcium	Not Available
magnesium	Not Available
silica amorphous	Not Available
molybdenum	Not Available
coke powder	Not Available
phosphorus, black	Not Available
copper	Not Available
manganese	Not Available
chromium	Not Available
bismuth	Not Available
cadmium	Not Available
potassium	Not Available
sodium	Not Available
arsenic	Not Available
antimony	Not Available
lead	Not Available
tin	Not Available
zirconium(IV) oxide - yttria stabilised	Not Available

SECTION 15 Regulatory information

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

tungsten 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)

iron is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

nickel is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

aluminium is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

Continued...

calcium is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

magnesium is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

silica amorphous is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

molybdenum 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)

coke powder 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)

phosphorus, black is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

copper is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

manganese is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

chromium is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

bismuth 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)

cadmium is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

potassium is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

Continued...

sodium is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

arsenic is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
FEI Equine Prohibited Substances List - Banned Substances
FEI Equine Prohibited Substances List (EPSL)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

antimony 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)
Chemical Footprint Project - Chemicals of High Concern List
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

lead is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

tin 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)

zirconium(IV) oxide - yttria stabilised is found on the following regulatory lists

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

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (zirconium(IV) oxide - yttria stabilised)
Canada - DSL	No (zirconium(IV) oxide - yttria stabilised)
Canada - NDSL	No (tungsten; iron; nickel; aluminium; calcium; magnesium; molybdenum; coke powder; phosphorus, black; copper; manganese; chromium; bismuth; cadmium; potassium; sodium; arsenic; antimony; lead; tin)
China - IECSC	No (zirconium(IV) oxide - yttria stabilised)
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (tungsten; iron; nickel; aluminium; calcium; magnesium; molybdenum; coke powder; phosphorus, black; copper; manganese; chromium; bismuth; cadmium; potassium; sodium; arsenic; antimony; lead; tin; zirconium(IV) oxide - yttria stabilised)
Korea - KECI	Yes
New Zealand - NZIoC	No (zirconium(IV) oxide - yttria stabilised)
Philippines - PICCS	No (zirconium(IV) oxide - yttria stabilised)
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory ‘Active'
Taiwan - TCSI	No (zirconium(IV) oxide - yttria stabilised)
Mexico - INSQ	No (zirconium(IV) oxide - yttria stabilised)
Vietnam - NCI	No (zirconium(IV) oxide - yttria stabilised)
Russia - FBEPH	No (zirconium(IV) oxide - yttria stabilised)

Continued...

National Inventory	Status
Legend:	***Yes = All CAS declared ingredients are on the inventory*** ***No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.***

SECTION 16 Other information

Revision Date	26/11/2024
Initial Date	26/11/2024

Other information

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

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

Definitions and abbreviations

- PC—TWA: Permissible Concentration-Time Weighted Average
- PC—STEL: Permissible Concentration-Short Term Exposure Limit
- IARC: International Agency for Research on Cancer
- ACGIH: American Conference of Governmental Industrial Hygienists
- STEL: Short Term Exposure Limit
- TEEL: Temporary Emergency Exposure Limit。
- IDLH: Immediately Dangerous to Life or Health Concentrations
- ES: Exposure Standard
- OSF: Odour Safety Factor
- NOAEL: No Observed Adverse Effect Level
- LOAEL: Lowest Observed Adverse Effect Level
- TLV: Threshold Limit Value
- LOD: Limit Of Detection
- OTV: Odour Threshold Value
- BCF: BioConcentration Factors
- BEI: Biological Exposure Index
- 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
- 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

end of SDS