KangarooArc - ER7OS-6 Welding Wire

Australian International Trading Group Pty Ltd

Part Number: **Kangaroo Arc ER7OS-6, KAMIG-6.9, KAMIG-61.2, KAMIG-61.6**
Version No: **2.5**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **09/12/2023**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	KangarooArc - ER7OS-6 Welding Wire
Synonyms	ER7OS-6, Solid Welding Wire, Copper Coated Solid Welding Wire, MIG Welding Wire
Other means of identification	Kangaroo Arc ER7OS-6, KAMIG-6.9, KAMIG-61.2, KAMIG-61.6

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

Relevant identified uses	For Arc welding. Do not use except for welding.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**Australian International Trading Group Pty Ltd**
Address	U23, 22-30 Wallace Ave, Suite #109 Point Cook Victoria 3030 Australia
Telephone	1300030099
Fax	Not Available
Website	ausintgroup.com.au
Email	info@ausintgroup.com.au

Emergency telephone number

Association / Organisation	**Not Available**
Emergency telephone numbers	**1300030099**
Other emergency telephone numbers	**Not Available**

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Poisons Schedule	Not Applicable
Classification [1]	Serious Eye Damage/Eye Irritation Category 2B, Acute Toxicity (Inhalation) Category 4, Carcinogenicity Category 2
Legend:	*1. Classification by vendor; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

Hazard pictogram(s)	
Signal word	**Warning**

Hazard statement(s)

H320	Causes eye irritation.
H332	Harmful if inhaled.
H351	Suspected of causing cancer.

Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves and protective clothing.
P261	Avoid breathing dust/fumes.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

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

CAS No	%[weight]	Name
Not Available	>90	Iron
Not Available	0.06-0.15	Carbon
Not Available	0.8-1.15	Silicon
Not Available	1.4-1.85	Manganese
Not Available	>0.025	Sulfur
Not Available	>0.025	Phosphorus
Not Available	>0.5	Copper
Not Available	>0.05	Nickel
Not Available	>0.05	Chromium
Not Available		Welding Fumes (upon use)
Not Available		Manganese fumes (upon use)
Not Available		Copper Fumes (upon use)
Not Available		Iron Dioxide Fumes (upon use)
Not Available		Silica Welding Fumes (Upon use)

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

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ 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. ▸ Particulate bodies from welding spatter may be removed carefully. ▸ **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. ▸ For "arc eye", i.e. welding flash or UV light burns to the eye: ▸ Place eye pads or light clean dressings over both eyes. ▸ Seek medical assistance. For THERMAL burns: ▸ Do NOT remove contact lens ▸ 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 contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation. 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. ▸ 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, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary.
Ingestion	▸ **IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY.** ▸ For advice, contact a Poisons Information Centre or a doctor. ▸ Urgent hospital treatment is likely to be needed.

- 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.

Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:

- **INDUCE** vomiting with fingers down the back of the throat, **ONLY IF CONSCIOUS**. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.

NOTE: Wear a protective glove when inducing vomiting by mechanical means.

Indication of any immediate medical attention and special treatment needed

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.
- 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]

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]

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.*]

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

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

SECTION 5 Firefighting measures

Extinguishing media

- **DO NOT** use halogenated fire extinguishing agents.

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.

Special hazards arising from the substrate or mixture

Fire Incompatibility	‣ Reacts with acids producing flammable / explosive hydrogen (H2) gas

Advice for firefighters

Fire Fighting	‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear breathing apparatus plus protective gloves in the event of a fire. ‣ Prevent, by any means available, spillage from entering drains or water courses. ‣ Use fire fighting procedures suitable for surrounding area. ‣ **DO NOT** approach containers suspected to be hot. ‣ Cool fire exposed containers with water spray from a protected location. ‣ If safe to do so, remove containers from path of fire. ‣ Equipment should be thoroughly decontaminated after use. ‣ When silica dust is dispersed in air, firefighters should wear inhalation protection as hazardous substances from the fire may be adsorbed on the silica particles. ‣ When heated to extreme temperatures, (>1700 deg.C) amorphous silica can fuse.
Fire/Explosion Hazard	‣ **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 When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles. May emit poisonous fumes. May emit corrosive fumes. Welding arc and metal sparks can ignite combustibles.
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. ‣ Remove all ignition sources. ‣ Clean up all spills immediately. ‣ Avoid contact with skin and eyes. ‣ Control personal contact with the substance, by using protective equipment. ‣ Use dry clean up procedures and avoid generating dust. ‣ Place in a suitable, labelled container for waste disposal.
Major Spills	Environmental hazard - contain spillage. · Do not use compressed air to remove metal dusts from floors, beams or equipment · Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation. · Use non-sparking handling equipment, tools and natural bristle brushes. · Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations · Cover and reseal partially empty containers. · Do not allow chips, fines or dusts to contact water, particularly in enclosed areas. If molten: ‣ Contain the flow using dry sand or salt flux as a dam. ‣ All tooling (e.g., shovels or hand tools) and containers which come in contact with molten metal must be preheated or specially coated, rust free and approved for such use. ‣ Allow the spill to cool before remelting scrap. Moderate hazard. ‣ **CAUTION**: Advise personnel in area. ‣ Alert Emergency Services and tell them location and nature of hazard. ‣ Control personal contact by wearing protective clothing. ‣ Prevent, by any means available, spillage from entering drains or water courses. ‣ Recover product wherever possible. ‣ **IF DRY:** Use dry clean up procedures and avoid generating dust. Collect residues and place in sealed plastic bags or other containers for disposal. **IF WET:** Vacuum/shovel up and place in labelled containers for disposal. ‣ **ALWAYS:** Wash area down with large amounts of water and prevent runoff into drains. ‣ If contamination of drains or waterways occurs, advise Emergency Services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	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. ‣ Store in a cool, dry area protected from environmental extremes. ‣ Store away from incompatible materials and foodstuff containers. ‣ Protect containers against physical damage and check regularly for leaks. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS. For major quantities: ‣ Consider storage in bunded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams}. ‣ Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities.

Conditions for safe storage, including any incompatibilities

Suitable container	‣ Bulk bags: Reinforced bags required for dense materials. ‣ **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
Storage incompatibility	The material is described as an electropositive metal. The activity or electromotive series of metals is a listing of the metals in decreasing order of their reactivity with hydrogen-ion sources such as water and acids. In the reaction with a hydrogen-ion source, the metal is oxidised to a metal ion, and the hydrogen ion is reduced to H2. The ordering of the activity series can be related to the standard reduction potential of a metal cation. The more positive the standard reduction potential of the cation, the more difficult it is to oxidise the metal to a hydrated metal cation and the later that metal falls in the series Three notable groups comprise the series ‣ very electropositive metals ‣ electropositive metals ‣ electronegative metals Electropositive metals.have electronegativities that fall between 1.4 and 1.9 Cations of these metals generally have standard reduction potentials between 0.0 and -1.6 V They: ‣ do not react very readily with water to release hydrogen ‣ react with H+ (acids) Electropositive metals do not burn in air as readily as do very electropositive metals. The surfaces of these metals will tarnish in the presence of oxygen forming a protective oxide coating. This coating protects the bulk of the metal against further oxidation (the metal is passivated). Reaction is reduced in the massive form (sheet, rod, or drop), compared with finely divided forms. The less active metals will not burn in air but: ‣ can react exothermically with oxidising acids to form noxious gases. ‣ catalyse polymerisation and other reactions, particularly when finely divided ‣ react with halogenated hydrocarbons (for example, copper dissolves when heated in carbon tetrachloride), sometimes forming explosive compounds. ‣ Elemental metals may react with azo/diazo compounds to form explosive products ‣ Finely divided metal powders develop pyrophoricity when a critical specific surface area is exceeded; this is ascribed to high heat of oxide formation on exposure to air. ‣ Safe handling is possible in relatively low concentrations of oxygen in an inert gas ‣ Several pyrophoric metals, stored in glass bottles have ignited when the container is broken on impact. Storage of these materials moist and in metal containers is recommended. ‣ The reaction residues from various metal syntheses (involving vacuum evaporation and co-deposition with a ligand) are often pyrophoric If the surface of the metal is in contact with both oxygen and water, corrosion can occur. In corrosion, the metal acts as an anode and is oxidised. Many metals may incandesce, react violently, ignite or react explosively upon addition of concentrated nitric acid. Some electropositive metals do not react with nitric acid because they are passivated. http://www.wou.edu/las/physci/ch412/activity.htm Inorganic derivative of Group 11 metal.

For aluminas (aluminium oxide):
Incompatible with hot chlorinated rubber.
In the presence of chlorine trifluoride may react violently and ignite.
-May initiate explosive polymerisation of olefin oxides including ethylene oxide.
-Produces exothermic reaction above 200°C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals.
-Produces exothermic reaction with oxygen difluoride.
-May form explosive mixture with oxygen difluoride.
-Forms explosive mixtures with sodium nitrate.
-Reacts vigorously with vinyl acetate.
Aluminium oxide is an amphoteric substance, meaning it can react with both acids and bases, such as hydrofluoric acid and sodium hydroxide, acting as an acid with a base and a base with an acid, neutralising the other and producing a salt.
The substance may be or contains a "metalloid"
The following elements are considered to be metalloids; boron,silicon, germanium, arsenic, antimony, tellurium and (possibly) polonium
The electronegativities and ionisation energies of the metalloids are between those of the metals and nonmetals, so the metalloids exhibit characteristics of both classes. The reactivity of the metalloids depends on the element with which they are reacting. For example, boron acts as a nonmetal when reacting with sodium yet as a metal when reacting with fluorine.
Unlike most metals, most metalloids are amphoteric- that is they can act as both an acid and a base. For instance, arsenic forms not only salts such as arsenic halides, by the reaction with certain strong acid, but it also forms arsenites by reactions with strong bases.
Most metalloids have a multiplicity of oxidation states or valences. For instance, tellurium has the oxidation states +2, -2, +4, and +6. Metalloids react like non-metals when they react with metals and act like metals when they react with non-metals.

- WARNING: Avoid or control reaction with peroxides. All *transition metal* peroxides should be considered as potentially explosive. For example transition metal complexes of alkyl hydroperoxides may decompose explosively.
- The pi-complexes formed between chromium(0), vanadium(0) and other transition metals (haloarene-metal complexes) and mono-or poly-fluorobenzene show extreme sensitivity to heat and are explosive.
- Avoid reaction with borohydrides or cyanoborohydrides
- Metals and their oxides or salts may react violently with chlorine trifluoride and bromine trifluoride.
- These trifluorides are hypergolic oxidisers. They ignite on contact (without external source of heat or ignition) with recognised fuels - contact with these materials, following an ambient or slightly elevated temperature, is often violent and may produce ignition.
- The state of subdivision may affect the results.

Welding electrodes should not be allowed to come into contact with strong acids or other substances which are corrosive to metals.

- Many metals may incandesce, react violently, ignite or react explosively upon addition of concentrated nitric acid.
- Reacts slowly with water.
- **CAUTION** contamination with moisture will liberate explosive hydrogen gas, causing pressure build up in sealed containers.
- Reacts violently with caustic soda, other alkalies - generating heat, highly flammable hydrogen gas.
- If alkali is dry, heat generated may ignite hydrogen - if alkali is in solution may cause violent foaming

Silicas:

- react with hydrofluoric acid to produce silicon tetrafluoride gas
- react with xenon hexafluoride to produce explosive xenon trioxide
- reacts exothermically with oxygen difluoride, and explosively with chlorine trifluoride (these halogenated materials are not commonplace industrial materials) and other fluorine-containing compounds
- may react with fluorine, chlorates
- are incompatible with strong oxidisers, manganese trioxide, chlorine trioxide, strong alkalis, metal oxides, concentrated orthophosphoric acid, vinyl acetate
- may react vigorously when heated with alkali carbonates.

+	x	+	o	+	+	+

X — *Must not be stored together*
0 — *May be stored together with specific preventions*
+ — *May be stored together*

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

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
KangarooArc - ER7OS-6 Welding Wire	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
KangarooArc - ER7OS-6 Welding Wire	Not Available	Not Available

MATERIAL DATA

Ceiling values were recommended for manganese and compounds in earlier publications. As manganese is a chronic toxin a TWA is considered more appropriate. Because workers exposed to fume exhibited manganism at air-borne concentrations below those that affect workers exposed to dust a lower value has been proposed to provide an extra margin of safety. This value is still above that experienced by two workers exposed to manganese fume in the course of one study.
for welding fume:
In addition to complying with any individual exposure standards for specific contaminants, where current manual welding processes are used, the fume concentration inside the welder's helmet **should not** exceed 5 mg/m3, when collected in accordance with the appropriate standard (AS 3640, for example).
ES* TWA: 5 mg/m3
TLV* TWA: 5 mg/m3, B2 (a substance of variable composition)
OES* TWA: 5 mg/m3
Most welding, even with primitive ventilation, does not produce exposures inside the welding helmet above 5 mg/m3. That which does should be controlled (ACGIH). Inspirable dust concentrations in a worker s breathing zone shall be collected and measured in accordance with AS 3640, for example. Metal content can be analytically determined by OSHA Method ID25 (ICP-AES) after total digestion of filters and dissolution of captured metals. Sampling of the Respirable Dust fraction requires cyclone separator devices (elutriators) and procedures to comply with AS 2985 (for example).
During use the gases nitric oxide, nitrogen peroxide and ozone may be produced by the consumption of the electrode or the action of the welding arc on the atmosphere.
NOTE: Detector tubes for carbon monoxide, measuring in excess of 2 ppm, are commercially available for detection of carbon monoxide.
200 ppm carbon monoxide in air will produce headache, mental dullness and dizziness in a few hours; 600 ppm will produce identical symptoms in less than half and hour and may produce unconsciousness in 1.5 hours; 4000 ppm is fatal in less than an hour.
The TLV-TWA and STEL is recommended to keep blood carboxyhaemoglobin (CoHb) levels below 3.5% in workers so as to prevent adverse neurobehavioural changes and to maintain cardiovascular exercise. Earlier recommendations did not take into account heavy labour, high temperature, high elevations (over 5000 feet above sea level), adverse effects on pregnant workers (i.e. the foetus) and the effects on those with chronic heart and respiratory disease. Workers who smoke frequently have CoHb saturations above 3.5%.
Coburn et al have calculated the time needed to reach 3.5% CoHb at various carbon monoxide exposures.

Carbon Monoxide Concentration (ppm)	Work Load (time in minutes)		
	Sedentary	Light	Moderate
50	191	102	87
75	171	62	53
100	86	46	39
150	58	31	27
200	46	24	21
300	34	18	15
500	24	13	11
1000	18	10	8

Work Load is defined in terms of alveolar ventilation as:

Work Load	Ventilation (l/min)
Sedentary	6
Light	15
Moderate	20

Coburn, R.F.: Foster, R.E.: Kane, P.B.: Considerations of the Physiological Variables that Determine the Carboxyhaemoglobin Concentration in Man. J. Clin Invest. 44(1):1899-1910 (1965)
Odour Safety Factor(OSF)
OSF=0.00025 (CARBON MONOXIDE)
for ozone:
NOTE: Detector tubes for ozone, measuring in excess of 0.05 ppm, are commercially available.
Exposure at 0.2 ppm appears to produce mild acute but not cumulative effects. It is thought that exposures of the order of 0.1 ppm will be tolerated by most workers including asthmatics. Chronic exposure at 0.1 ppm or more can induce significant adverse effects in the lower respiratory tract of both normal and atopic individuals.
Human exposure for 2 hours at an average concentration of 1.5 ppm ozone resulted in a 20% reduction in timed vital capacity of the lung and other effects.
Concentrations of ozone in excess of a few tenths ppm cause occasional discomfort to exposed individuals manifest as headache, dryness of the throat and mucous membranes of the eyes and nose following exposures of short duration.
Exposure to ozone during moderate to heavy work loads results in significantly decreased forced vital capacity (FVC) and forced expiratory volume in one second (FEV1) at 0.12 ppm; this is effect is greater at higher concentrations.
Odour Safety Factor(OSF)
OSF=1.1 (OZONE)
For nitric oxide:
Odour Threshold: 0.3 to 1 ppm.
NOTE: Detector tubes for nitrogen oxide, measuring in excess of 10 ppm, are commercially available.
Experimental animal date indicates that nitric oxide is one-fifth as toxic as nitrogen dioxide. The recommended TLV-TWA takes account of this relationship.
Exposure at or below the recommended TLV-TWA is thought to reduce the potential for immediate injury, adverse physiological effects, pulmonary disease (including the risk of increased airway resistance) from prolonged daily exposure
Odour Safety Factor (OSF)

OSF=7.7 (nitric oxide)

For silicon
CEL TWA: 5 mg/m3
(CEL = Chemwatch Exposure Limit)
NOTE: The CEL TWA is consistent with the value recommended in the Norwegian ferro-alloy industry (furnace room dust/mixed dust).
Silicon dust appears to have little adverse effect on the lungs and is not implicated in the genesis of organic disease or in the production of toxic effects. The TLV-TWA is thought to be protective against physical irritation and possible chronic respiratory effects encountered at higher levels.

For yellow (white) phosphorus:
Phosphorus (yellow) is an acute irritant of the respiratory tract. Chronic poisoning by phosphorus is characterised by necrosis of the bone, usually the mandible. Hepatic cirrhosis, electrolyte imbalance, myocardial disruption, and renal cortical necrosis appear in both acute and chronic phosphorus intoxication. Although exposure to phosphorus is expected in the particulate phase, yellow phosphorus possesses significant vapour pressure (0.026 torr, 20 C) and produces a saturated vapour concentration (170 mg/m3, 20 C).
The TLV-TWA for yellow phosphorus is recommended to minimise the potential for acute poisoning, but the margin of safety for this value in the prevention of possible effects of chronic occupational exposure to yellow phosphorus is not know.

Exposed individuals are **NOT** reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

Class	OSF	Description
A	550	Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities
B	26-550	As "A" for 50-90% of persons being distracted
C	1-26	As "A" for less than 50% of persons being distracted
D	0.18-1	10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached
E	<0.18	As "D" for less than 10% of persons aware of being tested

For aluminium oxide:
The experimental and clinical data indicate that aluminium oxide acts as an "inert" material when inhaled and seems to have little effect on the lungs nor does it produce significant organic disease or toxic effects when exposures are kept under reasonable control.
[Documentation of the Threshold Limit Values], ACGIH, Sixth Edition

For amorphous crystalline silica (precipitated silicic acid):
Amorphous crystalline silica shows little potential for producing adverse effects on the lung and exposure standards should reflect a particulate of low intrinsic toxicity. Mixtures of amorphous silicas/ diatomaceous earth and crystalline silica should be monitored as if they comprise only the crystalline forms.
The dusts from precipitated silica and silica gel produce little adverse effect on pulmonary functions and are not known to produce significant disease or toxic effect.
IARC has classified silica, amorphous as Group 3: **NOT** classifiable as to its carcinogenicity to humans.
Evidence of carcinogenicity may be inadequate or limited in animal testing.

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.
Special ventilation requirements apply for processes which result in the generation of barium, chromium, lead, or nickel fume and in those processes which generate ozone.
The use of mechanical ventilation by local exhaust systems is required as a minimum in all circumstances (including outdoor work). (In confined spaces always check that oxygen has not been depleted by excessive rusting of steel or snowflake corrosion of aluminium)
Local exhaust systems must be designed to provide a minimum capture velocity at the fume source, away from the worker, of 0.5 metre/sec. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
welding, brazing fumes (released at relatively low velocity into moderately still air)	0.5-1.0 m/s (100-200 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of welding or brazing fumes generated 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

For manual arc welding operations the nature of ventilation is determined by the location of the work.
- For outdoor work, natural ventilation is generally sufficient.
- For indoor work, conducted in open spaces, use mechanical (general exhaust or plenum) ventilation. (Open work spaces exceed 300 cubic metres per welder)
- For work conducted in limited or confined spaces, mechanical ventilation, using local exhaust systems, is required. (In confined spaces always check that oxygen has not been depleted by excessive rusting of steel or snowflake corrosion of aluminium)

Mechanical or local exhaust ventilation may not be required where the process working time does not exceed 24 mins. (in an 8 hr. shift) provided the work is intermittent (a maximum of 5 mins. every hour). Local exhaust systems must be designed to provide a minimum capture velocity at the fume source, away from the worker, of 0.5 metre/sec. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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Metal dusts must be collected at the source of generation as they are potentially explosive.
- Avoid ignition sources.
- Good housekeeping practices must be maintained.
- Dust accumulation on the floor, ledges and beams can present a risk of ignition, flame propagation and secondary explosions.
- Do not use compressed air to remove settled materials from floors, beams or equipment
- Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation.
- Use non-sparking handling equipment, tools and natural bristle brushes. Cover and reseal partially empty containers. Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations.
- Do not allow chips, fines or dusts to contact water, particularly in enclosed areas.
- Metal spraying and blasting should, where possible, be conducted in separate rooms. This minimises the risk of supplying oxygen, in the form of metal oxides, to potentially reactive finely divided metals such as aluminium, zinc, magnesium or titanium.
- Work-shops designed for metal spraying should possess smooth walls and a minimum of obstructions, such as ledges, on which dust accumulation is possible.
- Wet scrubbers are preferable to dry dust collectors.
- Bag or filter-type collectors should be sited outside the workrooms and be fitted with explosion relief doors.
- Cyclones should be protected against entry of moisture as reactive metal dusts are capable of spontaneous combustion in humid or partially wetted states.
- Local exhaust systems must be designed to provide a minimum capture velocity at the fume source, away from the worker, of

	0.5 metre/sec. ▸ Local ventilation and vacuum systems must be designed to handle explosive dusts. Dry vacuum and electrostatic precipitators must not be used, unless specifically approved for use with flammable/ explosive dusts. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant. Within each range the appropriate value depends on: Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

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Individual protection measures, such as personal protective equipment	
Eye and face protection	▸ Goggles or other suitable eye protection shall be used during all gas welding or oxygen cutting operations. Spectacles without side shields, with suitable filter lenses are permitted for use during gas welding operations on light work, for torch brazing or for inspection. ▸ For most open welding/brazing operations, goggles, even with appropriate filters, will not afford sufficient facial protection for operators. Where possible use welding helmets or handshields corresponding to EN 175, ANSI Z49:12005, AS 1336 and AS 1338 which provide the maximum possible facial protection from flying particles and fragments. [WRIA-WTIA Technical Note 7] ▸ An approved face shield or welding helmet can also have filters for optical radiation protection, and offer additional protection against debris and sparks. ▸ UV blocking protective spectacles with side shields or welding goggles are considered primary protection, with the face shield or welding helmet considered secondary protection. ▸ The optical filter in welding goggles, face mask or helmet must be a type which is suitable for the sort of work being done.A filter suitable for gas welding, for instance, should not be used for arc welding. ▸ Face masks which are self dimming are available for arc welding, MIG, TIG and plasma cutting, and allow better vision before the arc is struck and after it is extinguished. For submerged arc welding use a lens shade which gives just sufficient arc brightness to allow weld pool control.
Skin protection	See Hand protection below
Hands/feet protection	The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as:

	· Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. ‣ Protective gloves eg. Leather gloves or gloves with Leather facing ‣ Welding gloves conforming to Standards such as EN 12477:2001, ANSI Z49.1, AS/NZS 2161:2008 produced from leather, rubber, treated cotton,or alumininised ‣ These gloves protect against mechanical risk caused by abrasion, blade cut, tear and puncture ‣ Other gloves which protect against thermal risks (heat and fire) might also be considered - these comply with different standards to those mentioned above. ‣ One pair of gloves may not be suitable for all processes. For example, gloves that are suitable for low current Gas Tungsten Arc Welding (GTAW) (thin and flexible) would not be proper for high-current Air Carbon Arc Cutting (CAC-A) (insulated, tough, and durable) Experience indicates that the following polymers are suitable as glove materials for protection against undissolved, dry solids, where abrasive particles are not present. ‣ polychloroprene. ‣ nitrile rubber. ‣ butyl rubber. ‣ fluorocaoutchouc. ‣ polyvinyl chloride. Gloves should be examined for wear and/ or degradation constantly.
Body protection	See Other protection below
Other protection	Before starting; consider that protection should be provided for all personnel within 10 metres of any open arc welding operation. Welding sites must be adequately shielded with screens of non flammable materials. Screens should permit ventilation at floor and ceiling levels. ‣ Overalls. ‣ P.V.C apron. ‣ Barrier cream. ‣ Skin cleansing cream. ‣ Eye wash unit.

Respiratory protection

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

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	@1@ P2	-	-
	Air-line*	-	-
up to 50 x ES	Air-line**	@1@ P2	@1@ PAPR-P2
	-	Air-line*	-

up to 100 x ES	-	Air-line**	@1@ PAPR-P3

* - Negative pressure demand ** - Continuous flow
A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)
Welding of powder coated metal requires good general area ventilation, and ventilated mask as local heat causes minor coating decomposition releasing highly discomforting fume which may be harmful if exposure is regular.
Welding or flame cutting of metals with chromate pigmented primers or coatings may result in inhalation of highly toxic chromate fumes. Exposures may be significant in enclosed or poorly ventilated areas
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	Copper Colour

Physical state	Solid	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Applicable
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	‣ Unstable in the presence of incompatible materials. ‣ Product is considered stable. ‣ Hazardous polymerisation will not occur. ‣ Presence of heat source and ignition source
Possibility of hazardous reactions	See section 7

Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting. Fumes evolved during welding operations may be irritating to the upper-respiratory tract and may be harmful if inhaled. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure. Not normally a hazard due to non-volatile nature of product Acute carbon monoxide exposure can mimic acute gastroenteritis or food poisoning with accompanying nausea and vomiting. Rapidly fatal cases of poisoning are characterised by congestion and hemorrhages in all organs. The extent of the tissue and organ damage is related to the duration of the post-hypoxic unconsciousness. Exposure to carbon monoxide can result in immediate effects and, depending on the severity of the exposure, delayed effects. These delayed effects may occur days to weeks after the initial exposure. Signs of brain or nerve injury may appear at any time within three weeks following an acute exposure. Characteristically, those patients manifesting delayed neuropathology are middle aged or older. Most of the neurological symptoms associated with carbon monoxide exposure can resolve within a year but memory deficits and gait disturbances may remain Symptoms of poisoning resulting from carbon monoxide exposure include respiratory disorders, diarrhoea and shock. Carbon monoxide competes with oxygen for haemoglobin binding sites and has a 240-fold affinity for these sites compared to oxygen. In addition to oxygen deficiency further disability is produced by the formation of carboxymyoglobin (COHb) in muscles, to produce disturbances in muscle metabolism, particularly that of the heart. The tissues most affected by carbon monoxide are those which are most sensitive to oxygen deprivation such as the brain and the heart. The overt lesion in these tissues is mostly haemorrhage. The severe headache associated with exposure is believed to be caused by cerebral oedema and increased intracranial pressure resulting from excessive transudate leakage of fluids through the hypoxic capillaries. Carbon monoxide induced hypoxia in the cochlea and brain stem leads to central hearing loss and vestibular dysfunction (vertigo, nausea, vomiting) with the vestibular symptoms usually more prominent than the hearing loss At low levels carbon monoxide may cause poor concentration, memory and vision problems, vertigo, muscular weakness and loss of muscle coordination, rapid and stretorous breathing, intermittent heart beat, loss of sphincter control and rarely coma and death. At higher levels (200 ppm for 2-3 hours), it may cause headaches, fatigue and nausea. At very high levels (400 ppm) the symptoms intensify and will be life-threatening after three hours. Exposure to levels of 1200 ppm or greater are immediately dangerous to life. When carbon monoxide levels in air exceed 3% (30,000 ppm), death occurs almost at once. Carbon monoxide is not a cumulative poison since COHb is fully dissociable and once exposure has ceased, the hemoglobin will revert to oxyhemoglobin. The biological half life of carbon monoxide in the blood in sedentary adults is 2- 5 hours and the elimination becomes slower as the concentration decreases. Manganese fume is toxic and produces nervous system effects characterised by tiredness. Acute poisoning is rare although acute inflammation of the lungs may occur. A chemical pneumonia may also result from frequent exposure. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure. Copper poisoning following exposure to copper dusts and fume may result in headache, cold sweat and weak pulse. Capillary, kidney, liver and brain damage are the longer term manifestations of such poisoning. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure. Inhalation of dusts, generated by the material during the course of normal handling, may be damaging to the health of the individual.

Ingestion	Severely toxic effects may result from the accidental ingestion of the material; animal experiments indicate that ingestion of less than 5 gram may be fatal or may produce serious damage to the health of the individual. Not normally a hazard due to the physical form of product. The material is a physical irritant to the gastro-intestinal tract Numerous cases of a single oral exposure to high levels of copper have been reported. Consumption of copper-contaminated drinking water has been associated with mainly gastrointestinal symptoms including nausea, abdominal pain, vomiting and diarrhoea. A metallic taste, nausea, vomiting and epigastric burning often occur after ingestion of copper and its derivatives. The vomitus is usually green/blue and discolours contaminated skin. Acute poisonings from the ingestion of copper salts are rare due to their prompt removal by vomiting. Vomiting is due mainly to the local and astringent action of copper ion on the stomach and bowel. Emesis usually occurs within 5 to 10 minutes but may be delayed if food is present in the stomach. Should vomiting not occur, or is delayed, gradual absorption from the bowel may result in systemic poisoning with death, possibly, following within several days. Apparent recovery may be followed by lethal relapse. Systemic effects of copper resemble other heavy metal poisonings and produce wide-spread capillary damage, kidney and liver damage and central nervous system excitation followed by depression. Haemolytic anaemia (a result of red-blood cell damage) has been described in acute human poisoning. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products.] Other symptoms of copper poisoning include lethargy, neurotoxicity, and increased blood pressure and respiratory rates. Coma and death have followed attempted suicides using solutions of copper sulfate. Copper is an essential element and most animal tissues have measurable amounts of copper associated with them. Humans have evolved mechanisms which maintain is availability whilst limiting its toxicity (homeostasis). Copper is initially bound in the body to a blood-borne protein, serum albumin and thereafter is more firmly bound to another protein, alpha-ceruloplasmin. Such binding effectively "inactivates" the copper, thus reducing its potential to produce toxic damage. In healthy individuals, bound copper can reach relatively high levels without producing adverse health effects. Excretion in the bile represents the major pathway by which copper is removed from the body when it reaches potentially toxic levels. Copper may also be stored in the liver and bone marrow where it is bound to another protein, metallothionein. A combination of binding and excretion ensures that the body is able to tolerate relatively high loadings of copper. Poisonings rarely occur after oral administration of manganese salts as they are generally poorly absorbed from the gut (generally less than 4%) and seems to be dependent, in part, on levels of dietary iron and may increase following the consumption of alcohol. A side-effect of oral manganese administration is an increase in losses of calcium in the faeces and a subsequent lowering of calcium blood levels. Absorbed manganese tends to be slowly excreted in the bile. Divalent manganese appears to be 2.5-3 times more toxic than the trivalent form.
Skin Contact	Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. Limited evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. 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. Ultraviolet radiation (UV) is generated by the electric arc in the welding process. Skin exposure to UV can result in severe burns, in many cases without prior warning. Exposure to infrared radiation (IR), produced by the electric arc and other flame cutting equipment may heat the skin surface and the tissues immediately below the surface. Except for this effect, which can progress to thermal burns in some situations, infrared radiation is not dangerous to welders. Most welders protect themselves from IR (and UV) with a welder's helmet (or glasses) and protective clothing. 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. Special ventilation requirements apply for processes which result in the generation of aluminium, copper, fluoride, manganese or zinc fume. ‣ For work conducted outdoors and in open work spaces, the use of mechanical (general exhaust or plenum) ventilation is required as a minimum. (Open work spaces exceed 300 cubic meters per welder) ‣ For indoor work, conducted in limited or confined work spaces, use of mechanical ventilation by local exhaust systems is mandatory. (In confined spaces always check that oxygen has not been depleted by excessive rusting of steel or snowflake corrosion of aluminium) Local exhaust systems must be designed to provide a minimum capture velocity at the fume source, away from the worker, of 0.5 metre/sec. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

	<table><tr><td>Type of Contaminant:</td><td>Air Speed:</td></tr><tr><td>welding, brazing fumes (released at relatively low velocity into moderately still air)</td><td>0.5-1.0 m/s (100-200 f/min.)</td></tr></table> Within each range the appropriate value depends on: <table><tr><td>Lower end of the range</td><td>Upper end of the range</td></tr><tr><td>1: Room air currents minimal or favourable to capture</td><td>1: Disturbing room air currents</td></tr><tr><td>2: Contaminants of low toxicity or of nuisance value only.</td><td>2: Contaminants of high toxicity</td></tr><tr><td>3: Intermittent, low production.</td><td>3: High production, heavy use</td></tr><tr><td>4: Large hood or large air mass in motion</td><td>4: Small hood-local control only</td></tr></table> Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of welding or brazing fumes generated 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used. Irritation and skin reactions are possible with sensitive skin Exposure to copper, by skin, has come from its use in pigments, ointments, ornaments, jewellery, dental amalgams and IUDs and as an antifungal agent and an algicide. Although copper algicides are used in the treatment of water in swimming pools and reservoirs, there are no reports of toxicity from these applications. Reports of allergic contact dermatitis following contact with copper and its salts have appeared in the literature, however the exposure concentrations leading to any effect have been poorly characterised. In one study, patch testing of 1190 eczema patients found that only 13 (1.1%) cross-reacted with 2% copper sulfate in petrolatum. The investigators warned, however, that the possibility of contamination with nickel (an established contact allergen) might have been the cause of the reaction. Copper salts often produce an itching eczema in contact with skin. This is, likely, of a non-allergic nature. 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	Limited evidence exists, or practical experience suggests, that the material may cause eye irritation in a substantial number of individuals and/or is expected to produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. Ultraviolet (UV) radiation can also damage the lens of the eye. Many arc welders are aware of the condition known as "arc-eye," a sensation of sand in the eyes. This condition is caused by excessive eye exposure to UV. Exposure to ultraviolet rays may also increase the skin effects of some industrial chemicals (coal tar and cresol compounds, for example). Exposure of the human eye to intense visible light can produce adaptation, pupillary reflex, and shading of the eyes. Such actions are protective mechanisms to prevent excessive light from being focused on the retina. In the arc welding process, eye exposure to intense visible light is prevented for the most part by the welder's helmet. However, some individuals have sustained retinal damage due to careless "viewing" of the arc. At no time should the arc be observed without eye protection. Copper salts, in contact with the eye, may produce conjunctivitis or even ulceration and turbidity of the cornea. Contact with the eye, by metal dusts, may produce mechanical abrasion or foreign body penetration of the eyeball. Iron particles embedded in the eye may produce a condition known as ocular siderosis; effects include discolouration of the cornea and iris and pupillary effects such as poor reaction to light and accommodation. Particles entering the lens may produce cataracts. A rare consequence of ocular siderosis is glaucoma.
Chronic	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. 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 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. 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
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.
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.
Long-term (chronic) exposure to low levels of carbon monoxide may produce heart disease and damage to the nervous system. Exposure of pregnant animals to carbon monoxide may cause low birthweight, increased foetal mortality and nervous system damage to the offspring.
Carbon monoxide is a common cause of fatal poisoning in industry and homes. Non fatal poisoning may result in permanent nervous system damage. Carbon monoxide reduces the oxygen carrying capacity of the blood. Effects on the body are considered to be reversible as long as brain cell damage or heart failure has not occurred. Avoid prolonged exposure, even to small concentrations. A well-established and probably causal relationship exists between maternal smoking (resulting in carboxyhaemoglobin levels of 2-7% in the foetus) and low birth weight. There also appears to be a dose-related increase in perinatal deaths and a retardation of mental ability in infants born to smoking mothers.
The foetus and newborn infant are considered to be very susceptible to CO exposure for several reasons:

- Foetal hemoglobin has a greater affinity for CO than maternal hemoglobin.
- Due to differences in uptake and elimination of CO, the fetal circulation is likely to have COHb levels higher (up to 2.5 times) than seen in the maternal circulation.
- The half-life of COHb in fetal blood is 3 times longer than that of maternal blood.
- Since the fetus has a comparatively high rate of O2 consumption, and a lower O2 tension in the blood than adults, a compromised O2 transport has the potential to produce a serious hypoxia.

Carbon monoxide gas readily crosses the placenta and CO exposure during pregnancy can be teratogenic.
Carbon dioxide at low levels may initiate or enhance deleterious myocardial alterations in individuals with restricted coronary artery blood flow and decreased myocardial lactate production. - Linde
On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

Principal route of exposure is inhalation of welding fumes from electrodes and workpiece. Reaction products arising from electrode core and flux appear as welding fume depending on welding conditions, relative volatilities of metal oxides and any coatings on the workpiece. Studies of lung cancer among welders indicate that they may experience a 30-40% increased risk compared to the general population. Since smoking and exposure to other cancer-causing agents, such as asbestos fibre, may influence these results, it is not clear whether welding, in fact, represents a significant lung cancer risk. Whilst mild steel welding represents little risk, the stainless steel welder, exposed to chromium and nickel fume, may be at risk and it is this factor which may account for the overall increase in lung cancer incidence among welders. Cold isolated electrodes are relatively harmless.
Metal oxides generated by industrial processes such as welding, give rise to a number of potential health problems. Particles smaller than 5 micron (respirables) articles 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.

Exposure to fume containing high concentrations of water-soluble chromium (VI) during the welding of stainless steels in confined spaces has been reported to result in chronic chrome intoxication, dermatitis and asthma. Certain insoluble chromium (VI) compounds have been named as carcinogens (by the ACGIH) in other work environments. Chromium may also appear in welding fumes as Cr2O3 or double oxides with iron. These chromium (III) compounds are generally biologically inert.
Welding fume with high levels of ferrous materials may lead to particle deposition in the lungs (siderosis) after long exposure. This clears up when exposure stops. Chronic exposure to iron dusts may lead to eye disorders.
Silica and silicates in welding fumes are non-crystalline and believed to be non-harmful.
Other welding process exposures can arise from radiant energy UV flash burns, thermal burns or electric shock
The welding arc emits ultraviolet radiation at wavelengths that have the potential to produce skin tumours in animals and in over-exposed individuals, however, no confirmatory studies of this effect in welders have been reported.

KangarooArc - ER7OS-6 Welding Wire	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

Legend: *1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*

KangarooArc - ER7OS-6 Welding Wire

Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens).
Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis.
Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.
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 (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).
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.

WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
Most welding is performed using electric arc processes - manual metal arc, metal inert gas (MIG) and tungsten inert gas welding (TIG) – and most welding is on mild steel.
In 2017, an IARC working group has determined that "sufficient evidence exists that welding fume is a human lung carcinogen (Group 1).
A complicating factor in classifying welding fumes is its complexity. Generally, welding fume is a mixture of metal fumes (i.e., iron, manganese, chromium, nickel, silicon, titanium) and gases (i.e., carbon monoxide, ozone, argon, carbon dioxide). Welding fume can contain varying concentrations of individual components that are classified as human carcinogens, including hexavalent chrome and nickel. However the presence of such metals and the intensity of exposure to each differ significantly according to a number of variables, including the type of welding technique used and the composition of the base metal and consumable. Nonetheless, IARC did not differentiate between these variables in its decision.
There has been considerable evidence over several decades regarding cancer risks in relation to welding activities. Several case-control studies reported excess risks of ocular melanoma in welders. This association may be due to the presence in some

	welding environments of fumes of thorium-232, which is used in tungsten welding rods Different welding environments may present different and complex profiles of exposures. In one study to characterise welding fume aerosol nanoparticles in mild steel metal active gas welding showed a mass median diameter (MMMD) of 200-300 nm. A widespread consensus seems to have formed to the effect that some welding environments, notably in stainless steel welding, do carry risks of lung cancer. This widespread consensus is in part based on empirical evidence regarding risks among stainless steel welders and in part on the fact that stainless steel welding entails moderately high exposure to nickel and chromium VI compounds, which are recognised lung carcinogens. The corollary is that welding without the presence of nickel and chromium VI compounds, namely mild-steel welding, should not carry risk. But it appears that this line of reasoning in not supported by the accumulated body of epidemiologic evidence. While there remained some uncertainty about possible confounding by smoking and by asbestos, and some possible publication bias, the overwhelming evidence is that there has been an excess risk of lung cancer among welders as a whole in the order of 20%-40%.The most begrudging explanation is that there is an as-yet unexplained common reason for excess lung cancer risks that applies to all types of welders. It has been have proposed that iron fumes may play such a role, and some Finnish data appear to support this hypothesis, though not conclusively. This hypothesis would also imply that excess lung cancer risks among welders are not unique to welders, but rather may be shared among many types of metal working occupations. Welders are exposed to a range of fumes and gases (evaporated metal, metal oxides, hydrocarbons, nanoparticles, ozone, oxides of nitrogen (NOx)) depending on the electrodes, filler wire and flux materials used in the process, but also physical exposures such as electric and magnetic fields (EMF) and ultraviolet (UV) radiation. Fume particles contain a wide variety of oxides and salts of metals and other compounds, which are produced mainly from electrodes, filler wire and flux materials. Fumes from the welding of stainless-steel and other alloys contain nickel compounds and chromium[VI] and [III]. Ozone is formed during most electric arc welding, and exposures can be high in comparison to the exposure limit, particularly during metal inert gas welding of aluminium. Oxides of nitrogen are found during manual metal arc welding and particularly during gas welding. Welders who weld painted mild steel can also be exposed to a range of organic compounds produced by pyrolysis. In one study particle elemental composition was mainly iron and manganese. Ni and Cr exposures were very low in the vicinity of mild steel welders, but much higher in the background in the workshop where there presumably was some stainless steel welding. Personal exposures to manganese ranged from 0.01-4.93 mg/m3 and to iron ranged from 0.04-16.29 mg/m3 in eight Canadian welding companies. Types of welding identified were mostly (90%) MIG mild steel, MIG stainless steel, and TIG aluminum. Carbon monoxide levels were less than 5.0 ppm (at source) and ozone levels varied from 0.4-0.6 ppm (at source). Welders, especially in shipyards, may also be exposed to asbestos dust. Physical exposures such as electric and magnetic fields (EMF) and ultraviolet (UV) radiation are also common. In all, the in vivo studies suggest that different welding fumes cause varied responses in rat lungs in vivo , and the toxic effects typically correlate with the metal composition of the fumes and their ability to produce free radicals. In many studies both soluble and insoluble fractions of the stainless steel welding fumes were required to produce most types of effects, indicating that the responses are not dependent exclusively on the soluble metals Lung tumourigenicity of welding fumes was investigated in lung tumour susceptible (A/J) strain of mice. Male mice were exposed by pharyngeal aspiration four times (once every 3 days) to 85 ug of gas metal arc-mild steel (GMA-MS), GMA-SS, or manual metal arc-SS (MMA-SS) fume. At 48 weeks post-exposure, GMA-SS caused the greatest increase in tumour multiplicity and incidence, but did not differ from sham exposure. Tumour incidence in the GMA-SS group versus sham control was close to significance at 78 weeks post exposure. Histopathological analysis of the lungs of these mice showed the GMA-SS group having an increase in preneoplasia/tumour multiplicity and incidence compared to the GMA-MS and sham groups at 48 weeks. The increase in incidence in the GMA-SS exposed mice was significant compared to the GMA-MS group but not to the sham-exposed animals, and the difference in incidence between the GMA-SS and MMA-SS groups was of border-line significance (p = 0.06). At 78 week s post-exposure, no statistically significant differences A significantly higher frequency of micronuclei in peripheral blood lymphocytes (binucleated cell assay) and higher mean levels of both centromere-positive and centromere-negative micronuclei was observed in welders (n=27) who worked without protective device compared to controls (n=30).The rate of micronucleated cells did not correlate with the duration of exposure

Acute Toxicity	✔	**Carcinogenicity**	✔
Skin Irritation/Corrosion	✘	**Reproductivity**	✘
Serious Eye Damage/Irritation	✔	**STOT - Single Exposure**	✘
Respiratory or Skin sensitisation	✘	**STOT - Repeated Exposure**	✘
Mutagenicity	✘	**Aspiration Hazard**	✘

Legend: ✘ *– Data either not available or does not fill the criteria for classification*
✔ *– Data available to make classification*

SECTION 12 Ecological information

Toxicity

	Endpoint	Test Duration (hr)	Species	Value	Source
KangarooArc - ER7OS-6 Welding Wire	Not Available	Not Available	Not Available	Not Available	Not Available

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

	Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.
Harmful to aquatic organisms.
For Metal:
Atmospheric Fate - Metal-containing inorganic substances generally have negligible vapour pressure and are not expected to partition to air.
Environmental Fate: Environmental processes, such as oxidation, the presence of acids or bases and microbiological processes, may transform insoluble metals to more soluble ionic forms. Environmental processes may enhance bioavailability and may also be important in changing solubilities.
Aquatic/Terrestrial Fate: When released to dry soil, most metals will exhibit limited mobility and remain in the upper layer; some will leach locally into ground water and/ or surface water ecosystems when soaked by rain or melt ice. A metal ion is considered infinitely persistent because it cannot degrade further. Once released to surface waters and moist soils their fate depends on solubility and dissociation in water. A significant proportion of dissolved/ sorbed metals will end up in sediments through the settling of suspended particles. The remaining metal ions can then be taken up by aquatic organisms. Ionic species may bind to dissolved ligands or sorb to solid particles in water.
Ecotoxicity: Even though many metals show few toxic effects at physiological pH levels, transformation may introduce new or magnified effects.
For copper:
Atmospheric Fate - Copper is unlikely to accumulate in the atmosphere due to a short residence time for airborne copper aerosols. Airborne coppers, however, may be transported over large distances. Air Quality Standards: no data available.
Aquatic Fate: Toxicity of copper is affected by pH and hardness of water. Total copper is rarely useful as a predictor of toxicity. In natural sea water, more than 98% of copper is organically bound and in river waters a high percentage is often organically bound, but the actual percentage depends on the river water and its pH.
Ecotoxicity: Copper accumulates significantly in the food chain. The toxic effect of copper in the aquatic biota depends on the bio-availability of copper in water which, in turn, depends on its physico-chemical form (i.e. speciation). Bioavailability is decreased by complexation and adsorption of copper by natural organic matter, iron and manganese hydrated oxides, and chelating agents excreted by algae and other aquatic organisms. Copper exhibits significant toxicity in some aquatic organisms. Some algal species are very sensitive to copper. Silicate, iron, manganese and EDTA may reduce bioavailability.
For copper: Ecotoxicity - Significant effects are expected on various species of microalgae, some species of macroalgae, and a range of invertebrates, including crustaceans, gastropods and sea urchins. Copper is moderately toxic to crab and their larvae and is highly toxic to gastropods (mollusks, including oysters, mussels and clams). In fish, the acute lethal concentrations of copper depends both on test species and exposure conditions. Waters with high concentrations of copper can have significant effects on diatoms and sensitive invertebrates, notably cladocerans (water fleas). Most taxonomic groups of macroalgae and invertebrates will be severely affected.
For Copper: Typical foliar levels of copper are: Uncontaminated soils (0.3-250 mg/kg) ; Contaminated soils (150-450 mg/kg) ; Mining/smelting soils (6.1-25 mg/kg80 mg/kg300 mg/kg).
Terrestrial Fate: Plants - Generally, vegetation reflects soil copper levels in its foliage. This is dependent upon the bioavailability of copper and the physiological requirements of species concerned. Crops are often more sensitive to copper than the native flora. Soil: In soil, copper levels are raised by application of fertilizer, fungicides, from deposition of highway dusts and from urban, mining and industrial sources. Chronic and or acute effects on sensitive species occur as a result of human activities such as copper fertilizer addition and addition of sludge. When soil levels exceed 150 mg Cu/kg, native and agricultural species show chronic effects. Soils in the range 500-1000 mg Cu/kg act in a strongly selective fashion allowing the survival of only copper-tolerant species and strains. At 2000 Cu mg/kg, most species cannot survive. By 3500 mg Cu/kg, areas are largely devoid of vegetation cover. The organic content of the soil appears to be a key factor affecting the bioavailability of copper. On normal forest soils, non-rooted plants such as mosses and lichens show higher copper concentrations. The fruiting bodies and mycorrhizal sheaths of soil fungi associated with higher plants in forests often accumulate copper to much higher levels than plants at the same site.
For manganese and its compounds:
Environmental fate:
It has been established that while lower organisms (e.g., plankton, aquatic plants, and some fish) can significantly bioconcentrate manganese, higher organisms (including humans) tend to maintain manganese homeostasis. This indicates that the potential for biomagnification of manganese from lower trophic levels to higher ones is low.
There were two mechanisms involved in explaining the retention of manganese and other metals in the environment by soil. First, through cation exchange reactions, manganese ions and the charged surface of soil particles form manganese oxides, hydroxides, and oxyhydroxides which in turn form absorption sites for other metals. Secondly, manganese can be adsorbed to other oxides, hydroxides, and oxyhydroxides through ligand exchange reactions. When the soil solution becomes saturated, these manganese oxides, hydroxides, and oxyhydroxides can precipitate into a new mineral phase and act as a new surface to which other substances can absorb. The tendency of soluble manganese compounds to adsorb to soils and sediments depends mainly on the cation exchange capacity and the organic composition of the soil. The soil adsorption constants (the ratio of the concentration in soil to the concentration in water) for Mn(II) span five orders of magnitude, ranging from 0.2 to 10,000 mL/g, increasing as a function of the organic content and the ion exchange capacity of the soil; thus, adsorption may be highly variable. In some cases, adsorption of manganese to soils may not be a readily reversible process. At low concentrations, manganese may be "fixed" by clays and will not be released into solution readily. At higher concentrations, manganese may be desorbed by ion exchange mechanisms with other ions in solution. For example, the discharge of waste water effluent into estuarine environments resulted in the mobilization of manganese from the bottom sediments. The metals in the effluent may have been preferentially adsorbed resulting in the release of manganese. The oxidation state of manganese in soil and sediments may be altered by microbial activity; oxidation may lead to the precipitation of manganese. Bacteria and microflora can increase the mobility of manganese.
The transport and partitioning of manganese in water is controlled by the solubility of the specific chemical form present, which in turn is determined by pH, Eh (oxidation-reduction potential), and the characteristics of the available anions. The metal may exist in water in any of four oxidation states.
Manganese(II) predominates in most waters (pH 4-7) but may become oxidized at a pH >8 or 9. The principal anion associated with Mn(II) in water is usually carbonate (CO_3^{2}), and the concentration of manganese is limited by the relatively low solubility (65 mg/L) of $MnCO_3$. In relatively oxidized water, the solubility of Mn(II) may be controlled by manganese oxide equilibria, with manganese being converted to the Mn(II) or Mn(IV) oxidation states. In extremely reduced water, the fate of manganese tends to be controlled by formation of a poorly soluble sulfide. Manganese in water may undergo oxidation at high pH or Eh and is also subject to microbial activity. For example, Mn(II) in a lake was oxidized during the summer months, but this was inhibited by a microbial poison, indicating that the oxidation was mediated by bacteria . The microbial metabolism of manganese is presumed to be a function of pH, temperature, and other factors.
Manganese in water may be significantly bioconcentrated at lower trophic levels. A bioconcentration factor (BCF) relates the concentration of a chemical in plant and animal tissues to the concentration of the chemical in the water in which they live. The BCF of manganese was estimated as 2,500 - 6,300 for phytoplankton, 300 -5,500 for marine algae, 80 - 830 for intertidal mussels, and 35 - 930 for coastal fish. Similarly, the BCF of manganese was estimated to be 10,00 -20,000 for marine and freshwater plants, 10,000 - 40,000 for invertebrates, and 10 - 600 for fish. In general, these data indicate that lower organisms such as algae have larger BCFs than higher organisms. In order to protect consumers from the risk of manganese bioaccumulation in marine mollusks, the U.S. EPA has set a criterion for manganese at 0.1 mg/L for marine waters.
Elemental manganese and inorganic manganese compounds have negligible vapor pressures but may exist in air as suspended particulate matter derived from industrial emissions or the erosion of soils. Manganese-containing particles are mainly removed from the atmosphere by gravitational settling, with large particles tending to fall out faster than small particles. The half-life of airborne particles is usually on the order of days, depending on the size of the particle and

atmospheric conditions. Some removal by washout mechanisms such as rain may also occur, although it is of minor significance in comparison to dry deposition.
Ecotoxicity:
Manganese ion is toxic to aqueous organisms
Fish LC50 (28 d): orfe 2490 mg/l, trout 2.91 mg/l
Daphnia magna LC50: 50 mg/l
Pseudomonas putida LC50: 10.6 mg/l
Photobacterium phosphoreum LC50: 14.7 mg/l
Turbellarian worms (EC0): Polycelis nigra 660 mg/l (interference threshold); microregma 31 mg/l
For Amorphous Silica: Amorphous silica is chemically and biologically inert. It is not biodegradable.
Aquatic Fate: Due to its insolubility in water there is a separation at every filtration and sedimentation process. On a global scale, the level of man-made synthetic amorphous silicas (SAS) represents up to 2.4% of the dissolved silica naturally present in the aquatic environment and untreated SAS have a relatively low water solubility and an extremely low vapour pressure. Biodegradability in sewage treatment plants or in surface water is not applicable to inorganic substances like SAS.
Terrestrial Fate: Crystalline and/or amorphous silicas are common on the earth in soils and sediments, and in living organisms (e.g. diatoms), but only the dissolved form is bioavailable. On the basis of these properties it is expected that SAS released into the environment will be distributed mainly into soil/sediment. Surface treated silica will be wetted then adsorbed onto soils and sediments.
Atmospheric Fate: SAS is not expected to be distributed into the air if released.
Ecotoxicity: SAS is not toxic to environmental organisms (apart from physical desiccation in insects). SAS presents a low risk for adverse effects to the environment.
For Silica:
Environmental Fate: Most documentation on the fate of silica in the environment concerns dissolved silica, in the aquatic environment, regardless of origin, (man-made or natural), or structure, (crystalline or amorphous).
Terrestrial Fate: Silicon makes up 25.7% of the Earth's crust, by weight, and is the second most abundant element, being exceeded only by oxygen. Silicon is not found free in nature, but occurs chiefly as the oxide and as silicates. Once released into the environment, no distinction can be made between the initial forms of silica.
Aquatic Fate: At normal environmental pH, dissolved silica exists exclusively as monosilicic acid. At pH 9.4, amorphous silica is highly soluble in water. Crystalline silica, in the form of quartz, has low solubility in water. Silicic acid plays an important role in the biological/geological/chemical cycle of silicon, especially in the ocean. Marine organisms such as diatoms, silicoflagellates and radiolarians use silicic acid in their skeletal structures and their skeletal remains leave silica in sea sediment
Ecotoxicity: Silicon is important to plant and animal life and is practically non-toxic to fish including zebrafish, and Daphnia magna water fleas.

DO NOT discharge into sewer or waterways.

Persistence and degradability

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

Bioaccumulative potential

Ingredient	Bioaccumulation
	No Data available for all ingredients

Mobility in soil

Ingredient	Mobility
	No Data available for all ingredients

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ 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	NO
HAZCHEM	Not Applicable

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

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

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

14.7.1. 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
Iron	Not Available
Carbon	Not Available
Silicon	Not Available
Manganese	Not Available
Sulfur	Not Available
Phosphorus	Not Available
Copper	Not Available
Nickel	Not Available
Chromium	Not Available
Welding Fumes (upon use)	Not Available
Manganese fumes (upon use)	Not Available
Copper Fumes (upon use)	Not Available
Iron Dioxide Fumes (upon use)	Not Available
Silica Welding Fumes (Upon use)	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
Iron	Not Available
Carbon	Not Available
Silicon	Not Available
Manganese	Not Available
Sulfur	Not Available
Phosphorus	Not Available
Copper	Not Available
Nickel	Not Available
Chromium	Not Available
Welding Fumes (upon use)	Not Available
Manganese fumes (upon use)	Not Available
Copper Fumes (upon use)	Not Available
Iron Dioxide Fumes (upon use)	Not Available
Silica Welding Fumes (Upon use)	Not Available

SECTION 15 Regulatory information

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

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (Iron; Carbon; Silicon; Manganese; Sulfur; Phosphorus; Copper; Nickel; Chromium; Manganese fumes (upon use); Copper Fumes (upon use); Iron Dioxide Fumes (upon use); Silica Welding Fumes (Upon use))
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (Iron; Carbon; Silicon; Manganese; Sulfur; Phosphorus; Copper; Nickel; Chromium; Manganese fumes (upon use); Copper Fumes (upon use))
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (Silica Welding Fumes (Upon use))
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	09/12/2023
Initial Date	09/12/2023

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources using available literature references.
The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

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

- 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