



# XT-5183 GMAW Aluminium Mig Wire

## Betaweld Welding Products

Part Number: 2227; 2229

Version No: 1.2.20.11

Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: 29/09/2021

Print Date: 29/09/2021

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

#### Product Identifier

Product name	XT-5183 GMAW Aluminium Mig Wire
Chemical Name	Not Applicable
Synonyms	2227; 2229; ER5183
Other means of identification	Not Available

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

Relevant identified uses	A professional aluminium welding wire manufactured in Italy from European raw materials.
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#### Details of the supplier of the safety data sheet

Registered company name	Betaweld Welding Products
Address	9 Barrel Way Canning Vale WA 6155 Australia
Telephone	(08) 9456 8000
Fax	Not Available
Website	<a href="http://www.betaweld.com.au">www.betaweld.com.au</a>
Email	Not Available

#### Emergency telephone number

Association / Organisation	The Poisons Information Centre
Emergency telephone numbers	13 11 26
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]	Sensitisation (Respiratory) Category 1, Acute Toxicity (Inhalation) Category 3, Carcinogenicity Category 1A, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
Legend:	1. Classification by vendor; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

#### Label elements

XT-5183 GMAW Aluminium Mig Wire

Hazard pictogram(s)	 
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Signal word	<b>Danger</b>
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**Hazard statement(s)**

H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H331	Toxic if inhaled.
H350	May cause cancer.
H412	Harmful to aquatic life with long lasting effects.

**Supplementary statement(s)**

Not Applicable

**Precautionary statement(s) Prevention**

P201	Obtain special instructions before use.
P261	Avoid breathing dust/fumes.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves and protective clothing.
P284	[In case of inadequate ventilation] wear respiratory protection.
P273	Avoid release to the environment.

**Precautionary statement(s) Response**

P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER/doctor/physician/first aider.

**Precautionary statement(s) Storage**

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

**Precautionary statement(s) Disposal**

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

**Substances**

See section below for composition of Mixtures

**Mixtures**

CAS No	%[weight]	Name
7440-21-3	0.25	<u>silicon</u>
7439-89-6	0.4	<u>iron</u>
7440-50-8	0.05	<u>copper</u>
7439-96-5	0.6-1	<u>manganese</u>
7439-95-4	4.3-5.25	<u>magnesium</u>
7440-66-6	0.25	<u>zinc</u>
7440-32-6	0.07-0.15	<u>titanium</u>
7440-47-3	0.05-0.25	<u>chromium</u>
7429-90-5	90-95	<u>aluminium</u>
Not Available	0-60	<u>welding fumes</u>

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CAS No	%[weight]	Name
Not Available	0-60	<u>welding fumes generating barium, chromium, lead or nickel</u>
Not Available	0-60	<u>welding fumes generating beryllium, cadmium</u>
Not Available	0-60	<u>welding fumes generating aluminium, copper, manganese or zinc</u>
<b>Legend:</b> 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**

<b>Eye Contact</b>	<ul style="list-style-type: none"> <li>▶ <b>DO NOT attempt to remove particles attached to or embedded in eye .</b></li> <li>▶ Lay victim down, on stretcher if available and pad <b>BOTH</b> eyes, make sure dressing does not press on the injured eye by placing thick pads under dressing, above and below the eye.</li> <li>▶ Seek urgent medical assistance, or transport to hospital.</li> <li>▶ Particulate bodies from welding spatter may be removed carefully.</li> <li>▶ <b>DO NOT attempt to remove particles attached to or embedded in eye.</b></li> <li>▶ Lay victim down, on stretcher if available and pad <b>BOTH</b> eyes, make sure dressing does not press on the injured eye by placing thick pads under dressing, above and below the eye.</li> <li>▶ Seek urgent medical assistance, or transport to hospital.</li> <li>▶ For "arc eye", i.e. welding flash or UV light burns to the eye:</li> <li>▶ Place eye pads or light clean dressings over both eyes.</li> <li>▶ Seek medical assistance.</li> </ul> <p>For THERMAL burns:</p> <ul style="list-style-type: none"> <li>▶ <b>Do NOT remove contact lens</b></li> <li>▶ Lay victim down, on stretcher if available and pad <b>BOTH</b> eyes, make sure dressing does not press on the injured eye by placing thick pads under dressing, above and below the eye.</li> <li>▶ Seek urgent medical assistance, or transport to hospital.</li> </ul>
<b>Skin Contact</b>	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> <li>▶ Immediately remove all contaminated clothing, including footwear.</li> <li>▶ Flush skin and hair with running water (and soap if available).</li> <li>▶ Seek medical attention in event of irritation.</li> </ul> <p>For thermal burns:</p> <ul style="list-style-type: none"> <li>▶ Decontaminate area around burn.</li> <li>▶ Consider the use of cold packs and topical antibiotics.</li> </ul> <p>For first-degree burns (affecting top layer of skin)</p> <ul style="list-style-type: none"> <li>▶ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides.</li> <li>▶ Use compresses if running water is not available.</li> <li>▶ Cover with sterile non-adhesive bandage or clean cloth.</li> <li>▶ Do NOT apply butter or ointments; this may cause infection.</li> <li>▶ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur.</li> </ul> <p>For second-degree burns (affecting top two layers of skin)</p> <ul style="list-style-type: none"> <li>▶ Cool the burn by immerse in cold running water for 10-15 minutes.</li> <li>▶ Use compresses if running water is not available.</li> <li>▶ Do NOT apply ice as this may lower body temperature and cause further damage.</li> <li>▶ Do NOT break blisters or apply butter or ointments; this may cause infection.</li> <li>▶ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape.</li> </ul> <p>To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort):</p> <ul style="list-style-type: none"> <li>▶ Lay the person flat.</li> <li>▶ Elevate feet about 12 inches.</li> <li>▶ Elevate burn area above heart level, if possible.</li> <li>▶ Cover the person with coat or blanket.</li> <li>▶ Seek medical assistance.</li> </ul> <p>For third-degree burns</p> <p>Seek immediate medical or emergency assistance.</p> <p>In the mean time:</p> <ul style="list-style-type: none"> <li>▶ 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.</li> <li>▶ Separate burned toes and fingers with dry, sterile dressings.</li> <li>▶ Do not soak burn in water or apply ointments or butter; this may cause infection.</li> <li>▶ To prevent shock see above.</li> <li>▶ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway.</li> <li>▶ Have a person with a facial burn sit up.</li> <li>▶ Check pulse and breathing to monitor for shock until emergency help arrives.</li> </ul>
<b>Inhalation</b>	<ul style="list-style-type: none"> <li>▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area.</li> <li>▶ Other measures are usually unnecessary.</li> </ul>

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#### Ingestion

- ▶ Immediately give a glass of water.
- ▶ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

#### Indication of any immediate medical attention and special treatment needed

Magnesium is present in the blood, as a normal constituent, at concentrations between 1.6 to 2.2 meq/L. Some 30% is plasma bound. At serum magnesium levels of 3-4 meq/L, signs of CNS depression, loss of reflexes, muscular tone and power, and bradycardia occur. Cardiac arrest (sometimes fatal) and/or respiratory paralysis can occur at plasma levels of 10-15 meq/L. For acute or short term repeated exposures to magnesium:

- ▶ Symptomatic hypermagnesaemia appears rarely in the absence of intestinal or renal disease.
- ▶ Elevated magnesium levels may cause hypocalcaemia because of decreased parathyroid hormone activity and decreased end-organ responsiveness.
- ▶ Patients with severe hypermagnesemia may develop sudden respiratory arrest and must be watched closely for apnoea.
- ▶ Use fluids, then vasopressors for hypotension. Frequently hypotension responds to calcium administration.
- ▶ Induce emesis or administer lavage if patient presents within 4 hours of ingestion. Use sodium cathartics, with caution, in presence of cardiac or renal failure.
- ▶ Activated charcoal is not useful.
- ▶ Calcium is an antagonist of magnesium action and is an effective antidote when serum levels exceed 5 meq/L and the patient exhibits symptoms. The adult dose of calcium gluconate is 10 ml of a 10% solution over several minutes. [Ellenhorn and Barceloux: Medical Toxicology]

Treat symptomatically.

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.]

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

[Ellenhorn and Barceloux: Medical Toxicology]

For carbon monoxide intoxications:

- ▶ Administer pure oxygen by the best means possible. An oro-nasal mask is usually best. Artificial respiration is necessary wherever breathing is inadequate. Apnoeic patients have often been saved by persistent and efficient artificial ventilation. A patent airway must be carefully maintained. Patients with 40% carboxyhaemoglobin or more and an uncompensated metabolic acidosis (arterial pH less than 7.4) should be managed aggressively with ventilatory support/hyperbaric oxygenation.
- ▶ Gastric aspiration and lavage early in the course of therapy may prevent aspiration pneumonitis and reveal the presence of ingested intoxicants.
- ▶ Avoid stimulant drugs including carbon dioxide. **DO NOT inject methylene blue.**
- ▶ Hypothermia has been employed to reduce the patient's oxygen requirement.
- ▶ Consider antibiotics as prophylaxis against pulmonary infection.
- ▶ A whole blood transfusion may be useful if it can be given early in the treatment program.
- ▶ Infuse sodium bicarbonate and balanced electrolyte solutions if blood analyses indicate a significant metabolic acidosis.
- ▶ Ancillary therapy for brain oedema may be necessary if hypoxia has been severe.
- ▶ Ensure absolute rest in bed for at least 48 hours; in severe poisonings, 2 to 4 weeks in bed may prevent sequelae.
- ▶ Watch for late neurological, psychiatric and cardiac complications. GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products 5th Ed.

#### BIOLOGICAL EXPOSURE INDEX (BEI)

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

Determinant	Sampling time	Index	Comments
Carboxyhaemoglobin in blood	end of shift	3.5% of haemoglobin	B, NS
Carbon monoxide in end-exhaled air	end of shift	20 ppm	B, NS

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

NS: Non-specific determinant; also observed after exposure to other material

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**SECTION 5 Firefighting measures**

**Extinguishing media**

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

**DO NOT USE WATER, CO2 or FOAM.**

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

**Special hazards arising from the substrate or mixture**

<b>Fire Incompatibility</b>	▸ Reacts with acids producing flammable / explosive hydrogen (H2) gas
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**Advice for firefighters**

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

**SECTION 6 Accidental release measures**

**Personal precautions, protective equipment and emergency procedures**

See section 8

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**Environmental precautions**

See section 12

**Methods and material for containment and cleaning up**

<b>Minor Spills</b>	<ul style="list-style-type: none"> <li>▸ Clean up all spills immediately.</li> <li>▸ Avoid breathing dust and contact with skin and eyes.</li> <li>▸ Wear protective clothing, gloves, safety glasses and dust respirator.</li> <li>▸ Use dry clean up procedures and avoid generating dust.</li> <li>▸ Sweep up, shovel up or</li> <li>▸ Vacuum up (consider explosion-proof machines designed to be grounded during storage and use).</li> <li>▸ Place spilled material in clean, dry, sealable, labelled container.</li> </ul>
<b>Major Spills</b>	<ul style="list-style-type: none"> <li>· Do not use compressed air to remove metal dusts from floors, beams or equipment</li> <li>· Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation.</li> <li>· Use non-sparking handling equipment, tools and natural bristle brushes.</li> <li>· Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations</li> <li>· Cover and reseal partially empty containers.</li> <li>· Do not allow chips, fines or dusts to contact water, particularly in enclosed areas.</li> </ul> <p>If molten:</p> <ul style="list-style-type: none"> <li>▸ Contain the flow using dry sand or salt flux as a dam.</li> <li>▸ 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.</li> <li>▸ Allow the spill to cool before remelting scrap.</li> </ul> <p>Moderate hazard.</p> <ul style="list-style-type: none"> <li>▸ <b>CAUTION:</b> Advise personnel in area.</li> <li>▸ Alert Emergency Services and tell them location and nature of hazard.</li> <li>▸ Control personal contact by wearing protective clothing.</li> <li>▸ Prevent, by any means available, spillage from entering drains or water courses.</li> <li>▸ Recover product wherever possible.</li> <li>▸ <b>IF DRY:</b> Use dry clean up procedures and avoid generating dust. Collect residues and place in sealed plastic bags or other containers for disposal. <b>IF WET:</b> Vacuum/shovel up and place in labelled containers for disposal.</li> <li>▸ <b>ALWAYS:</b> Wash area down with large amounts of water and prevent runoff into drains.</li> <li>▸ If contamination of drains or waterways occurs, advise Emergency Services.</li> </ul>

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

**SECTION 7 Handling and storage**

**Precautions for safe handling**

<b>Safe handling</b>	<p>For molten metals:</p> <ul style="list-style-type: none"> <li>· 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.</li> <li>· 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.</li> <li>· Any surfaces that may contact molten metal (e.g. concrete) should be specially coated</li> <li>· 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.</li> </ul> <p>During melting operations, the following minimum guidelines should be observed:</p> <ul style="list-style-type: none"> <li>· 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.</li> <li>· Store materials in dry, heated areas with any cracks or cavities pointed downwards.</li> <li>· 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.</li> </ul> <ul style="list-style-type: none"> <li>▸ Thermit explosions have been reported when aluminium alloys were mixed in furnaces for alloying with lead, bismuth and other metals with low melting temperature. These metals when added as high purity ingots, can seep through cracks in furnace liners and become oxidised. During subsequent melts in the furnace, molten aluminium can contact these metal oxides resulting in a thermit explosion.</li> <li>▸ Avoid all personal contact, including inhalation.</li> <li>▸ Wear protective clothing when risk of exposure occurs.</li> <li>▸ Use in a well-ventilated area.</li> </ul>
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	<ul style="list-style-type: none"> <li>▶ Prevent concentration in hollows and sumps.</li> <li>▶ <b>DO NOT enter confined spaces until atmosphere has been checked.</b></li> <li>▶ <b>DO NOT allow material to contact humans, exposed food or food utensils.</b></li> <li>▶ Avoid contact with incompatible materials.</li> <li>▶ <b>When handling, DO NOT eat, drink or smoke.</b></li> <li>▶ Keep containers securely sealed when not in use.</li> <li>▶ Avoid physical damage to containers.</li> <li>▶ Always wash hands with soap and water after handling.</li> <li>▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use.</li> <li>▶ Use good occupational work practice.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.</li> </ul>
<p><b>Other information</b></p>	<ul style="list-style-type: none"> <li>▶ Store in original containers.</li> <li>▶ Keep containers securely sealed.</li> <li>▶ Store in a cool, dry area protected from environmental extremes.</li> <li>▶ Store away from incompatible materials and foodstuff containers.</li> <li>▶ Protect containers against physical damage and check regularly for leaks.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> </ul> <p>For major quantities:</p> <ul style="list-style-type: none"> <li>▶ Consider storage in bunded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams).</li> <li>▶ Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities.</li> </ul>

**Conditions for safe storage, including any incompatibilities**

<p><b>Suitable container</b></p>	<ul style="list-style-type: none"> <li>▶ Bulk bags: Reinforced bags required for dense materials.</li> </ul>
<p><b>Storage incompatibility</b></p>	<p>The material is described as a very electropositive metal.</p> <p>The activity or electromotive series of metals is a listing of the metals in decreasing order of their reactivity with hydrogen-ion sources such as water and acids. In the reaction with a hydrogen-ion source, the metal is oxidised to a metal ion, and the hydrogen ion is reduced to H<sub>2</sub>. The ordering of the activity series can be related to the standard reduction potential of a metal cation. The more positive the standard reduction potential of the cation, the more difficult it is to oxidise the metal to a hydrated metal cation and the later that metal falls in the series</p> <p>Three notable groups comprise the series</p> <ul style="list-style-type: none"> <li>▶ very electropositive metals</li> <li>▶ electropositive metals</li> <li>▶ electronegative metals</li> </ul> <p>Very electropositive metals have low electronegativities (EN &lt; 1.4) . These cations generally have reduction potentials of -1.6 V or below.</p> <p>They:</p> <ul style="list-style-type: none"> <li>▶ react with water to release hydrogen</li> <li>▶ are good reducing agents</li> <li>▶ are not very good oxidising agents</li> <li>▶ cannot be reduced to the metal, from their ionic forms, in aqueous solution</li> </ul> <p>Very electropositive metals readily ignite in air (burn) forming the oxides. Fires which result can't be extinguished with water (produces flammable H<sub>2</sub>), CCl<sub>4</sub> (an oxidizing agent) or CO<sub>2</sub>. These fires are best extinguished with sand which smothers the flames and does not react</p> <p>Reaction is reduced in the massive form (sheet, rod, or drop), compared with finely divided forms. Active metals:</p> <ul style="list-style-type: none"> <li>▶ can react exothermically with oxidising acids to form noxious gases.</li> <li>▶ catalyse polymerisation and other reactions, particularly when finely divided</li> <li>▶ react with halogenated hydrocarbons (for example, copper dissolves when heated in carbon tetrachloride), sometimes forming explosive compounds.</li> <li>▶ Elemental metals may react with azo/diazo compounds to form explosive products</li> </ul> <ul style="list-style-type: none"> <li>▶ 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.</li> <li>▶ Safe handling is possible in relatively low concentrations of oxygen in an inert gas</li> <li>▶ 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.</li> <li>▶ The reaction residues from various metal syntheses (involving vacuum evaporation and co-deposition with a ligand) are often pyrophoric</li> </ul> <p>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.</p> <p>Many metals may incandesce, react violently, ignite or react explosively upon addition of concentrated nitric acid.</p>

### XT-5183 GMAW Aluminium Mig Wire

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

Inorganic alkaline earth metal derivative.

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 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 H<sub>2</sub>. 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<sup>+</sup> (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/phisci/ch412/activity.htm>

- 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.

Nitric oxide:

- is reactive with alkalis, flammable and combustible materials, organic compounds and solvents, reducing agents, copper and aluminium.



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- ▶ forms nitric / nitrous acid in contact with water and is therefore very corrosive to metals when wet.
- ▶ explosions may occur on contact with ammonia, boron trichloride, carbon disulfide, cyclohexane, fluorine, formaldehyde, nitrobenzene, toluene, incompletely halogenated hydrocarbons, propylene, alcohols, and ozone.
- ▶ Incidents involving interaction of active oxidants and reducing agents, either by design or accident, are usually very energetic and examples of so-called redox reactions.
- ▶ Segregate from alcohol, water.



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

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

**SECTION 8 Exposure controls / personal protection**

**Control parameters**

**Occupational Exposure Limits (OEL)**

**INGREDIENT DATA**

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	silicon	Silicon	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	copper	Copper (fume)	0.2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	copper	Copper, dusts & mists (as Cu)	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	manganese	Manganese, fume (as Mn)	1 mg/m3	3 mg/m3	Not Available	Not Available
Australia Exposure Standards	chromium	Chromium (metal)	0.5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium	Aluminium, pyro powders (as Al)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium	Aluminium (welding fumes) (as Al)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium	Aluminium (metal dust)	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	welding fumes	Welding fumes (not otherwise classified)	5 mg/m3	Not Available	Not Available	Not Available

**Emergency Limits**

Ingredient	TEEL-1	TEEL-2	TEEL-3
silicon	45 mg/m3	100 mg/m3	630 mg/m3
iron	3.2 mg/m3	35 mg/m3	150 mg/m3
copper	3 mg/m3	33 mg/m3	200 mg/m3
manganese	3 mg/m3	5 mg/m3	1,800 mg/m3
magnesium	18 mg/m3	200 mg/m3	1,200 mg/m3
zinc	6 mg/m3	21 mg/m3	120 mg/m3
titanium	30 mg/m3	330 mg/m3	2,000 mg/m3
chromium	1.5 mg/m3	17 mg/m3	99 mg/m3

Ingredient	Original IDLH	Revised IDLH
silicon	Not Available	Not Available
iron	Not Available	Not Available
copper	100 mg/m3	Not Available

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Ingredient	Original IDLH	Revised IDLH
manganese	500 mg/m3	Not Available
magnesium	Not Available	Not Available
zinc	Not Available	Not Available
titanium	Not Available	Not Available
chromium	250 mg/m3	Not Available
aluminium	Not Available	Not Available
welding fumes	Not Available	Not Available
welding fumes generating barium, chromium, lead or nickel	Not Available	Not Available
welding fumes generating beryllium, cadmium	Not Available	Not Available
welding fumes generating aluminium, copper, manganese or zinc	Not Available	Not Available

**Occupational Exposure Banding**

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
welding fumes generating barium, chromium, lead or nickel	E	≤ 0.01 mg/m³
welding fumes generating beryllium, cadmium	E	≤ 0.01 mg/m³
welding fumes generating aluminium, copper, manganese or zinc	E	≤ 0.01 mg/m³
<b>Notes:</b>	<i>Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.</i>	

**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

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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 data 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 aluminium oxide and pyrophoric grades of aluminium:

Twenty seven year experience with aluminium oxide dust (particle size 96% 1,2 um) without adverse effects either systemically or on the lung, and at a calculated concentration equivalent to 2 mg/m3 over an 8-hour shift has lead to the current recommendation of the TLV-TWA.

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

For aluminium oxide:

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

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

**Exposure controls**

<b>Appropriate engineering controls</b>	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>Special ventilation requirements apply for processes which result in the generation of aluminium, copper, fluoride, manganese or zinc fume.</p> <ul style="list-style-type: none"> <li>▶ 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)</li> <li>▶ 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)</li> </ul> <p>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.</p> <div style="display: flex; justify-content: space-between; margin-top: 10px;"> <div style="border: 1px solid black; padding: 2px;">Type of Contaminant:</div> <div style="border: 1px solid black; padding: 2px;">Air Speed:</div> </div>
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welding, brazing fumes (released at relatively low velocity into moderately still air)	0.5-1.0 m/s (100-200 f/min.)
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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.

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.

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.

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- ▶ 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.

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:

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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.

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 beryllium or cadmium fume.

The use of mechanical ventilation by local exhaust systems is required as a minimum in all circumstances (including outdoor work). Personal respirators and special glove boxes afford additional protection

(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.


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<p><b>Personal protection</b></p>	
<p><b>Eye and face protection</b></p>	<ul style="list-style-type: none"> <li>▸ 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.</li> <li>▸ 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]</li> <li>▸ An approved face shield or welding helmet can also have filters for optical radiation protection, and offer additional protection against debris and sparks.</li> <li>▸ UV blocking protective spectacles with side shields or welding goggles are considered primary protection, with the face shield or welding helmet considered secondary protection.</li> <li>▸ 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.</li> <li>▸ 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.</li> </ul> <p>For submerged arc welding use a lens shade which gives just sufficient arc brightness to allow weld pool control.</p>
<p><b>Skin protection</b></p>	<p>See Hand protection below</p>
<p><b>Hands/feet protection</b></p>	<p><b>NOTE:</b></p> <ul style="list-style-type: none"> <li>▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.</li> <li>▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.</li> </ul> <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> <li>· frequency and duration of contact,</li> <li>· chemical resistance of glove material,</li> <li>· glove thickness and</li> <li>· dexterity</li> </ul> <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> <li>· 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.</li> <li>· 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.</li> <li>· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.</li> <li>· Contaminated gloves should be replaced.</li> </ul> <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> <li>· Excellent when breakthrough time &gt; 480 min</li> <li>· Good when breakthrough time &gt; 20 min</li> <li>· Fair when breakthrough time &lt; 20 min</li> <li>· Poor when glove material degrades</li> </ul> <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> <li>· 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.</li> <li>· 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</li> </ul> <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <ul style="list-style-type: none"> <li>▸ Protective gloves eg. Leather gloves or gloves with Leather facing</li> <li>▸ Welding gloves conforming to Standards such as EN 12477:2001, ANSI Z49.1, AS/NZS 2161:2008 produced from leather, rubber, treated cotton, or aluminised</li> <li>▸ These gloves protect against mechanical risk caused by abrasion, blade cut, tear and puncture</li> <li>▸ Other gloves which protect against thermal risks (heat and fire) might also be considered - these comply with different standards to those mentioned above.</li> </ul>

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	<ul style="list-style-type: none"> <li>▶ 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)</li> </ul> <p>Experience indicates that the following polymers are suitable as glove materials for protection against undissolved, dry solids, where abrasive particles are not present.</p> <ul style="list-style-type: none"> <li>▶ polychloroprene.</li> <li>▶ nitrile rubber.</li> <li>▶ butyl rubber.</li> <li>▶ fluorocautchouc.</li> <li>▶ polyvinyl chloride.</li> </ul> <p>Gloves should be examined for wear and/ or degradation constantly.</p>
<b>Body protection</b>	See Other protection below
<b>Other protection</b>	<p>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.</p> <ul style="list-style-type: none"> <li>▶ Overalls.</li> <li>▶ P.V.C apron.</li> <li>▶ Barrier cream.</li> <li>▶ Skin cleansing cream.</li> <li>▶ Eye wash unit.</li> </ul>

**Respiratory protection**

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

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

\* - Negative pressure demand \*\* - Continuous flow

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

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

**SECTION 9 Physical and chemical properties**

**Information on basic physical and chemical properties**

<b>Appearance</b>	Silver		
<b>Physical state</b>	Solid	<b>Relative density (Water = 1)</b>	Not Available

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

**SECTION 10 Stability and reactivity**

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

**SECTION 11 Toxicological information**

**Information on toxicological effects**

<b>Inhaled</b>	<p>The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.</p> <p>Fumes evolved during welding operations may be irritating to the upper-respiratory tract and may be harmful if inhaled.</p> <p>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.</p> <p>Not normally a hazard due to non-volatile nature of product</p> <p>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</p>
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	<p>disturbances may remain</p> <p>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.</p> <p>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.</p> <p>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</p> <p>At low levels carbon monoxide may cause poor concentration, memory and vision problems, vertigo, muscular weakness and loss of muscle coordination, rapid and strenuous 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.</p> <p>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.</p> <p>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. Inhalation of dusts, generated by the material, during the course of normal handling, may be harmful.</p>
<p><b>Ingestion</b></p>	<p>Magnesium salts are generally absorbed so slowly that oral administration causes few toxic effects with purging being the most significant. If evacuation fails due to bowel obstruction or atony, mucosal irritation and absorption may result.</p> <p>Side effects of magnesium salts include upset stomach, dry mouth, dry nose and dry throat, drowsiness, nausea, heartburn and thickening of the mucous in the throat and nose.</p> <p>Systemically the magnesium ion produces electrolyte imbalance, central nervous system depression, neurological and cardiac involvement, reflex abolition and death from respiratory paralysis. These effects are rare in the absence of intestinal or renal disorders.</p> <p>Early signs and symptoms of magnesium intoxication include nausea, vomiting, malaise and confusion. Deep tendon reflexes may be diminished. central nervous system depression may progress to coma and paralysis of the release of acetylcholine at myoneuronal junctions. Central nervous system depression may be compounded by depressed function of the respiratory musculature. Hypotension may also ensue as a result of peripheral vasodilation and/ or decreased cardiac output secondary to conduction defects. Bradycardia is common, leading to eventual arrest in diastole.</p> <p>Acute toxic responses to aluminium are confined to the more soluble forms.</p> <p>The material has <b>NOT</b> been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.</p> <p>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.</p> <p>Accidental ingestion of the material may be damaging to the health of the individual.</p>
<p><b>Skin Contact</b></p>	<p>The material is not thought to produce adverse health effects or skin irritation following contact (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting.</p> <p>Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus.</p> <p>Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles.</p> <p>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.</p> <p>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.</p> <p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p>

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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.

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.

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**

Although the material is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may cause transient discomfort characterised by tearing or conjunctival redness (as with windburn). Slight abrasive damage may also result. The material may produce foreign body irritation in certain individuals.

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.

**Chronic**

Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.

Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.

Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers

Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.

Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.

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

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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 Al<sub>2</sub>O<sub>3</sub> powder was not fibrogenic in rats, guinea pigs, or hamsters when inhaled for 6 to 12 months and sacrificed at periods up to 12 months following the last exposure.

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

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

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

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

Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic.

Because aluminium competes with calcium for absorption, increased amounts of dietary aluminium may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies, aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels.

Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseous tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory. These salts' estrogen-like effects have led to their classification as metalloestrogen. Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.

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

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

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

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A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 mg/l aluminium compared with communities where the aluminium level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminium exposure to brain disease. Aluminium concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramid-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations.

Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of "tau" a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminium hyperphosphorylates tau in vitro. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade. Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA). Aluminium enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminium include baking powder, antacids and aluminium products used for general food preparation and storage (over 12 months, aluminium levels in soft drink packed in aluminium cans rose from 0.05 to 0.9 mg/l). [Walton, J and Bryson-Taylor, D. - *Chemistry in Australia*, August 1995]

A case of chronic abuse of magnesium citrate ( a mild purgative), by a 62 year-old woman, has been reported. Symptoms of abuse included lethargy and severe refractory hypotension. Pathology revealed extreme hypermagnesaemia [6.25 mmol per litre]. She also was found to have a perforated duodenal ulcer. She died after peritoneal dialysis (which reduced serum-magnesium and reduced hypotension).

A patient with normal kidney function developed symptomatic hypermagnesaemia with respiratory arrest and bradycardia after receiving 90 grams of magnesium sulfate over 18 hours.

When magnesium sulfate was given to pregnant rats, a sharp reduction of both the number and the weight of the offspring was observed.

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 O<sub>2</sub> consumption, and a lower O<sub>2</sub> tension in the blood than adults, a compromised O<sub>2</sub> 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.

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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.

<b>XT-5183 GMAW Aluminium Mig Wire</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
<b>silicon</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >5000 mg/kg <sup>[1]</sup> Oral(Rat) LD50; >5000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
<b>iron</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral(Human) LD50; 200 mg/kg <sup>[2]</sup>	Not Available
<b>copper</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup> Inhalation(Rat) LC50; 0.733 mg/l4h <sup>[1]</sup> Oral(Mouse) LD50; 0.7 mg/kg <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
<b>manganese</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Inhalation(Rat) LC50; >5.14 mg/l4h <sup>[1]</sup> Oral(Rat) LD50; >2000 mg/kg <sup>[1]</sup>	Eye (rabbit): 500 mg/24h - mild Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin (rabbit): 500 mg/24h - mild Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
<b>magnesium</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup> Inhalation(Rat) LC50; >2.1 mg/L4h <sup>[1]</sup> Oral(Rat) LD50; >2000 mg/kg <sup>[1]</sup>	Not Available
<b>zinc</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 1130 mg/kg <sup>[2]</sup> Oral(Rat) LD50; >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
<b>titanium</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral(Rat) LD50; >2000 mg/kg <sup>[1]</sup>	Not Available
<b>chromium</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Inhalation(Rat) LC50; >5.41 mg/l4h <sup>[1]</sup> Oral(Rat) LD50; >5000 mg/kg <sup>[1]</sup>	Not Available

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aluminium	<b>TOXICITY</b>	<b>IRRITATION</b>
	Inhalation(Rat) LC50; >2.3 mg/4h <sup>[1]</sup> Oral(Rat) LD50; >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
welding fumes	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
welding fumes generating barium, chromium, lead or nickel	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
welding fumes generating beryllium, cadmium	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
welding fumes generating aluminium, copper, manganese or zinc	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
<b>Legend:</b>	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	

<p><b>XT-5183 GMAW Aluminium Mig Wire</b></p>	<p>The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p> <p>For aluminium compounds:</p> <p>Aluminium present in food and drinking water is poorly absorbed through the gastrointestinal tract. The bioavailability of aluminium is dependent on the form in which it is ingested and the presence of dietary constituents with which the metal cation can complex. Ligands in food can have a marked effect on absorption of aluminium, as they can either enhance uptake by forming absorbable (usually water soluble) complexes (e.g., with carboxylic acids such as citric and lactic), or reduce it by forming insoluble compounds (e.g., with phosphate or dissolved silicate).</p> <p>Considering the available human and animal data it is likely that the oral absorption of aluminium can vary 10-fold based on chemical form alone. Although bioavailability appears to generally parallel water solubility, insufficient data are available to directly extrapolate from solubility in water to bioavailability.</p> <p>For oral intake from food, the European Food Safety Authority (EFSA) has derived a tolerable weekly intake (TWI) of 1 milligram (mg) of aluminium per kilogram of bodyweight. In its health assessment, the EFSA states a medium bioavailability of 0.1 % for all aluminium compounds which are ingested with food. This corresponds to a systemically available tolerable daily dose of 0.143 microgrammes (µg) per kilogramme (kg) of body weight. This means that for an adult weighing 60 kg, a systemically available dose of 8.6 µg per day is considered safe.</p> <p>Based on a neuro-developmental toxicity study of aluminium citrate administered via drinking water to rats, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) established a Provisional Tolerable Weekly Intake (PTWI) of 2 mg/kg bw (expressed as aluminium) for all aluminium compounds in food, including food additives. The Committee on Toxicity of chemicals in food, consumer products and the environment (COT) considers that the derivation of this PTWI was sound and that it should be used in assessing potential risks from dietary exposure to aluminium.</p> <p>The Federal Institute for Risk Assessment (BfR) of Germany has assessed the estimated aluminium absorption from antiperspirants. For this purpose, the data, derived from experimental studies, on dermal absorption of aluminium from antiperspirants for healthy and damaged skin was used as a basis. At about 10.5 µg, the calculated systemic intake values for healthy skin are above the 8.6 µg per day that are considered safe for an adult weighing 60 kg. If aluminium-containing antiperspirants are used on a daily basis, the tolerable weekly intake determined by the EFSA is therefore exceeded. The values for damaged skin, for example injuries from shaving, are many times higher. This means that in case of daily use of an aluminium-containing antiperspirant alone, the TWI may be completely exhausted. In addition, further aluminium absorption sources such as food, cooking utensils and other cosmetic products must be taken into account</p> <p><b>Systemic toxicity after repeated exposure</b></p> <p>No studies were located regarding dermal effects in animals following intermediate or chronic-duration dermal exposure to various forms of aluminium.</p> <p>When orally administered to rats, aluminium compounds (including aluminium nitrate, aluminium sulfate and potassium aluminium sulfate) have produced various effects, including decreased gain in body weight and mild histopathological changes in the spleen, kidney and liver of rats (104 mg Al/kg bw/day) and dogs (88-93 mg Al/kg bw/day) during subchronic oral exposure. Effects on nerve cells, testes, bone and stomach have been reported at higher doses. Severity of effects increased with dose.</p> <p>The main toxic effects of aluminium that have been observed in experimental animals are neurotoxicity and nephrotoxicity. Neurotoxicity has also been described in patients dialysed with water containing high concentrations of aluminium, but epidemiological data on possible adverse effects in humans at lower exposures are inconsistent</p> <p><b>Reproductive and developmental toxicity:</b></p> <p>Studies of reproductive toxicity in male mice (intraperitoneal or subcutaneous administration of aluminium nitrate or chloride) and</p>
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rabbits (administration of aluminium chloride by gavage) have demonstrated the ability of aluminium to cause testicular toxicity, decreased sperm quality in mice and rabbits and reduced fertility in mice. No reproductive toxicity was seen in females given aluminium nitrate by gavage or dissolved in drinking water. Multi-generation reproductive studies in which aluminium sulfate and aluminium ammonium sulfate were administered to rats in drinking water, showed no evidence of reproductive toxicity. High doses of aluminium compounds given by gavage have induced signs of embryotoxicity in mice and rats in particular, reduced fetal body weight or pup weight at birth and delayed ossification. Developmental toxicity studies in which aluminium chloride was administered by gavage to pregnant rats showed evidence of foetotoxicity, but it was unclear whether the findings were secondary to maternal toxicity. A twelve-month neuro-development with aluminium citrate administered via the drinking water to Sprague-Dawley rats, was conducted according to Good Laboratory Practice (GLP). Aluminium citrate was selected for the study since it is the most soluble and bioavailable aluminium salt. Pregnant rats were exposed to aluminium citrate from gestational day 6 through lactation, and then the offspring were exposed post-weaning until postnatal day 364. An extensive functional observational battery of tests was performed at various times. Evidence of aluminium toxicity was demonstrated in the high (300 mg/kg bw/day of aluminium) and to a lesser extent, the mid-dose groups (100 mg/kg bw/day of aluminium). In the high-dose group, the main effect was renal damage, resulting in high mortality in the male offspring. No major neurological pathology or neurobehavioural effects were observed, other than in the neuromuscular subdomain (reduced grip strength and increased foot splay). Thus, the lowest observed adverse effect level (LOAEL) was 100 mg/kg bw/day and the no observed adverse effect level (NOAEL) was 30 mg/kg bw/day. Bioavailability of aluminium chloride, sulfate and nitrate and aluminium hydroxide was much lower than that of aluminium citrate. This study was used by JECFA as key study to derive the PTWI.

#### Genotoxicity

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

#### Carcinogenicity

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

#### Neurodegenerative diseases.

Following the observation that high levels of aluminium in dialysis fluid could cause a form of dementia in dialysis patients, a number of studies were carried out to determine if aluminium could cause dementia or cognitive impairment as a consequence of environmental exposure over long periods. Aluminium was identified, along with other elements, in the amyloid plaques that are one of the diagnostic lesions in the brain for Alzheimer disease, a common form of senile and pre-senile dementia. Some of the epidemiology studies suggest the possibility of an association of Alzheimer disease with aluminium in water, but other studies do not confirm this association. All studies lack information on ingestion of aluminium from food and how concentrations of aluminium in food affect the association between aluminium in water and Alzheimer disease." There are suggestions that persons with some genetic variants may absorb more aluminium than others, but there is a need for more analytical research to determine whether aluminium from various sources has a significant causal association with Alzheimer disease and other neurodegenerative diseases. Aluminium is a neurotoxicant in experimental animals. However, most of the animal studies performed have several limitations and therefore cannot be used for quantitative risk assessment.

#### Contact sensitivity:

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

Aluminium acts not only as an adjuvant, stimulating the immune system either to fend off infections or to tolerate antigens, it also acts as a sensitiser causing contact allergy and allergic contact dermatitis. In general, metal allergies are very common and aluminium is considered to be a weak allergen. A metal must be ionised to be able to act as a contact allergen, then it has to undergo haptensation to be immunogenic and to initiate an immune response. Once inside the skin, the metal ions must bind to proteins to become immunologically reactive. The most important routes of exposure and sensitisation to aluminium are through aluminium-containing vaccines. One Swedish study showed a statistically significant association between contact allergy to aluminium and persistent itching nodules in children treated with allergen-specific immunotherapy (ASIT). Nodules were overrepresented in patients with contact allergy to aluminium.

Other routes of sensitisation reported in the literature are the prolonged use of aluminium-containing antiperspirants, topical medication, and tattooing of the skin with aluminium-containing pigments. Most of the patients experienced eczematous reactions whereas tattooing caused granulomas. Even though aluminium is used extensively in industry, only a low number of cases of occupational skin sensitisation to aluminium have been reported. Systemic allergic contact dermatitis in the form of flare-up reactions after re-exposure to aluminium has been documented: pruritic nodules at present and previous injection sites, eczema at the site of vaccination as well as at typically atopic localisations after vaccination with aluminium-containing vaccines and/or patch testing with aluminium, and also after use of aluminium-containing toothpaste.

**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.

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<p><b>SILICON</b></p>	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.</p> <p>Intraperitoneal injection of silicon produced only minor local trauma and foreign body reaction. Parenterally administered elemental silica is considered biologically inert.</p> <p>Dogs and rats fed 800 mg silicon/kg/day (as the dioxide) for 1 month showed no clinical signs or histological changes. The compound was largely eliminated in the faeces.</p> <p>Normal human cerebral cortex tissue contains about 3.8 ug/g silicon</p>
<p><b>COPPER</b></p>	<p>WARNING: Inhalation of high concentrations of copper fume may cause "metal fume fever", an acute industrial disease of short duration. Symptoms are tiredness, influenza like respiratory tract irritation with fever.</p> <p>for copper and its compounds (typically copper chloride):</p> <p><b>Acute toxicity:</b> 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.</p> <p><b>Repeat dose toxicity:</b> 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.</p> <p><b>Genotoxicity:</b> 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.</p> <p><b>Carcinogenicity:</b> there was insufficient information to evaluate the carcinogenic activity of copper monochloride.</p> <p><b>Reproductive and developmental toxicity:</b> 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).</p>
<p><b>CHROMIUM</b></p>	<p>Gastrointestinal tumours, lymphoma, musculoskeletal tumours and tumours at site of application recorded.</p> <p>For chrome(III) and other valence states (except hexavalent):</p> <p>For inhalation exposure, all trivalent and other chromium compounds are treated as particulates, not gases.</p> <p>The mechanisms of chromium toxicity are very complex, and although many studies on chromium are available, there is a great deal of uncertainty about how chromium exerts its toxic influence. Much more is known about the mechanisms of hexavalent chromium toxicity than trivalent chromium toxicity. There is an abundance of information available on the carcinogenic potential of chromium compounds and on the genotoxicity and mutagenicity of chromium compounds in experimental systems. The consensus from various reviews and agencies is that evidence of carcinogenicity of elemental, divalent, or trivalent chromium compounds is lacking. Epidemiological studies of workers in a number of industries (chromate production, chromate pigment production and use, and chrome plating) conclude that while occupational exposure to hexavalent chromium compounds is associated with an increased risk of respiratory system cancers (primarily bronchogenic and nasal), results from occupational exposure studies to mixtures that were mainly elemental and trivalent (ferrochromium alloy worker) were inconclusive. Studies in leather tanners, who were exposed to trivalent chromium were consistently negative. In addition to the lack of direct evidence of carcinogenicity of trivalent or elemental chromium and its compounds, the genotoxic evidence is overwhelmingly negative. The lesser potency of trivalent chromium relative to hexavalent chromium is likely related to the higher redox potential of hexavalent chromium and its greater ability to enter cells.</p> <p>The general inability of trivalent chromium to traverse membranes and thus be absorbed or reach peripheral tissue in significant amounts is generally accepted as a probable explanation for the overall absence of systemic trivalent chromium toxicity. Elemental and divalent forms of chromium are not able to traverse membranes readily either. This is not to say that elemental,</p>



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divalent, or trivalent chromium compounds cannot traverse membranes and reach peripheral tissue, the mechanism of absorption is simply less efficient in comparison to absorption of hexavalent chromium compounds. Hexavalent chromium compounds exist as tetrahedral chromate anions, resembling the forms of other natural anions like sulfate and phosphate which are permeable across nonselective membranes. Trivalent chromium forms octahedral complexes which cannot easily enter through these channels, instead being absorbed via passive diffusion and phagocytosis. Although trivalent chromium is less well absorbed than hexavalent chromium, workers exposed to trivalent compounds have had detectable levels of chromium in the urine at the end of a workday. Absorbed chromium is widely distributed throughout the body via the bloodstream, and can reach the foetus. Although there is ample in vivo evidence that hexavalent chromium is efficiently reduced to trivalent chromium in the gastrointestinal tract and can be reduced to the trivalent form by ascorbate and glutathione in the lungs, there is no evidence that trivalent chromium is converted to hexavalent chromium in biological systems. In general, trivalent chromium compounds are cleared rapidly from the blood and more slowly from the tissues. Although not fully characterized, the biologically active trivalent chromium molecule appears to be chromodulin, also referred to as (GTF). Chromodulin is an oligopeptide complex containing four chromic ions. Chromodulin may facilitate interactions of insulin with its receptor site, influencing protein, glucose, and lipid metabolism. Inorganic trivalent chromium compounds, which do not appear to have insulin-potentiating properties, are capable of being converted into biologically active forms by humans and animals

Chromium can be a potent sensitiser in a small minority of humans, both from dermal and inhalation exposures.

The most sensitive endpoint identified in animal studies of acute exposure to trivalent chromium appears to involve the respiratory system. Specifically, acute exposure to trivalent chromium is associated with impaired lung function and lung damage. Based on what is known about absorption of chromium in the human body, its potential mechanism of action in cells, and occupational data indicating that valence states other than hexavalent exhibit a relative lack of toxicity the toxicity of elemental and divalent chromium compounds is expected to be similar to or less than common trivalent forms.

The substance is classified by IARC as Group 3:

**NOT** classifiable as to its carcinogenicity to humans.

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

Tenth Annual Report on Carcinogens: Substance known to be Carcinogenic

[National Toxicology Program: U.S. Dep. of Health and Human Services 2002]

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 is 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 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/m<sup>3</sup> and to iron ranged from 0.04-16.29 mg/m<sup>3</sup> in eight Canadian welding companies. Types of welding identified were mostly (90%) MIG mild steel, MIG stainless steel, and TIG aluminium. 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

**XT-5183 GMAW Aluminium  
Mig Wire & WELDING  
FUMES & WELDING  
FUMES GENERATING  
BARIUM, CHROMIUM,  
LEAD OR NICKEL &  
WELDING FUMES  
GENERATING BERYLLIUM,  
CADMIUM & WELDING  
FUMES GENERATING  
ALUMINIUM, COPPER,  
MANGANESE OR ZINC**

XT-5183 GMAW Aluminium Mig Wire

	<p>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</p> <p>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</p> <p>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</p>
<b>SILICON &amp; ZINC &amp; TITANIUM &amp; CHROMIUM &amp; ALUMINIUM</b>	No significant acute toxicological data identified in literature search.
<b>SILICON &amp; MANGANESE</b>	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
<b>MANGANESE &amp; ZINC</b>	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
<b>WELDING FUMES &amp; WELDING FUMES GENERATING BARIUM, CHROMIUM, LEAD OR NICKEL &amp; WELDING FUMES GENERATING BERYLLIUM, CADMIUM &amp; WELDING FUMES GENERATING ALUMINIUM, COPPER, MANGANESE OR ZINC</b>	<p><b>WARNING:</b> This substance has been classified by the IARC as Group 1: <b>CARCINOGENIC TO HUMANS.</b></p> <p>Not available. Refer to individual constituents.</p>

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**

XT-5183 GMAW Aluminium Mig Wire	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
silicon	Endpoint	Test Duration (hr)	Species	Value	Source
	EC10(ECx)	1.28h	Algae or other aquatic plants	>=66<=88mg/l	2
	EC50	72h	Algae or other aquatic plants	~250mg/l	2
iron	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	48h	Algae or other aquatic plants	0.1-4mg/l	4
	EC50	72h	Algae or other aquatic plants	18mg/l	2
	LC50	96h	Fish	0.05mg/l	2
EC50	48h	Crustacea	>100mg/l	2	

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copper	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50(ECx)	24h	Algae or other aquatic plants	<0.001mg/L	4
	EC50	72h	Algae or other aquatic plants	0.011-0.017mg/L	4
	LC50	96h	Fish	~0.005mg/L	4
	EC50	48h	Crustacea	<0.001mg/L	4
EC50	96h	Algae or other aquatic plants	0.03-0.058mg/l	4	
manganese	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	NOEC(ECx)	504h	Algae or other aquatic plants	0.05-3.7mg/l	4
	EC50	72h	Algae or other aquatic plants	2.8mg/l	2
	LC50	96h	Fish	>3.6mg/l	2
EC50	48h	Crustacea	>1.6mg/l	2	
magnesium	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	NOEC(ECx)	72h	Algae or other aquatic plants	>=12mg/l	2
	EC50	72h	Algae or other aquatic plants	>12mg/l	2
	LC50	96h	Fish	541mg/l	2
	EC50	48h	Crustacea	344mg/l	2
EC50	96h	Algae or other aquatic plants	222.37mg/l	2	
zinc	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50(ECx)	72h	Algae or other aquatic plants	0.005mg/l	4
	EC50	72h	Algae or other aquatic plants	0.005mg/l	4
	LC50	96h	Fish	0.16mg/L	4
	EC50	48h	Crustacea	1.4mg/l	2
EC50	96h	Algae or other aquatic plants	0.264-0.881mg/l	4	
titanium	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	NOEC(ECx)	48h	Crustacea	<=1mg/l	2
	EC50	72h	Algae or other aquatic plants	13mg/l	2
	LC50	96h	Fish	>100mg/l	2
EC50	48h	Crustacea	>100mg/l	2	
chromium	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50(ECx)	48h	Crustacea	<0.001mg/l	2
	EC50	72h	Algae or other aquatic plants	0.026-0.208mg/L	4
	LC50	96h	Fish	0.106mg/L	4
	EC50	48h	Crustacea	<0.001mg/l	2
EC50	96h	Algae or other aquatic plants	36mg/L	4	
aluminium	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	NOEC(ECx)	48h	Crustacea	>100mg/l	1
	EC50	72h	Algae or other aquatic plants	0.2mg/l	2
	LC50	96h	Fish	0.078-0.108mg/l	2
	EC50	48h	Crustacea	1.5mg/l	2
EC50	96h	Algae or other aquatic plants	0.024mg/l	2	
welding fumes	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	Not Available	Not Available	Not Available	Not Available	Not Available
welding fumes generating barium, chromium, lead or nickel	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	Not Available	Not Available	Not Available	Not Available	Not Available

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welding fumes generating beryllium, cadmium	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

welding fumes generating aluminium, copper, manganese or zinc	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

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

Harmful to aquatic organisms.

Metal-containing inorganic substances generally have negligible vapour pressure and are not expected to partition to air. Once released to surface waters and moist soils their fate depends on solubility and dissociation in water. Environmental processes (such as oxidation and the presence of acids or bases) may transform insoluble metals to more soluble ionic forms. Microbiological processes may also transform insoluble metals to more soluble forms. Such ionic species may bind to dissolved ligands or sorb to solid particles in aquatic or aqueous media. 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.

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. Environmental processes may also be important in changing solubilities.

Even though many metals show few toxic effects at physiological pHs, transformation may introduce new or magnified effects.

A metal ion is considered infinitely persistent because it cannot degrade further.

The current state of science does not allow for an unambiguous interpretation of various measures of bioaccumulation.

The counter-ion may also create health and environmental concerns once isolated from the metal. Under normal physiological conditions the counter-ion may be essentially insoluble and may not be bioavailable.

Environmental processes may enhance bioavailability.

for magnesium compounds in general:

Fish LC50: 100-400 mg/l

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 adsorb. 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<sub>3</sub><sup>2-</sup>), and the concentration of manganese is limited by the relatively low solubility (65 mg/L) of MnCO<sub>3</sub>. 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 10,000 - 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

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Turbellarian worms (EC0): *Polycelis nigra* 660 mg/l (interference threshold); *microregma* 31 mg/l

For aluminium and its compounds and salts:

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

#### Environmental fate:

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

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

As an element, aluminum cannot be degraded in the environment, but may undergo various precipitation or ligand exchange reactions. Aluminum in compounds has only one oxidation state (+3), and would not undergo oxidation-reduction reactions under environmental conditions. Aluminum can be complexed by various ligands present in the environment (e.g., fulvic and humic acids). The solubility of aluminum in the environment will depend on the ligands present and the pH. The trivalent aluminum ion is surrounded by six water molecules in solution. The hydrated aluminum ion,  $[Al(H_2O)_6]^{3+}$ , undergoes hydrolysis, in which a stepwise deprotonation of the coordinated water ligands forms bound hydroxide ligands (e.g.,  $[Al(H_2O)_5(OH)]^{2+}$ ,  $[Al(H_2O)_4(OH)_2]^+$ ). The speciation of aluminum in water is pH dependent. The hydrated trivalent aluminum ion is the predominant form at pH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are  $Al(OH)_2^+$  and  $Al(OH)_3$ , while the solid  $Al(OH)_3$  is most prevalent between pH 5.2 and 8.8. The soluble species  $Al(OH)_4^-$  is the predominant species above pH 9, and is the only species present above pH 10. Polymeric aluminum hydroxides appear between pH 4.7 and 10.5, and increase in size until they are transformed into colloidal particles of amorphous  $Al(OH)_3$ , which crystallise to gibbsite in acid waters. Polymerisation is affected by the presence of dissolved silica; when enough silica is present, aluminum is precipitated as poorly crystallised clay mineral species.

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

Monomeric aluminum compounds, typified by aluminum fluoride, chloride, and sulfate, are considered reactive or labile compounds, whereas polymeric aluminum species react much more slowly in the environment. Aluminum has a stronger attraction for fluoride in an acidic environment compared to other inorganic ligand. The adsorption of aluminum onto clay surfaces can be a significant factor in controlling aluminum mobility in the environment, and these adsorption reactions, measured in one study at pH 3.0-4.1, have been observed to be very rapid. However, clays may act either as a sink or a source for soluble aluminum depending on the degree of aluminum saturation on the clay surface.

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

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

Aluminum concentrations in rainbow trout from an alum-treated lake, an untreated lake, and a hatchery were highest in gill tissue and lowest in muscle. Aluminum residue analyses in brook trout have shown that whole-body aluminum content decreases as the fish advance from larvae to juveniles. These results imply that the aging larvae begin to decrease their rate of aluminum uptake, to eliminate aluminum at a rate that exceeds uptake, or to maintain approximately the same amount of aluminum while the body mass increases. The decline in whole-body aluminum residues in juvenile brook trout may be related to growth and dilution by edible muscle tissue that accumulated less aluminum than did the other tissues.

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

Bioconcentration of aluminum has also been reported for several aquatic invertebrate species. BCF values ranging from 0.13 to 0.5 in the whole-body were reported for the snail. Bioconcentration of aluminum has also been reported for aquatic insects.

#### Ecotoxicity:

##### Freshwater species pH >6.5

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

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

Crustaceans LC50 (48 h): 1 sp 2.3-36.9 mg/L; Chronic NOEC (7-28 d) 3 spp, 0.136-1.72 mg/L

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

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

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

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

Alga: 1 sp NOEC growth 2.0 mg/L

Among freshwater aquatic plants, single-celled plants are generally the most sensitive to aluminium. Fish are generally more sensitive to aluminium than aquatic invertebrates. Aluminium is a gill toxicant to fish, causing both ionoregulatory and respiratory effects.

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

Drinking Water Standards:

aluminium: 200 ug/l (UK max.)

**XT-5183 GMAW Aluminium Mig Wire**

200 ug/l (WHO guideline)  
chloride: 400 mg/l (UK max.)  
250 mg/l (WHO guideline)  
fluoride: 1.5 mg/l (UK max.)  
1.5 mg/l (WHO guideline)  
nitrate: 50 mg/l (UK max.)  
50 mg/l (WHO guideline)  
sulfate: 250 mg/l (UK max.)  
Soil Guideline: none available.  
Air Quality Standards: none available.  
For carbon monoxide:

**Environmental fate:**

Although carbon monoxide is not considered a greenhouse gas, it is a precursor to greenhouse gases. Carbon monoxide elevates the concentrations of methane (a greenhouse gas) and ozone in the atmosphere. It eventually oxidises into carbon dioxide. Greenhouse gases are linked to global warming. Very high levels of carbon monoxide will cause the same problems to birds and animals that are experienced by people, although these levels are very unlikely to be encountered in the environment except during extreme events like bushfires.

**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**

<b>Product / Packaging disposal</b>	<ul style="list-style-type: none"> <li>▶ Containers may still present a chemical hazard/ danger when empty.</li> <li>▶ Return to supplier for reuse/ recycling if possible.</li> </ul> <p>Otherwise:</p> <ul style="list-style-type: none"> <li>▶ 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.</li> <li>▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product.</li> <li>▶ <b>DO NOT allow wash water from cleaning or process equipment to enter drains.</b></li> <li>▶ It may be necessary to collect all wash water for treatment before disposal.</li> <li>▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.</li> <li>▶ Where in doubt contact the responsible authority.</li> <li>▶ Recycle wherever possible or consult manufacturer for recycling options.</li> <li>▶ Consult State Land Waste Management Authority for disposal.</li> <li>▶ Bury residue in an authorised landfill.</li> <li>▶ Recycle containers if possible, or dispose of in an authorised landfill.</li> </ul>
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**SECTION 14 Transport information**

**Labels Required**

<b>Marine Pollutant</b>	NO
<b>HAZCHEM</b>	Not Applicable

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

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

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

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

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Not Applicable

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

Product name	Group
silicon	Not Available
iron	Not Available
copper	Not Available
manganese	Not Available
magnesium	Not Available
zinc	Not Available
titanium	Not Available
chromium	Not Available
aluminium	Not Available
welding fumes	Not Available
welding fumes generating barium, chromium, lead or nickel	Not Available
welding fumes generating beryllium, cadmium	Not Available
welding fumes generating aluminium, copper, manganese or zinc	Not Available

**Transport in bulk in accordance with the ICG Code**

Product name	Ship Type
silicon	Not Available
iron	Not Available
copper	Not Available
manganese	Not Available
magnesium	Not Available
zinc	Not Available
titanium	Not Available
chromium	Not Available
aluminium	Not Available
welding fumes	Not Available
welding fumes generating barium, chromium, lead or nickel	Not Available
welding fumes generating beryllium, cadmium	Not Available
welding fumes generating aluminium, copper, manganese or zinc	Not Available

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

Australian Inventory of Industrial Chemicals (AIIC)

**iron is found on the following regulatory lists**

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2

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

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

Australian Inventory of Industrial Chemicals (AIIC)

**copper is found on the following regulatory lists**

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4  
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6  
 Australian Inventory of Industrial Chemicals (AIIC)

**manganese is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

**magnesium is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

**zinc is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

**titanium is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

**chromium is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

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

**aluminium is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

**welding fumes is found on the following regulatory lists**

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

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

**welding fumes generating barium, chromium, lead or nickel is found on the following regulatory lists**

Not Applicable

**welding fumes generating beryllium, cadmium is found on the following regulatory lists**

Not Applicable

**welding fumes generating aluminium, copper, manganese or zinc is found on the following regulatory lists**

Not Applicable

**National Inventory Status**

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (silicon; iron; copper; manganese; magnesium; zinc; titanium; chromium; aluminium)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (silicon; iron; copper; manganese; magnesium; zinc; titanium; chromium; aluminium)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes



## XT-5183 GMAW Aluminium Mig Wire

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

### SECTION 16 Other information

<b>Revision Date</b>	29/09/2021
<b>Initial Date</b>	29/09/2021

### Other information

#### Ingredients with multiple cas numbers

Name	CAS No
silicon	7440-21-3, 152284-21-4, 157383-37-4, 160371-18-6, 17375-03-0, 71536-23-7, 72516-01-9, 72516-02-0, 72516-03-1, 90337-93-2
iron	7439-89-6, 8053-60-9, 129048-51-7, 161135-39-3, 1867181-06-3, 190454-13-8, 195161-83-2, 199281-22-6, 39344-71-3, 443783-52-6, 675141-17-0, 70884-35-4, 73135-38-3, 8011-79-8, 14067-02-8, 8048-10-0, 12597-68-1
copper	7440-50-8, 133353-46-5, 133353-47-6, 195161-80-9, 65555-90-0, 72514-83-1, 1441640-38-5, 1993435-25-8, 2056901-56-3
chromium	7440-47-3, 188785-87-7, 195161-82-1
aluminium	7429-90-5, 91728-14-2

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

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