



XT-110S GMAW Low Alloy CuZn Coated MIG Wire

Betaweld Welding Products

Part Number: 2213; 2214

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-110S GMAW Low Alloy CuZn Coated MIG Wire
Chemical Name	Not Applicable
Synonyms	2213; 2214; ER110S-1
Other means of identification	Not Available

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

Relevant identified uses	A premium coated low alloy mig wire designed for welding of high strength steels with a tensile strength greater than 800MPa..
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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	www.betaweld.com.au
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, Specific Target Organ Toxicity - Repeated Exposure Category 2, Acute Toxicity (Inhalation) Category 3, Carcinogenicity Category 1A, Sensitisation (Skin) Category 1
Legend:	1. Classification by vendor; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

XT-110S GMAW Low Alloy CuZn Coated Mig Wire

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

H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H373	May cause damage to organs through prolonged or repeated exposure.
H331	Toxic if inhaled.
H350	May cause cancer.
H317	May cause an allergic skin reaction.

Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves and protective clothing.
P284	[In case of inadequate ventilation] wear respiratory protection.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

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
7439-96-5	1.6	<u>manganese</u>
7440-21-3	0.85	<u>silicon</u>
7440-02-0	1.4	<u>nickel</u>
7440-47-3	0.3	<u>chromium</u>
7439-98-7	0.25	<u>molybdenum</u>
7440-50-8	0.25	<u>copper</u>

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CAS No	%[weight]	Name
7440-62-2	0.07	<u>vanadium</u>
Not Available	0-60	<u>welding fumes</u>
Not Available	0-60	<u>welding fumes generating aluminium, copper, manganese or zinc</u>
Not Available	0-60	<u>welding fumes generating barium, chromium, lead or nickel</u>

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

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	<ul style="list-style-type: none"> ▶ Particulate bodies from welding spatter may be removed carefully. ▶ DO NOT attempt to remove particles attached to or embedded in eye. ▶ Lay victim down, on stretcher if available and pad BOTH eyes, make sure dressing does not press on the injured eye by placing thick pads under dressing, above and below the eye. ▶ Seek urgent medical assistance, or transport to hospital. ▶ For "arc eye", i.e. welding flash or UV light burns to the eye: ▶ Place eye pads or light clean dressings over both eyes. ▶ Seek medical assistance. <p>For THERMAL burns:</p> <ul style="list-style-type: none"> ▶ Do NOT remove contact lens ▶ Lay victim down, on stretcher if available and pad BOTH eyes, make sure dressing does not press on the injured eye by placing thick pads under dressing, above and below the eye. ▶ Seek urgent medical assistance, or transport to hospital.
Skin Contact	<p>If skin or hair contact occurs:</p> <ul style="list-style-type: none"> ▶ Quickly but gently, wipe material off skin with a dry, clean cloth. ▶ Immediately remove all contaminated clothing, including footwear. ▶ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▶ Transport to hospital, or doctor. <p>For thermal burns:</p> <ul style="list-style-type: none"> ▶ Decontaminate area around burn. ▶ Consider the use of cold packs and topical antibiotics. <p>For first-degree burns (affecting top layer of skin)</p> <ul style="list-style-type: none"> ▶ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides. ▶ Use compresses if running water is not available. ▶ Cover with sterile non-adhesive bandage or clean cloth. ▶ Do NOT apply butter or ointments; this may cause infection. ▶ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur. <p>For second-degree burns (affecting top two layers of skin)</p> <ul style="list-style-type: none"> ▶ Cool the burn by immerse in cold running water for 10-15 minutes. ▶ Use compresses if running water is not available. ▶ Do NOT apply ice as this may lower body temperature and cause further damage. ▶ Do NOT break blisters or apply butter or ointments; this may cause infection. ▶ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape. <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"> ▶ Lay the person flat. ▶ Elevate feet about 12 inches. ▶ Elevate burn area above heart level, if possible. ▶ Cover the person with coat or blanket. ▶ Seek medical assistance. <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"> ▶ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound. ▶ Separate burned toes and fingers with dry, sterile dressings. ▶ Do not soak burn in water or apply ointments or butter; this may cause infection. ▶ To prevent shock see above. ▶ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. ▶ Have a person with a facial burn sit up. ▶ Check pulse and breathing to monitor for shock until emergency help arrives.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.

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	<ul style="list-style-type: none"> ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay.
Ingestion	<ul style="list-style-type: none"> ▶ Give a slurry of activated charcoal in water to drink. NEVER GIVE AN UNCONSCIOUS PATIENT WATER TO DRINK. ▶ At least 3 tablespoons in a glass of water should be given. ▶ Although induction of vomiting may be recommended (IN CONSCIOUS PERSONS ONLY), such a first aid measure is dissuaded due to the risk of aspiration of stomach contents. (i) It is better to take the patient to a doctor who can decide on the necessity and method of emptying the stomach. (ii) Special circumstances may however exist; these include non-availability of charcoal and the ready availability of the doctor. <p>NOTE: If vomiting is induced, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.</p> <p>NOTE: Wear protective gloves when inducing vomiting.</p> <ul style="list-style-type: none"> ▶ REFER FOR MEDICAL ATTENTION WITHOUT DELAY. ▶ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. ▶ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist. ▶ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS. (ICSC20305/20307)

Indication of any immediate medical attention and special treatment needed

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

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

[Ellenhorn and Barceloux: Medical Toxicology]

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

[Ellenhorn and Barceloux: Medical Toxicology]

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

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

For 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

SECTION 5 Firefighting measures

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Extinguishing media

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves in the event of a fire. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ Use fire fighting procedures suitable for surrounding area. ▸ DO NOT approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire. ▸ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▸ Non combustible. ▸ Not considered a significant fire risk, however containers may burn. <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.</p> <p>Welding arc and metal sparks can ignite combustibles.</p>
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

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

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ Avoid all personal contact, including inhalation.
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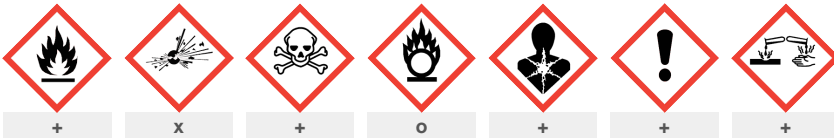
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	<ul style="list-style-type: none"> ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ DO NOT allow material to contact humans, exposed food or food utensils. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
<p>Other information</p>	<ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ Store in a cool, dry area protected from environmental extremes. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. <p>For major quantities:</p> <ul style="list-style-type: none"> ▶ Consider storage in banded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams). ▶ Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities.

Conditions for safe storage, including any incompatibilities

<p>Suitable container</p>	<ul style="list-style-type: none"> ▶ Polyethylene or polypropylene container. ▶ Check all containers are clearly labelled and free from leaks.
<p>Storage incompatibility</p>	<p>Derivative of electropositive metal. Derivative of electronegative metal. For aluminas (aluminium oxide): Incompatible with hot chlorinated rubber. In the presence of chlorine trifluoride may react violently and ignite. -May initiate explosive polymerisation of olefin oxides including ethylene oxide. -Produces exothermic reaction above 200°C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals. -Produces exothermic reaction with oxygen difluoride. -May form explosive mixture with oxygen difluoride. -Forms explosive mixtures with sodium nitrate. -Reacts vigorously with vinyl acetate.</p> <p>Aluminium oxide is an amphoteric substance, meaning it can react with both acids and bases, such as hydrofluoric acid and sodium hydroxide, acting as an acid with a base and a base with an acid, neutralising the other and producing a salt.</p> <ul style="list-style-type: none"> ▶ WARNING: Avoid or control reaction with peroxides. All <i>transition metal</i> peroxides should be considered as potentially explosive. For example transition metal complexes of alkyl hydroperoxides may decompose explosively. ▶ The pi-complexes formed between chromium(0), vanadium(0) and other transition metals (haloarene-metal complexes) and mono- or poly-fluorobenzene show extreme sensitivity to heat and are explosive. ▶ Avoid reaction with borohydrides or cyanoborohydrides ▶ 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. <p>Welding electrodes should not be allowed to come into contact with strong acids or other substances which are corrosive to metals.</p> <p>Nitric oxide:</p> <ul style="list-style-type: none"> ▶ is reactive with alkalis, flammable and combustible materials, organic compounds and solvents, reducing agents, copper and aluminium. ▶ 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.

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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	manganese	Manganese, fume (as Mn)	1 mg/m3	3 mg/m3	Not Available	Not Available
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	nickel	Nickel, metal	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	nickel	Nickel, powder	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	chromium	Chromium (metal)	0.5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	copper	Copper (fume)	0.2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	copper	Copper, dusts & mists (as Cu)	1 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
manganese	3 mg/m3	5 mg/m3	1,800 mg/m3
silicon	45 mg/m3	100 mg/m3	630 mg/m3
nickel	4.5 mg/m3	50 mg/m3	99 mg/m3
chromium	1.5 mg/m3	17 mg/m3	99 mg/m3
molybdenum	30 mg/m3	330 mg/m3	2,000 mg/m3
copper	3 mg/m3	33 mg/m3	200 mg/m3
vanadium	3 mg/m3	5.8 mg/m3	35 mg/m3

Ingredient	Original IDLH	Revised IDLH
manganese	500 mg/m3	Not Available
silicon	Not Available	Not Available
nickel	10 mg/m3	Not Available
chromium	250 mg/m3	Not Available
molybdenum	Not Available	Not Available
copper	100 mg/m3	Not Available
vanadium	Not Available	Not Available
welding fumes	Not Available	Not Available
welding fumes generating aluminium, copper, manganese or zinc	Not Available	Not Available

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Ingredient	Original IDLH	Revised IDLH
welding fumes generating barium, chromium, lead or nickel	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
molybdenum	E	≤ 0.01 mg/m ³
welding fumes generating aluminium, copper, manganese or zinc	E	≤ 0.01 mg/m ³
welding fumes generating barium, chromium, lead or nickel	E	≤ 0.01 mg/m ³

Notes: 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.

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.

An increased incidence of non-specific symptoms including headache, weakness, fatigue, anorexia and joint and muscle weakness has been reported to occur in mining and metallurgy workers exposed to 60-600 mg (as Mo). Some investigators have attributed gout and elevated uric acid concentration found in some Armenians to result from exposures to Armenian soils rich in molybdenum, whilst exposure has been implicated as a cause of bone disease amongst Indians. "These involvements are speculative". [US National Research Council]. As far as it is known, the recommended TLV-TWA incorporates a large margin of safety against potential pulmonary or systemic effects.

Exposure to vanadium dusts can induce cough, rhinorrhea, ocular burning and conjunctivitis, nasal catarrh and hemorrhage, wheezing, rales, green to black tongue and rhonchi.

The recommended TLV is thought to minimise the potential of upper respiratory tract irritation, pulmonary disease, and systemic vanadium intoxication. Although no data has been produced to suggest vanadium pentoxide is a carcinogen, increases in the number of normal cells (chronic hyperplasia) in the nasal epithelium of workers exposed to high concentrations of the dust, continues to raise concerns.

The TWA was established to limit the total daily dose rather than a ceiling (compare NIOSH recommendation) limit whose prime use limits peak exposure concentrations. New documentation is currently being reviewed to establish whether a ceiling value is more appropriate.

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 an hour and may produce unconsciousness in 1.5 hours; 4000 ppm is fatal in less than an hour.

The TLV-TWA and STEL is recommended to keep blood carboxyhaemoglobin (CoHb) levels below 3.5% in workers so as to prevent adverse neurobehavioural changes and to maintain cardiovascular exercise. Earlier recommendations did not take into account heavy labour, high temperature, high elevations (over 5000 feet above sea level), adverse effects on pregnant workers (i.e. the foetus) and the effects on those with chronic heart and respiratory disease. Workers who smoke frequently have CoHb saturations above 3.5%.

Coburn et al have calculated the time needed to reach 3.5% CoHb at various carbon monoxide exposures.

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

Work Load is defined in terms of alveolar ventilation as:

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

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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:

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

Appropriate engineering controls	<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"> ▶ 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) <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>														
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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 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:

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1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
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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 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.

Personal protection



Eye and face protection

- ▶ Goggles or other suitable eye protection shall be used during all gas welding or oxygen cutting operations. Spectacles without side shields, with suitable filter lenses are permitted for use during gas welding operations on light work, for torch brazing or for inspection.
 - ▶ For most open welding/brazing operations, goggles, even with appropriate filters, will not afford sufficient facial protection for operators. Where possible use welding helmets or handshields corresponding to EN 175, ANSI Z49:12005, AS 1336 and AS 1338 which provide the maximum possible facial protection from flying particles and fragments. [WRIA-WTIA Technical Note 7]
 - ▶ An approved face shield or welding helmet can also have filters for optical radiation protection, and offer additional protection against debris and sparks.
 - ▶ UV blocking protective spectacles with side shields or welding goggles are considered primary protection, with the face shield or welding helmet considered secondary protection.
 - ▶ The optical filter in welding goggles, face mask or helmet must be a type which is suitable for the sort of work being done. A filter suitable for gas welding, for instance, should not be used for arc welding.
 - ▶ Face masks which are self dimming are available for arc welding, MIG, TIG and plasma cutting, and allow better vision before the arc is struck and after it is extinguished.
- For submerged arc welding use a lens shade which gives just sufficient arc brightness to allow weld pool control.

Skin protection

See Hand protection below

Hands/feet protection

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.

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	<p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <ul style="list-style-type: none"> ▸ 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 ▸ These gloves protect against mechanical risk caused by abrasion, blade cut, tear and puncture ▸ Other gloves which protect against thermal risks (heat and fire) might also be considered - these comply with different standards to those mentioned above. ▸ One pair of gloves may not be suitable for all processes. For example, gloves that are suitable for low current Gas Tungsten Arc Welding (GTAW) (thin and flexible) would not be proper for high-current Air Carbon Arc Cutting (CAC-A) (insulated, tough, and durable) <p>Experience indicates that the following polymers are suitable as glove materials for protection against undissolved, dry solids, where abrasive particles are not present.</p> <ul style="list-style-type: none"> ▸ polychloroprene. ▸ nitrile rubber. ▸ butyl rubber. ▸ fluoroacoutchouc. ▸ polyvinyl chloride. <p>Gloves should be examined for wear and/ or degradation constantly.</p>
<p>Body protection</p>	<p>See Other protection below</p>
<p>Other protection</p>	<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"> ▸ Overalls. ▸ P.V.C apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.

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 -

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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 discomfoting 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

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

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	<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>Strong evidence exists that exposure to the material may produce serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation. 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>Bronchial and alveolar exudate are apparent in animals exposed to molybdenum by inhalation. Molybdenum fume may produce bronchial irritation and moderate fatty changes in liver and kidney.</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 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 stertorous 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>
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Ingestion

Strong evidence exists that exposure to the material may produce serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing.

The material has **NOT** been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.

Ingestion of large doses may result in severe distress, cramping, vomiting and hypertension. Molybdenum is rapidly excreted from the body as the molybdate and does not accumulate in mammals. The biological half-life is of the order of hours in experimental animals and weeks in humans.

Molybdenum is of biological importance as an essential trace element in the Mo-flavoprotein enzyme, xanthine oxidase. It is also necessary for nitrogen-fixation by soil bacteria; livestock poisoning has been recorded in animals feeding on herbage containing high levels of molybdenum. Signs of molybdenum poisoning include loss of appetite, listlessness, diarrhoea and reduced growth rate. Anaemia is characteristic of molybdenum toxicity with low haemoglobin concentration and reduced red blood cell count. Livers and kidneys of severely poisoned animals show fatty degeneration. Other symptoms include achromotrichia (loss of hair pigment), testicular degeneration, poor conception and deficient lactation, dyspnoea, incoordination and irritation of the mucous membranes.

Molybdenum depresses liver sulfide oxidase activity and the resulting sulfide accumulation leads to the formation of highly insoluble cupric sulfide and the subsequent appearance of copper deficiency. Symptoms of molybdenosis described above are similar to those of hypocuprosis.

Poisonings rarely occur after oral administration of manganese salts as they are generally poorly absorbed from the gut (generally less than 4%) and seems to be dependent, in part, on levels of dietary iron and may increase following the consumption of alcohol. A side-effect of oral manganese administration is an increase in losses of calcium in the faeces and a subsequent lowering of calcium blood levels. Absorbed manganese tends to be slowly excreted in the bile. Divalent manganese appears to be 2.5-3 times more toxic than the trivalent form.

Skin Contact

Strong evidence exists that exposure to the material may produce serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact.

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.

Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus. Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles.

Ultraviolet radiation (UV) is generated by the electric arc in the welding process. Skin exposure to UV can result in severe burns, in many cases without prior warning.

Exposure to infrared radiation (IR), produced by the electric arc and other flame cutting equipment may heat the skin surface and the tissues immediately below the surface. Except for this effect, which can progress to thermal burns in some situations, infrared radiation is not dangerous to welders. Most welders protect themselves from IR (and UV) with a welder's helmet (or glasses) and protective clothing.

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.

The basic types of engineering controls are:

- Process controls which involve changing the way a job activity or process is done to reduce the risk.
- Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.

Special ventilation requirements apply for processes which result in the generation of aluminium, copper, fluoride, manganese or zinc fume.

- For work conducted outdoors and in open work spaces, the use of mechanical (general exhaust or plenum) ventilation is required as a minimum. (Open work spaces exceed 300 cubic meters per welder)
- For indoor work, conducted in limited or confined work spaces, use of mechanical ventilation by local exhaust systems is mandatory. (In confined spaces always check that oxygen has not been depleted by excessive rusting of steel or snowflake corrosion of aluminium)

Local exhaust systems must be designed to provide a minimum capture velocity at the fume source, away from the worker, of 0.5 metre/sec. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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

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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

There is sufficient evidence to provide a strong presumption that human exposure to the material may produce heritable genetic damage.

There is sufficient evidence to provide a strong presumption that human exposure to the material may result in the development of heritable genetic damage, generally on the basis of

- appropriate animal studies,
- other relevant information

There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: - clear evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects.

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

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

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

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

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

There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours.

Only those particles small enough to enter the alveoli (sub 5 um) are able to produce pathogenic effects in the lungs.

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

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confusion, aggressiveness and hallucinations. The final stage is characterised by speech difficulties, muscular twitching, spastic gait and other nervous system effects. Symptoms resemble those of Parkinson's disease. Rat studies indicate the gradual accumulation of brain manganese to produce lesions mimicking those found in Parkinsonism. If the disease is diagnosed whilst still in the early stages and the patient is removed from exposure, the course may be reversed.

Inhalation of manganese fumes may cause 'metal fume fever' characterised by flu-like symptoms: fever, chill, nausea, weakness and body aches. Manganese dust is no longer believed to be a causative factor in pneumonia. If there is any relationship at all, it appears to be as an aggravating factor to a preexisting condition.

Prolonged or repeated eye contact may result in conjunctivitis.

Manganese is an essential trace element in all living organisms with the level of tissue manganese remaining remarkably constant throughout life.

Persons, exposed for long periods to molybdenum oxides, suffer from anaemia. Animals exposed to certain insoluble molybdenum compounds show anorexia, diarrhoea, weight loss, listlessness, and liver and kidney damage. Molybdenum disturbs bone metabolism, giving rise to lameness, bone joint abnormalities, osteoporosis and high serum phosphatase levels. Cattle, rabbits, and chicks on high dietary levels of molybdenum exhibited deformities of joints of the extremities. Low molybdenum intake has been attributed to the high incidence of oesophageal cancer in South Africa among the Bantu of Transkei, in China and in Russia.

Chronic exposure of workers in Russian molybdenum-copper plant resulted in a fall in the albumin/globulin ratio owing to a rise in globulins (particularly alpha-globulins) which is interpreted as evidence of liver dysfunction with hyperbilirubinaemia. Hepatotoxic effects are also found in animals given molybdenum salts with a rise in alpha-globulin levels, hypoalbuminaemia and increased serum bilirubin reported. Other reported biochemical effects include an early depletive effect on tissue nicotinamide nucleotides, hyperaminoaciduria, reduction in red blood cell life-span and hyper-thyroidism. Industrial exposure to some insoluble molybdenum compounds is thought to have resulted in an increased incidence of weakness, fatigue, anorexia, headache and joint and muscular pain. Under the conditions of a 2-year inhalation study* there was equivocal evidence of carcinogenic activity of molybdenum trioxide in male rats, male mice and female mice based on a marginally significant positive trend of alveolar/bronchiolar adenoma or carcinoma. There was no evidence of carcinogenic activity in female rats exposed to 10, 30 and 100 mg/m³. Exposure of male and female rats to molybdenum trioxide by inhalation resulted in increased incidences of chronic alveolar inflammation, hyaline degeneration of the olfactory epithelium (females), hyaline degeneration of the respiratory epithelium and squamous metaplasia of the epiglottis. Exposure of female and male mice to molybdenum trioxide resulted in an increased incidence of metaplasia of the alveolar epithelium, histiocyte cellular inflammation (males), hyaline degeneration of the respiratory epithelium, hyaline degeneration of the olfactory epithelium (females), squamous metaplasia of the epiglottis, and hyperplasia of the larynx.

Guinea pigs exposed to molybdenum trioxide dust for 1 hour daily at 250 mg/m³ showed extreme irritation. Symptoms include loss of appetite, weight loss, diarrhoea, muscular incoordination and loss of hair. Of the 51 animals exposed, 26 died after the tenth exposure. Exposure to freshly generated MoO₂ fume under about the same exposure conditions proved unexpectedly less toxic, with only 8.3% mortality compared with 51% mortality with the dust, and no mortality when the exposure level was reduced to about one-third (57 mg Mo/m³). Explanation for this unexpected finding was felt to reside in the more rapid solution and elimination of the large surface area fume particle. [Patty's]

Exposure of male and female rats to molybdenum trioxide resulted in the development of respiratory system lesions. In the lung, the incidence and severity of chronic alveolar inflammation increased with increasing exposure concentration in male and female rats. In some male rats, exposure to the material resulted in alveolar/ bronchiolar adenomas or carcinomas. Lesions in the nose (hyaline degeneration) and larynx (squamous metaplasia) were considered to be a non-specific defensive or adaptive response to chronic inhalation exposure. Inhalation exposure of mice to molybdenum trioxide was associated with the development of lung neoplasms and an increased incidence of alveolar/ bronchiolar carcinoma or adenoma in both sexes. Chronic inflammatory lesions were not present in the lungs. Lesions of the nose and larynx were similar to those observed in rats.

Molybdenum trioxide was not mutagenic in any of five strains of *Salmonella typhimurium* and did not induce sister chromatid exchanges or chromosomal aberrations in cultured Chinese hamster ovary cells in vitro. All tests were conducted with or without S9 metabolic activation enzymes.

Pneumoconiosis has been described in experimental animals exposed sub-chronically to molybdenum trioxide.

The mechanism of molybdenum trioxide action in lung carcinogenicity is not known; the material is not mutagenic.

Non-neoplastic lesions of the nose and larynx of rats and in the nose, larynx and lungs of mice were apparently due to the development of a more durable epithelium in response to chronic exposure.

The US Department of Health and Human Services (1) concluded that there was equivocal evidence of carcinogenic activity in male F344/N rats based on a marginally significant positive trend of alveolar/ bronchiolar adenoma or carcinoma; that there was no evidence of carcinogenic activity in female F344/N rats; that there was some evidence of carcinogenic activity in male B6C3F1 mice and that there was evidence of carcinogenic activity in female B6C3F1 mice

National Toxicology Program: Technical Report Series 462, April 1997

Long-term (chronic) exposure to low levels of carbon monoxide may produce heart disease and damage to the nervous system.

Exposure of pregnant animals to carbon monoxide may cause low birthweight, increased foetal mortality and nervous system damage to the offspring.

Carbon monoxide is a common cause of fatal poisoning in industry and homes. Non fatal poisoning may result in permanent nervous system damage. Carbon monoxide reduces the oxygen carrying capacity of the blood. Effects on the body are considered to be reversible as long as brain cell damage or heart failure has not occurred. Avoid prolonged exposure, even to small concentrations. A well-established and probably causal relationship exists between maternal smoking (resulting in carboxyhaemoglobin levels of 2-7% in the foetus) and low birth weight. There also appears to be a dose-related increase in perinatal deaths and a retardation of mental ability in infants born to smoking mothers.

The foetus and newborn infant are considered to be very susceptible to CO exposure for several reasons:

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

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Carbon monoxide gas readily crosses the placenta and CO exposure during pregnancy can be teratogenic. Carbon dioxide at low levels may initiate or enhance deleterious myocardial alterations in individuals with restricted coronary artery blood flow and decreased myocardial lactate production. - Linde

On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

Principal route of exposure is inhalation of welding fumes from electrodes and workpiece. Reaction products arising from electrode core and flux appear as welding fume depending on welding conditions, relative volatilities of metal oxides and any coatings on the workpiece. Studies of lung cancer among welders indicate that they may experience a 30-40% increased risk compared to the general population. Since smoking and exposure to other cancer-causing agents, such as asbestos fibre, may influence these results, it is not clear whether welding, in fact, represents a significant lung cancer risk. Whilst mild steel welding represents little risk, the stainless steel welder, exposed to chromium and nickel fume, may be at risk and it is this factor which may account for the overall increase in lung cancer incidence among welders. Cold isolated electrodes are relatively harmless. Metal oxides generated by industrial processes such as welding, give rise to a number of potential health problems. Particles smaller than 5 micron (respirables) articles may cause lung deterioration. Particles of less than 1.5 micron can be trapped in the lungs and, dependent on the nature of the particle, may give rise to further serious health consequences.

Exposure to fume containing high concentrations of water-soluble chromium (VI) during the welding of stainless steels in confined spaces has been reported to result in chronic chrome intoxication, dermatitis and asthma. Certain insoluble chromium (VI) compounds have been named as carcinogens (by the ACGIH) in other work environments. Chromium may also appear in welding fumes as Cr2O3 or double oxides with iron. These chromium (III) compounds are generally biologically inert.

Welding fume with high levels of ferrous materials may lead to particle deposition in the lungs (siderosis) after long exposure. This clears up when exposure stops. Chronic exposure to iron dusts may lead to eye disorders.

Silica and silicates in welding fumes are non-crystalline and believed to be non-harmful.

Other welding process exposures can arise from radiant energy UV flash burns, thermal burns or electric shock

The welding arc emits ultraviolet radiation at wavelengths that have the potential to produce skin tumours in animals and in over-exposed individuals, however, no confirmatory studies of this effect in welders have been reported.

XT-110S GMAW Low Alloy CuZn Coated Mig Wire	TOXICITY	IRRITATION
	Not Available	Not Available
manganese	TOXICITY	IRRITATION
	Inhalation(Rat) LC50; >5.14 mg/l4h ^[1]	Eye (rabbit): 500 mg/24h - mild
	Oral(Rat) LD50; >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin (rabbit): 500 mg/24h - mild
		Skin: no adverse effect observed (not irritating) ^[1]
silicon	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral(Rat) LD50; >5000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
nickel	TOXICITY	IRRITATION
	Oral(Rat) LD50; >9000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
chromium	TOXICITY	IRRITATION
	Inhalation(Rat) LC50; >5.41 mg/l4h ^[1]	Not Available
	Oral(Rat) LD50; >5000 mg/kg ^[1]	
molybdenum	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Not Available
	Inhalation(Rat) LC50; >1.93 mg/l4h ^[1]	
	Oral(Rat) LD50; >2000 mg/kg ^[1]	
copper	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation(Rat) LC50; 0.733 mg/l4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral(Mouse) LD50; 0.7 mg/kg ^[2]	
vanadium	TOXICITY	IRRITATION

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	Inhalation(Rat) LC50; >5.05 mg/l4h ^[1]	Not Available
	Oral(Rat) LD50; >2000 mg/kg ^[1]	
welding fumes	TOXICITY	IRRITATION
	Not Available	Not Available
welding fumes generating aluminium, copper, manganese or zinc	TOXICITY	IRRITATION
	Not Available	Not Available
welding fumes generating barium, chromium, lead or nickel	TOXICITY	IRRITATION
	Not Available	Not Available
Legend:	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

MANGANESE	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.
SILICON	<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>
NICKEL	<p>Oral (rat) TDLo: 500 mg/kg/5D-I Inhalation (rat) TCLo: 0.1 mg/m³/24H/17W-C</p> <p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>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>Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [National Toxicology Program: U.S. Dep. of Health & Human Services 2002]</p>
CHROMIUM	<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.</p> <p>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, 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</p>

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	<p>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</p> <p>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.</p> <p>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 <i>[National Toxicology Program: U.S. Dep. of Health and Human Services 2002]</i></p>
<p>COPPER</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>Acute toxicity: There are no reliable acute oral toxicity results available. In an acute dermal toxicity study (OECD TG 402), one group of 5 male rats and 5 groups of 5 female rats received doses of 1000, 1500 and 2000 mg/kg bw via dermal application for 24 hours. The LD50 values of copper monochloride were 2,000 mg/kg bw or greater for male (no deaths observed) and 1,224 mg/kg bw for female. Four females died at both 1500 and 2000 mg/kg bw, and one at 1,000 mg/kg bw. Symptom of the hardness of skin, an exudation of hardness site, the formation of scar and reddish changes were observed on application sites in all treated animals. Skin inflammation and injury were also noted. In addition, a reddish or black urine was observed in females at 2,000, 1,500 and 1,000 mg/kg bw. Female rats appeared to be more sensitive than male based on mortality and clinical signs. No reliable skin/eye irritation studies were available. The acute dermal study with copper monochloride suggests that it has a potential to cause skin irritation.</p> <p>Repeat dose toxicity: In repeated dose toxicity study performed according to OECD TG 422, copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39 - 51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL value was 5 and 1.3 mg/kg bw/day for male and female rats, respectively. No deaths were observed in male rats. One treatment-related death was observed in female rats in the high dose group. Erythropoietic toxicity (anaemia) was seen in both sexes at the 80 mg/kg bw/day. The frequency of squamous cell hyperplasia of the forestomach was increased in a dose-dependent manner in male and female rats at all treatment groups, and was statistically significant in males at doses of =20 mg/kg bw/day and in females at doses of =5 mg/kg bw/day doses. The observed effects are considered to be local, non-systemic effect on the forestomach which result from oral (gavage) administration of copper monochloride.</p> <p>Genotoxicity: An in vitro genotoxicity study with copper monochloride showed negative results in a bacterial reverse mutation test with Salmonella typhimurium strains (TA 98, TA 100, TA 1535, and TA 1537) with and without S9 mix at concentrations of up to 1,000 ug/plate. An in vitro test for chromosome aberration in Chinese hamster lung (CHL) cells showed that copper monochloride induced structural and numerical aberrations at the concentration of 50, 70 and 100 ug/mL without S9 mix. In the presence of the metabolic activation system, significant increases of structural aberrations were observed at 50 and 70 ug/mL and significant increases of numerical aberrations were observed at 70 ug/mL. In an in vivo mammalian erythrocyte micronucleus assay, all animals dosed (15 - 60 mg/kg bw) with copper monochloride exhibited similar PCE/(PCE+NCE) ratios and MNPCE frequencies compared to those of the negative control animals. Therefore copper monochloride is not an in vivo mutagen.</p> <p>Carcinogenicity: there was insufficient information to evaluate the carcinogenic activity of copper monochloride.</p> <p>Reproductive and developmental toxicity: In the combined repeated dose toxicity study with the reproduction/developmental toxicity screening test (OECD TG 422), copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39-51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL of copper monochloride for fertility toxicity was 80 mg/kg bw/day for the parental animals. No treatment-related effects were observed on the reproductive organs and the fertility parameters assessed. For developmental toxicity the NOAEL was 20 mg/kg bw/day. Three of 120 pups appeared to have icterus at birth; 4 of 120 pups appeared runted at the highest dose tested (80 mg/kg bw/day).</p>
<p>XT-110S GMAW Low Alloy CuZn Coated Mig Wire & NICKEL</p>	<p>WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.</p>
<p>XT-110S GMAW Low Alloy CuZn Coated Mig Wire & WELDING FUMES & WELDING FUMES GENERATING ALUMINIUM, COPPER, MANGANESE OR ZINC & WELDING FUMES GENERATING</p>	<p>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.</p> <p>In 2017, an IARC working group has determined that "sufficient evidence exists that welding fume is a human lung carcinogen (Group 1).</p> <p>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</p>

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<p>BARIUM, CHROMIUM, LEAD OR NICKEL</p>	<p>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.</p> <p>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</p> <p>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 have proposed that iron fumes may play such a role, and some Finnish data appear to support this hypothesis, though not conclusively. This hypothesis would also imply that excess lung cancer risks among welders are not unique to welders, but rather may be shared among many types of metal working occupations.</p> <p>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.</p> <p>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.</p> <p>Personal exposures to manganese ranged from 0.01-4.93 mg/m3 and to iron ranged from 0.04-16.29 mg/m3 in eight Canadian welding companies. Types of welding identified were mostly (90%) MIG mild steel, MIG stainless steel, and TIG aluminum. Carbon monoxide levels were less than 5.0 ppm (at source) and ozone levels varied from 0.4-0.6 ppm (at source). Welders, especially in shipyards, may also be exposed to asbestos dust. Physical exposures such as electric and magnetic fields (EMF) and ultraviolet (UV) radiation are also common.</p> <p>In all, the in vivo studies suggest that different welding fumes cause varied responses in rat lungs in vivo , and the toxic effects typically correlate with the metal composition of the fumes and their ability to produce free radicals. In many studies both soluble and insoluble fractions of the stainless steel welding fumes were required to produce most types of effects, indicating that the responses are not dependent exclusively on the soluble metals</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>
<p>MANGANESE & SILICON</p>	<p>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
<p>SILICON & CHROMIUM & MOLYBDENUM</p>	<p>No significant acute toxicological data identified in literature search.</p>
<p>WELDING FUMES & WELDING FUMES GENERATING ALUMINIUM, COPPER, MANGANESE OR ZINC & WELDING FUMES GENERATING BARIUM, CHROMIUM, LEAD OR NICKEL</p>	<p>WARNING: This substance has been classified by the IARC as Group 1: CARCINOGENIC TO HUMANS.</p> <p>Not available. Refer to individual constituents.</p>

<p>Acute Toxicity</p>	<p>✓</p>	<p>Carcinogenicity</p>	<p>✓</p>
<p>Skin Irritation/Corrosion</p>	<p>✗</p>	<p>Reproductivity</p>	<p>✗</p>
<p>Serious Eye Damage/Irritation</p>	<p>✗</p>	<p>STOT - Single Exposure</p>	<p>✗</p>

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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-110S GMAW Low Alloy CuZn Coated Mig Wire	Endpoint	Test Duration (hr)	Species	Value	Source
		Not Available	Not Available	Not Available	Not Available

manganese	Endpoint	Test Duration (hr)	Species	Value	Source
	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

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	

nickel	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	72h	Algae or other aquatic plants	0.18mg/l	1
	EC50	72h	Algae or other aquatic plants	0.18mg/l	1
	LC50	96h	Fish	0.168mg/L	4
	EC50	48h	Crustacea	>100mg/l	1
EC50	96h	Algae or other aquatic plants	0.36mg/l	2	

chromium	Endpoint	Test Duration (hr)	Species	Value	Source
	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	

molybdenum	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	48h	Algae or other aquatic plants	0.5-80mg/l	4
	EC50	72h	Algae or other aquatic plants	26mg/l	2
	LC50	96h	Fish	211mg/l	2
EC50	48h	Crustacea	130.9mg/l	2	

copper	Endpoint	Test Duration (hr)	Species	Value	Source
	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	

vanadium	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	48h	Algae or other aquatic plants	0.5-18mg/l	4
LC50	96h	Fish	1.8mg/l	4	

welding fumes	Endpoint	Test Duration (hr)	Species	Value	Source

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	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
welding fumes generating barium, chromium, lead or nickel	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
Legend:	Extracted from 1. IUCALD 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, may cause long-term adverse effects in the aquatic environment.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

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 manganese and its compounds:

Environmental fate:

It has been established that while lower organisms (e.g., plankton, aquatic plants, and some fish) can significantly bioconcentrate manganese, higher organisms (including humans) tend to maintain manganese homeostasis. This indicates that the potential for biomagnification of manganese from lower trophic levels to higher ones is low.

There were two mechanisms involved in explaining the retention of manganese and other metals in the environment by soil. First, through cation exchange reactions, manganese ions and the charged surface of soil particles form manganese oxides, hydroxides, and oxyhydroxides which in turn form absorption sites for other metals. Secondly, manganese can be adsorbed to other oxides, hydroxides, and oxyhydroxides through ligand exchange reactions. When the soil solution becomes saturated, these manganese oxides, hydroxides, and oxyhydroxides can precipitate into a new mineral phase and act as a new surface to which other substances can absorb. The tendency of soluble manganese compounds to adsorb to soils and sediments depends mainly on the cation exchange capacity and the organic composition of the soil. The soil adsorption constants (the ratio of the concentration in soil to the concentration in water) for Mn(II) span five orders of magnitude, ranging from 0.2 to 10,000 mL/g, increasing as a function of the organic content and the ion exchange capacity of the soil; thus, adsorption may be highly variable. In some cases, adsorption of manganese to soils may not be a readily reversible process. At low concentrations, manganese may be "fixed" by clays and will not be released into solution readily. At higher concentrations, manganese may be desorbed by ion exchange mechanisms with other ions in solution. For example, the discharge of waste water effluent into estuarine environments resulted in the mobilization of manganese from the bottom sediments. The metals in the effluent may have been preferentially adsorbed resulting in the release of manganese. The oxidation state of manganese in soil and sediments may be altered by microbial activity; oxidation may lead to the precipitation of manganese. Bacteria and microflora can increase the mobility of manganese.

The transport and partitioning of manganese in water is controlled by the solubility of the specific chemical form present, which in turn is determined by pH, Eh (oxidation-reduction potential), and the characteristics of the available anions. The metal may exist in water in any of four oxidation states.

Manganese(II) predominates in most waters (pH 4-7) but may become oxidized at a pH >8 or 9. The principal anion associated with Mn(II) in water is usually carbonate (CO₃²⁻), and the concentration of manganese is limited by the relatively low solubility (65 mg/L) of MnCO₃. 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

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Daphnia magna LC50: 50 mg/l

Pseudomonas putida LC50: 10.6 mg/l

Photobacterium phosphoreum LC50: 14.7 mg/l

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

Based on the high concentration of molybdenum in all analysed waste types, the exposure of the environment to molybdenum is regarded as significant. The limited amount of data regarding its toxicity makes it impossible to evaluate the potential for adverse environmental and health effects from molybdenum exposure. Molybdenum cause adverse effects in ruminant animals. Livestock have been injured by forage grown on soils with excessive geochemically-derived molybdenum. Soil molybdenum is a potentially toxic element, but no cases have been reported of molybdenum toxicity to animals from consumption of forage grown on sludge-amended soils. In pot studies, where clover was grown on alkaline soils containing up to 16 kg of molybdenum per ha, concentrations in the plant tissue reached levels that could be harmful to animals if the clover were to make up a substantial portion of the diet for an extended period of time.

Molybdenum is generally found in two oxidation states in nature, Mo(IV) and Mo(VI). In oxidising environments Mo(VI) dominates and it is commonly present as the oxyanion molybdate (MoO_4^{2-})

In a laboratory experiment it was found that Mo was lost from solution under reducing conditions and remobilised under oxidizing conditions, and hypothesised that MoS_2 , a low-solubility mineral, formed in the system.

In this study it was also found that Fe minerals were important sinks for Mo accumulation in reducing sediments. It has been proposed that under reducing conditions and with the reduction of sulfate, molybdate is converted to thiomolybdate (MoS_4^{2-}) which then binds to Fe, Al, and organic matter phases a via sulfur bridges. This mechanism could also account for decreased Mo solubility under reducing conditions.

Another study in wetland found that Mo accumulated in the sediments with most of the accumulation occurring in the top 2 cm and decreasing with depth. It appears that Mo accumulation (as well as As and V accumulation) or retention in the surface sediments is dependent on the depth of the overlying water column and correspondingly on redox status.

For vanadium compounds:

Environmental fate:

The global biogeochemical cycling of vanadium is characterized by releases to the atmosphere, water, and land by natural and anthropogenic sources, long-range transportation of particles in both air and water, wet and dry deposition, adsorption, and complexing. Vanadium generally enters the atmosphere as an aerosol.

From natural sources, vanadium is probably in the form of mineral particles; it has been suggested that these may frequently be in the less-soluble trivalent form.

From man-made sources almost all the vanadium released to the atmosphere is in the form of simple or complex vanadium oxides. The size distribution of vanadium-bearing particles in the atmosphere is substantially altered during long-range transportation.

Natural sources of vanadium, as well as man-made sources such as ore-processing dust, tend to release large particles that are more likely to settle near the source. Smaller particles, such as those emitted from oil-fueled power plants, have a longer residence time in the atmosphere and are more likely to be transported farther away from the site of release. Vanadium transported within the atmosphere is eventually transferred to soil and water on the earth's surface by wet and dry deposition and dissolution in sea water. Eventually, in the course of biogeochemical movement between soil and water, these particulates are adsorbed to hydroxides or associated with organic compounds and are deposited on the sea bed.

The transport and partitioning of vanadium in water and soil is influenced by pH, redox potential, and the presence of particulate. In fresh water, vanadium generally exists in solution as the vanadyl ion (V^{4+}) under reducing conditions and the vanadate ion (V^{5+}) under oxidizing conditions, or as an integral part of, or adsorbed onto, particulate matter. The chemical formulas of the vanadyl species most commonly reported in fresh water are VO^{2+} and $\text{VO}(\text{OH})^+$, and the vanadate species are H_2VO_4^- and HVO_4^- . The partitioning of vanadium between water and sediment is strongly influenced by the presence of particulate in the water. Both vanadate and vanadyl species are known to bind strongly to mineral or biogenic surfaces by adsorption or complexing. Thus, vanadium is transported in water in one of two ways: solution or suspension. It has been estimated that only 13% is transported in solution, while the remaining 87% is in suspension. Upon entering the ocean, vanadium in suspension or sorbed onto particulate is deposited upon the sea bed. The fate of the remaining dissolved vanadium is more complex. Only about 0.001% of vanadium entering the oceans is estimated to persist in soluble form. Sorption and biochemical processes are thought to contribute to the extraction of vanadium from sea water. Adsorption to organic matter as well as to manganese oxide and ferric hydroxide, demonstrated by the high particle-water partition coefficient of $5.7 \times 10^5 \text{ L/kg}$ for the adsorption of manganese oxide in sea water, results in the precipitation of the dissolved vanadium.

Biochemical processes are also of importance in the partitioning from sea water to sediment. Some marine organisms, in particular the ascidians (sea squirts), bioconcentrate vanadium very efficiently, attaining body concentrations approximately 10,000 times greater than the ambient sea water. Upon the death of the organism, the body burden adds to the accumulation of vanadium in silt. The extent to which either bioconcentration or adsorption dominates is uncertain. In general, marine plants and invertebrates contain higher levels of vanadium than terrestrial plants and animals. In the terrestrial environment bioconcentration is more commonly observed amongst the lower plant phyla than in the higher, seed-producing phyla. The vanadium levels in terrestrial plants are dependent upon the amount of water-soluble vanadium available in the soil, pH, and growing conditions. It has been found that the uptake of vanadium into the above-ground parts of many plants is low, although root concentrations have shown some correlation with levels in the soil. Certain legumes, such as *Astragalus preussi*, have been shown to be vanadium accumulators. Vanadium is believed to replace molybdenum as a specific catalyst in nitrogen fixation and the root nodules of these plants may contain vanadium levels three times greater than those of the surrounding soil. Of the few plants known to actively accumulate vanadium, *Amanita muscaria*, a poisonous mushroom, has been demonstrated to contain levels up to 112 ppm (dry weight). Vanadium appears to be present in all terrestrial animals, but, in vertebrates, tissue concentrations are often so low that detection is difficult. The highest levels of vanadium in terrestrial mammals are generally found in the liver and skeletal tissues. No data are available regarding biomagnification of vanadium within the food chain, but human studies suggest that it is unlikely; most of the 1%-2% vanadium that appears to be absorbed by humans following ingestion is rapidly excreted in the urine with no evidence of long-term accumulation.

The form of vanadium present in the soil is determined largely by the parent rock. Ferric hydroxides and solid bitumens (organic) constitute the main carriers of vanadium in the sedimentation process. Iron acts as a carrier for trivalent vanadium due to the great affinity between trivalent vanadium and trivalent iron, and is responsible for its diffusion through molten rocks where it becomes trapped during crystallization. The mobility of vanadium in soils is affected by the pH of the soil. Relative to other metals, vanadium is fairly mobile in neutral or alkaline soils, but its mobility decreases in acidic soils. Similarly, under oxidizing, unsaturated conditions some mobility is observed, but under reducing, saturated conditions vanadium is immobile.

Ecotoxicity:

The available reliable ecotoxicity results selected for the effect of vanadium on aquatic organisms are all based on pentavalent V substances (NaVO_3 , NH_4VO_3 , Na_3VO_4 , V_2O_5 and ammonium polyvanadate).

Reliable short-term toxicity data for freshwater organisms are available for three trophic levels: aquatic invertebrates, fish and algae.

Fish LC50 (96 h): *Leuciscus idus* 0.7 mg V/l (V_2O_5 flake)

Algae EC50 (72 h): *Scenedesmus subspicatus* 2.9 mg V/l (V_2O_5)

Mysid shrimp LC50 (48 h): 13.3 mg V/l

Flagship larvae NOEC (30 d): 76 ug V/l (V_2O_5)

Reliable long-term toxicity data are also available for the effect of V_2O_5 on the development of eggs from two marine organisms (mollusc *Crassostrea gigas* and echinoderm *Paracentrotus lividus*). The NOEC values varied between 25 and 50 ug V/L with the lowest value observed for a 48-h development test of *Crassostrea gigas* eggs.

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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

Product / Packaging disposal	<ul style="list-style-type: none"> ▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Management Authority for disposal. ▸ Bury residue in an authorised landfill. ▸ Recycle containers if possible, or dispose of in an authorised landfill.
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SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

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

Product name	Group
manganese	Not Available
silicon	Not Available
nickel	Not Available
chromium	Not Available
molybdenum	Not Available
copper	Not Available
vanadium	Not Available

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Product name	Group
welding fumes	Not Available
welding fumes generating aluminium, copper, manganese or zinc	Not Available
welding fumes generating barium, chromium, lead or nickel	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
manganese	Not Available
silicon	Not Available
nickel	Not Available
chromium	Not Available
molybdenum	Not Available
copper	Not Available
vanadium	Not Available
welding fumes	Not Available
welding fumes generating aluminium, copper, manganese or zinc	Not Available
welding fumes generating barium, chromium, lead or nickel	Not Available

SECTION 15 Regulatory information

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

manganese is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

silicon is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

nickel is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

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

molybdenum is found on the following regulatory lists

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)

vanadium is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

welding fumes is found on the following regulatory lists

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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 aluminium, copper, manganese or zinc is found on the following regulatory lists

Not Applicable

welding fumes generating barium, chromium, lead or nickel 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 (manganese; silicon; nickel; chromium; molybdenum; copper; vanadium)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (manganese; silicon; nickel; chromium; molybdenum; copper; vanadium)
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
Legend:	<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

Revision Date	29/09/2021
Initial Date	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
chromium	7440-47-3, 188785-87-7, 195161-82-1
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

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

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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
AII: 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