

## **Betaweld Welding Products**

Part Number: 2110, 2111, 2112, 2113

Version No: 1.4.13.9

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

Issue Date: **18/08/2021** Print Date: **18/08/2021** L.GHS.AUS.EN

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

#### **Product Identifier**

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Product name	BW-308LP, BW-309LP Stainless Steel Flux Cored Mig Wire	
Chemical Name	ot Applicable	
Synonyms	0, 2111, 2112, 2113	
Chemical formula	Not Applicable	
Other means of identification	Not Available	

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

Relevant identified uses	A rutile flux cored stainless steel wire for gas shielded welding.
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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 <sup>[1]</sup>	Sensitisation (Respiratory) Category 1, Acute Toxicity (Inhalation) Category 4, Carcinogenicity Category 1A, Sensitisation (Skin) Category 1, Specific Target Organ Toxicity - Repeated Exposure 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

Hazard pictogram(s)	
Signal word	Danger

## Hazard statement(s)

H334	H334 May cause allergy or asthma symptoms or breathing difficulties if inhaled.	
H332	Harmful if inhaled.	
H350	May cause cancer.	
H317	May cause an allergic skin reaction.	
H372	Causes damage to organs through prolonged or repeated exposure.	

## Supplementary statement(s)

Not Applicable

## Precautionary statement(s) Prevention

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

## Precautionary statement(s) Response

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.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
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

P405

## Precautionary statement(s) Disposal

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

## **SECTION 3 Composition / information on ingredients**

Store locked up.

#### Substances

See section below for composition of Mixtures

## Mixtures

CAS No	%[weight]	Name
7439-96-5	1.48-1.58	manganese
7440-21-3	0.51-0.61	silicon
7704-34-9.	0.011-0.013	sulfur
7440-47-3	19.34-22.43	chromium

CAS No		%[weight]	Name
7440-02-0		10.01-12.45	nickel
Not Available		0.012-0.013	phosphorus
Not Available		0-60	welding fumes
Not Available		0-60	welding fumes generating aluminium, copper, manganese or zinc
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> <li>DO NOT attempt to remove particles attached to or embedded in eye .</li> <li>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.</li> <li>Seek urgent medical assistance, or transport to hospital.</li> <li>Particulate bodies from welding spatter may be removed carefully.</li> <li>DO NOT attempt to remove particles attached to or embedded in eye.</li> <li>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.</li> <li>Seek urgent medical assistance, or transport to hospital.</li> <li>For "arc eye", i.e. welding flash or UV light burns to the eye:</li> <li>Place eye pads or light clean dressings over both eyes.</li> <li>Seek medical assistance.</li> <li>For THERMAL burns:</li> <li>Do NOT remove contact lens</li> <li>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.</li> <li>Seek urgent medical assistance.</li> </ul>
Skin Contact	<ul> <li>If skin or hair contact occurs:</li> <li>Quickly but gently, wipe material off skin with a dry, clean cloth.</li> <li>Immediately remove all contaminated clothing, including footwear.</li> <li>Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre.</li> <li>Transport to hospital, or doctor.</li> <li>For thermal burns:</li> <li>Decontaminate area around burn.</li> <li>Consider the use of cold packs and topical antibiotics.</li> <li>For first-degree burns (affecting top layer of skin)</li> <li>Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides.</li> <li>Use compresses if running water is not available.</li> <li>Cover with sterile non-adhesive bandage or clean cloth.</li> <li>Do NOT apply butter or ointments; this may cause infection.</li> <li>Go ise over-the counter pain relievers if pain increases or swelling, redness, fever occur.</li> <li>For second-degree burns (affecting top layer of skin)</li> <li>Cool the burn by immerse in cold running water for 10-15 minutes.</li> <li>Use compresses if running water is not available.</li> <li>Do NOT apply butter or ointments; this may cause infection.</li> <li>Go ise over-the counter pain relieved to players of skin)</li> <li>Cool the burn by immerse in cold running water for 10-15 minutes.</li> <li>Use compresses if running water is not available.</li> <li>Do NOT break bilsters or apply butter or ointments; this may cause infection.</li> <li>Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape.</li> <li>To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort):</li> <li>Lay the person flat.</li> <li>Elevate feet about 12 inches.</li> <li>Elevate burn area above heart level, if possible.</li> <li>Cover the person with coat or blanket.</li> <li>See third-degree burns</li> <li>See third-degree burns</li> <li>See thurnedical ansistance.</li> <li>For third-degree b</li></ul>

Inhalation	<ul> <li>If fumes or combustion products are inhaled remove from contaminated area.</li> <li>Lay patient down. Keep warm and rested.</li> <li>Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.</li> <li>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.</li> <li>Transport to hospital, or doctor, without delay.</li> </ul>
Ingestion	<ul> <li>Give a slurry of activated charcoal in water to drink. NEVER GIVE AN UNCONSCIOUS PATIENT WATER TO DRINK.</li> <li>At least 3 tablespoons in a glass of water should be given.</li> <li>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.</li> <li>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.</li> <li>NOTE: Wear protective gloves when inducing vomiting.</li> <li>REFER FOR MEDICAL ATTENTION WITHOUT DELAY.</li> <li>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.</li> <li>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.</li> <li>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)</li> </ul>

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

Treat symptomatically.

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

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

[Ellenhorn and Barceloux: Medical Toxicology]

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

[Ellenhorn and Barceloux: Medical Toxicology]

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

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

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**

## Extinguishing media

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

DO NOT USE WATER, CO2 or FOAM.

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

## Special hazards arising from the substrate or mixture

Fire Incompatibility	Reacts with acids producing flammable / explosive hydrogen (H2) gas
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#### Advice for firefighters

Fire Fighting	<ul> <li>Alert Fire Brigade and tell them location and nature of hazard.</li> <li>Wear breathing apparatus plus protective gloves in the event of a fire.</li> <li>Prevent, by any means available, spillage from entering drains or water courses.</li> <li>Use fire fighting procedures suitable for surrounding area.</li> <li>DO NOT approach containers suspected to be hot.</li> <li>Cool fire exposed containers with water spray from a protected location.</li> <li>If safe to do so, remove containers from path of fire.</li> <li>Equipment should be thoroughly decontaminated after use.</li> </ul>
Fire/Explosion Hazard	<ul> <li>Do NOT disturb burning dust. Explosion may result if dust is stirred into a cloud, by providing oxygen to a large surface of hot metal.</li> <li>Do NOT use water or foam as generation of explosive hydrogen may result.</li> <li>With the exception of the metals that burn in contact with air or water (for example, sodium), masses of combustible metals do not represent unusual fire risks because they have the ability to conduct heat away from hot spots so efficiently that the heat of combustion cannot be maintained - this means that it will require a lot of heat to ignite a mass of combustible metal. Generally, metal fire risks exist when sawdust, machine shavings and other metal 'fines' are present.</li> <li>Metal powders, while generally regarded as non-combustible:</li> <li>May burn when metal is finely divided and energy input is high.</li> <li>May react explosively with water.</li> <li>May react explosively with water.</li> <li>May REIGNITE after fire is extinguished.</li> <li>Will burn with intense heat.</li> <li>Note:</li> <li>Metal dust fires are slow moving but intense and difficult to extinguish.</li> <li>Containers may explode on heating.</li> <li>Dusts or fumes may form explosive mixtures with air.</li> <li>Gases generated in fire may be poisonous, corrosive or irritating.</li> <li>Hot or burning metals may react violently upon contact with other materials, such as oxidising agents and extinguishing agents used on fires involving ordinary combustibles or flammable liquids.</li> <li>Temperatures produced by burning metals can be higher than temperatures generated by burning flammable liquids</li> <li>Some metals can continue to burn in carbon dioxide, nitrogen, water, or steam atmospheres in which ordinary combustibles or flammable liquids would be incapable of burning.</li> <li>Decomposition may produce toxic fumes of:</li> <li>metal oxides</li> <li>When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain haz</li></ul>
HAZCHEM	Not Applicable
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## **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

	ith water to prevent dusting before sweeping. uitable containers for disposal.
<ul> <li>Do not of Vacuum</li> <li>Use nor</li> <li>Provide</li> <li>transfer opera</li> <li>Cover a</li> <li>Do not a</li> <li>If molten:</li> <li>Contain th</li> <li>All tooling specially c</li> <li>Allow the s</li> <li>Clear area</li> <li>Alert Fire E</li> <li>Control pe</li> <li>Prevent sp</li> <li>Avoid gend</li> <li>Sweep, sh</li> </ul>	Ind reseal partially empty containers. allow chips, fines or dusts to contact water, particularly in enclosed areas. e flow using dry sand or salt flux as a dam. (e.g., shovels or hand tools) and containers which come in contact with molten metal must be preheated or oated, rust free and approved for such use. spill to cool before remelting scrap. of personnel and move upwind. Brigade and tell them location and nature of hazard. rsonal contact with the substance, by using protective equipment and dust respirator. billage from entering drains, sewers or water courses.

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

## **SECTION 7 Handling and storage**

## Precautions for safe handling

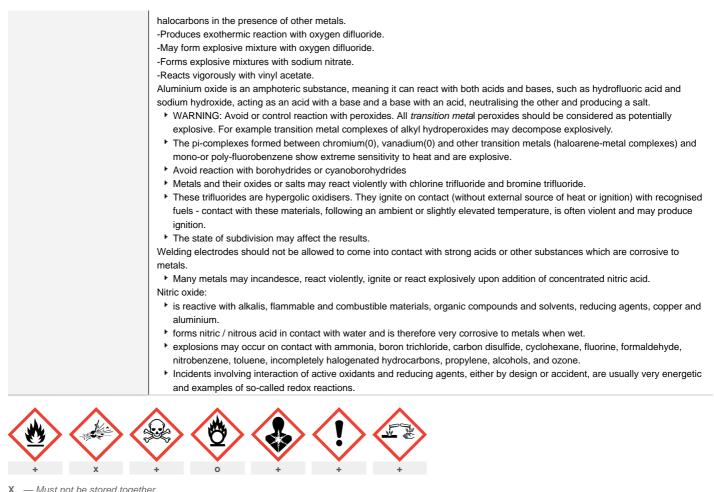
	Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	<ul> <li>Store in original containers.</li> <li>Keep containers securely sealed.</li> <li>Store in a cool, dry area protected from environmental extremes.</li> <li>Store away from incompatible materials and foodstuff containers.</li> <li>Protect containers against physical damage and check regularly for leaks.</li> <li>Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>For major quantities:</li> <li>Consider storage in bunded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams).</li> <li>Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities.</li> </ul>

## Conditions for safe storage, including any incompatibilities

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Suitable container	<ul> <li>Lined metal can, lined metal pail/ can.</li> <li>Plastic pail.</li> <li>Polyliner drum.</li> <li>Packing as recommended by manufacturer.</li> <li>Check all containers are clearly labelled and free from leaks.</li> <li>Bulk bags: Reinforced bags required for dense materials.</li> <li>CARE: Packing of high density product in light weight metal or plastic packages may result in container collapse with product release</li> </ul>
Storage incompatibility	The material is described as an electropositive metal. The activity or electronotive series of metals is a listing of the metals in decreasing order of their reactivity with hydrogen-ion sources such as water and acids. In the reaction with a hydrogen-ion source, the metal is oxidised to a metal ion, and the hydrogen ion is reduced to H2. The ordering of the activity series can be related to the standard reduction potential of a metal cation. The more positive the standard reduction potential of the cation, the more difficult it is to oxidise the metal to a hydrated metal cation and the later that metal falls in the series Three notable groups comprise the series • very electropositive metals • electropositive metals • electropositive metals Electropositive metals have electronegativities that fall between 1.4 and 1.9. Cations of these metals generally have standard reduction potentials between 0.0 and -1.6 V They: • do not react very readily with water to release hydrogen • react with H+ (acids) Electropositive metals do not burn in air as readily as do very electropositive metals. The surfaces of these metals will tarnish in the presence of oxygen forming a protective oxide coating. This coating protects the bulk of the metal against further oxidation (the metal is passivated). Reaction is reduced in the massive form (sheet, rod, or drop), compared with finely divided forms. The less active metals will not burn in air but: • can react exothermically with oxidising acids to form noxious gases. • catalyse polymerisation and other reactions, particularly when finely divided • read with Halogenated hydrocarbons (for example, copper disolves when heated in carbon tetrachloride), sometimes forming explosive compounds. • Elemental metals may react with azo/diazo compounds to form explosive products • Finely divided metal powders develop pyrophoricity when a critical specific surface area is exceeded; this is ascribed to high heat of oxids formation on exposure to air. • Saveral pyrophoric



X — Must not be stored together

- May be stored together with specific preventions 0

- 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	<ul> <li>(a) This value is for inhalable dust</li> <li>containing no asbestos and &lt; 1%</li> <li>crystalline silica.</li> </ul>
Australia Exposure Standards	chromium	Chromium (metal)	0.5 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	nickel	Nickel, metal	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

Ingredient	TEEL-1	TEEL-2		TEEL-3
chromium	1.5 mg/m3	17 mg/m3		99 mg/m3
nickel	4.5 mg/m3	50 mg/m3		99 mg/m3
Ingredient	Original IDLH		Revised IDLH	
manganese	500 mg/m3		Not Available	
silicon	Not Available		Not Available	
sulfur	Not Available		Not Available	
chromium	250 mg/m3		Not Available	
nickel	10 mg/m3		Not Available	
welding fumes	Not Available		Not Available	
welding fumes generating aluminium, copper, manganese or zinc	Not Available		Not Available	

#### **Occupational Exposure Banding**

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit	
sulfur	E	≤ 0.01 mg/m³	
welding fumes generating aluminium, copper, manganese or zinc	E	≤ 0.01 mg/m³	
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.

for welding fume:

In addition to complying with any individual exposure standards for specific contaminants, where current manual welding processes are used, the fume concentration inside the welder's helmet should not exceed 5 mg/m3, when collected in accordance with the appropriate standard (AS 3640, for example). ES\* TWA: 5 mg/m3

TLV\* TWA: 5 mg/m3, B2 (a substance of variable composition)

OES\* TWA: 5 mg/m3

Most welding, even with primitive ventilation, does not produce exposures inside the welding helmet above 5 mg/m3. That which does should be controlled (ACGIH). Inspirable dust concentrations in a worker's breathing zone shall be collected and measured in accordance with AS 3640, for example. Metal content can be analytically determined by OSHA Method ID25 (ICP-AES) after total digestion of filters and dissolution of captured metals. Sampling of the Respirable Dust fraction requires cyclone separator devices (elutriators) and procedures to comply with AS 2985 (for example).

During use the gases nitric oxide, nitrogen peroxide and ozone may be produced by the consumption of the electrode or the action of the welding arc on the atmosphere

NOTE: Detector tubes for carbon monoxide, measuring in excess of 2 ppm, are commercially available for detection of carbon monoxide.

200 ppm carbon monoxide in air will produce headache, mental dullness and dizziness in a few hours; 600 ppm will produce identical symptoms in less than half and hour and may produce unconsciousness in 1.5 hours; 4000 ppm is fatal in less than an hour.

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

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

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

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

Coburn, R.F.: Foster, R.E.: Kane, P.B.: Considerations of the Physiological Variables that Determine the Carboxyhaemoglobin Concentration in Man. J. Clin Invest. 44(1):1899-1910 (1965)

Odour Safety Factor(OSF)

OSF=0.00025 (CARBON MONOXIDE)

for ozone:

NOTE: Detector tubes for ozone, measuring in excess of 0.05 ppm, are commercially available.

Exposure at 0.2 ppm appears to produce mild acute but not cumulative effects. It is thought that exposures of the order of 0.1 ppm will be tolerated by most workers including asthmatics. Chronic exposure at 0.1 ppm or more can induce significant adverse effects in the lower respiratory tract of both normal and atopic individuals.

Human exposure for 2 hours at an average concentration of 1.5 ppm ozone resulted in a 20% reduction in timed vital capacity of the lung and other effects. Concentrations of ozone in excess of a few tenths ppm cause occasional discomfort to exposed individuals manifest as headache, dryness of the throat and mucous membranes of the eyes and nose following exposures of short duration.

Exposure to ozone during moderate to heavy work loads results in significantly decreased forced vital capacity (FVC) and forced expiratory volume in one second (FEV1) at 0.12 ppm; this is effect is greater at higher concentrations.

Odour Safety Factor(OSF)

OSF=1.1 (OZONE)

For nitric oxide:

Odour Threshold: 0.3 to 1 ppm.

NOTE: Detector tubes for nitrogen oxide, measuring in excess of 10 ppm, are commercially available.

Experimental animal date indicates that nitric oxide is one-fifth as toxic as nitrogen dioxide. The recommended TLV-TWA takes account of this relationship. Exposure at or below the recommended TLV-TWA is thought to reduce the potential for immediate injury, adverse physiological effects, pulmonary disease (including the risk of increased airway resistance) from prolonged daily exposure Odour Safety Factor (OSF)

OSF=7.7 (nitric oxide)

For silicon

CEL TWA: 5 mg/m3

(CEL = Chemwatch Exposure Limit)

NOTE: The CEL TWA is consistent with the value recommended in the Norwegian ferro-alloy industry (furnace room dust/mixed dust).

Silicon dust appears to have little adverse effect on the lungs and is not implicated in the genesis of organic disease or in the production of toxic effects. The TLV-TWA is thought to be protective against physical irritation and possible chronic respiratory effects encountered at higher levels.

For 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	<ul> <li>Engineering controls are used to remove a hazard or place a engineering controls can be highly effective in protecting worprovide this high level of protection.</li> <li>The basic types of engineering controls are:</li> <li>Process controls which involve changing the way a job activitienclosure and/or isolation of emission source which keeps at that strategically "adds" and "removes" air in the work environ designed properly. The design of a ventilation system must remployers may need to use multiple types of controls to presse source for the sum of the set of the set</li></ul>	rkers and will typically be ity or process is done to a selected hazard "physic inment. Ventilation can re natch the particular proce vent employee overexpo esult in the generation of the use of mechanical (g cubic meters per welder paces, use of mechanica	independent of worker interactions reduce the risk. ally" away from the worker and vent emove or dilute an air contaminant if ess and chemical or contaminant in sure. <sup>a</sup> aluminium, copper, fluoride, manga eneral exhaust or plenum) ventilation ) I ventilation by local exhaust system	ilation use. nese or n is s is
controls	Interview       Interview         Corrosion of aluminium)       Local exhaust systems must be designed to provide a minim metre/sec. Air contaminants generated in the workplace pos "capture velocities" of fresh circulating air required to effective         Type of Contaminant:       welding, brazing fumes (released at relatively low velocity)         Within each range the appropriate value depends on:	num capture velocity at th sess varying "escape" ve rely remove the contamin	e fume source, away from the worke elocities which, in turn, determine the	er, of 0.5
	corrosion of aluminium) Local exhaust systems must be designed to provide a minim metre/sec. Air contaminants generated in the workplace pos "capture velocities" of fresh circulating air required to effectiv Type of Contaminant: welding, brazing fumes (released at relatively low velocity	num capture velocity at th sess varying "escape" ve rely remove the contamin	e fume source, away from the worke elocities which, in turn, determine the ant. Air Speed: 0.5-1.0 m/s (100-200 f/min.)	er, of 0.5
	corrosion of aluminium) Local exhaust systems must be designed to provide a minim metre/sec. Air contaminants generated in the workplace pos "capture velocities" of fresh circulating air required to effective Type of Contaminant: welding, brazing fumes (released at relatively low velocity) Within each range the appropriate value depends on:	num capture velocity at the sess varying "escape" ve vely remove the contamin into moderately still air)	e fume source, away from the worke elocities which, in turn, determine the aant. Air Speed: 0.5-1.0 m/s (100-200 f/min.)	er, of 0.5
	corrosion of aluminium) Local exhaust systems must be designed to provide a minim metre/sec. Air contaminants generated in the workplace pos "capture velocities" of fresh circulating air required to effectiv Type of Contaminant: welding, brazing fumes (released at relatively low velocity Within each range the appropriate value depends on: Lower end of the range	num capture velocity at th sess varying "escape" ve rely remove the contamin into moderately still air) Upper end of the rang	e fume source, away from the worke elocities which, in turn, determine the aant. Air Speed: 0.5-1.0 m/s (100-200 f/min.) e currents	er, of 0.5

4: Large hood or large air mass in motion		
	4: Small hood-local co	ntrol only
Simple theory shows that air velocity falls rapidly with distance generally decreases with the square of distance from the ext extraction point should be adjusted, accordingly, after referent extraction fan, for example, should be a minimum of 1-2 m/s generated 2 meters distant from the extraction point. Other r extraction apparatus, make it essential that theoretical air ve systems are installed or used. Engineering controls are used to remove a hazard or place a engineering controls can be highly effective in protecting wor provide this high level of protection.	traction point (in simple c nce to distance from the c (200-400 f/min.) for extra mechanical consideration elocities are multiplied by a barrier between the wo	ases). Therefore the air speed at the contaminating source. The air velocity at action of welding or brazing fumes is, producing performance deficits within factors of 10 or more when extraction rker and the hazard. Well-designed
The basic types of engineering controls are:		
Process controls which involve changing the way a job activi Enclosure and/or isolation of emission source which keeps a that strategically "adds" and "removes" air in the work enviro designed properly. The design of a ventilation system must r Employers may need to use multiple types of controls to pre Special ventilation requirements apply for processes which r in those processes which generate ozone. The use of mechanical ventilation by local exhaust systems	a selected hazard "physic onment. Ventilation can re match the particular proce vent employee overexpo result in the generation of	ally" away from the worker and ventilatic emove or dilute an air contaminant if ess and chemical or contaminant in use. sure. f barium, chromium, lead, or nickel fume
work). (In confined spaces always check that oxygen has no		
of aluminium) Local exhaust systems must be designed to provide a minim	num capture velocity at th	e fume source, away from the worker, of
metre/sec. Air contaminants generated in the workplace pos "capture velocities" of fresh circulating air required to effective		
Type of Contaminant:		Air Speed:
		0.5-1.0 m/s
welding, brazing fumes (released at relatively low velocity	into moderately still air)	(100-200 f/min.)
Within each range the appropriate value depends on:		
Lower end of the range	Upper end of the rang	e
1: Room air currents minimal or favourable to capture	1: Disturbing room air	currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of hig	gh toxicity
3: Intermittent, low production.	3: High production, he	avy use
4: Large hood or large air mass in motion Simple theory shows that air velocity falls rapidly with distance	4: Small hood-local co ce away from the opening	ntrol only g of a simple extraction pipe. Velocity
4: Large hood or large air mass in motion	4: Small hood-local co ce away from the openin traction point (in simple c nce to distance from the s (200-400 f/min.) for extra mechanical consideration	ntrol only g of a simple extraction pipe. Velocity ases). Therefore the air speed at the contaminating source. The air velocity at action of welding or brazing fumes is, producing performance deficits within
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Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture

	velocities" of fresh circulating air required to effectively remov	ve the contaminant.	
	Type of Contaminant:		Air Speed:
	welding, brazing fumes (released at relatively low velocity i	nto 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	2
	1: Room air currents minimal or favourable to capture	1: Disturbing room air of	currents
	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of hig	h toxicity
	3: Intermittent, low production.	3: High production, hea	avy use
	4: Large hood or large air mass in motion	4: Small hood-local cor	ntrol only
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Personal protection			
Eye and face protection	<ul> <li>Goggles or other suitable eye protection shall be used during all gas welding or oxygen cutting operations. Spectacles without side shields, with suitable filter lenses are permitted for use during gas welding operations on light work, for torch brazing or for inspection.</li> <li>For most open welding/brazing operations, goggles, even with appropriate filters, will not afford sufficient facial protection for operators. Where possible use welding helmets or handshields corresponding to EN 175, ANSI Z49:12005, AS 1336 and AS 1338 which provide the maximum possible facial protection from flying particles and fragments. [WRIA-WTIA Technical Note 7]</li> <li>An approved face shield or welding helmet can also have filters for optical radiation protection, and offer additional protection against debris and sparks.</li> <li>UV blocking protective spectacles with side shields or welding goggles are considered primary protection, with the face shield or welding helmet considered secondary protection.</li> <li>The optical filter in welding goggles, face mask or helmet must be a type which is suitable for the sort of work being done.A filter suitable for gas welding, for instance, should not be used for arc welding.</li> <li>Face masks which are self dimming are available for arc welding, MIG, TIG and plasma cutting, and allow better vision before the arc is struck and after it is extinguished.</li> </ul>		
Skin protection	See Hand protection below		
Hands/feet protection	The selection of suitable gloves does not only depend on the manufacturer to manufacturer. Where the chemical is a prep can not be calculated in advance and has therefore to be che The exact break through time for substances has to be obtai observed when making a final choice. Personal hygiene is a key element of effective hand care. Gl should be washed and dried thoroughly. Application of a non Suitability and durability of glove type is dependent on usage frequency and duration of contact, chemical resistance of glove material, glove thickness and dexterity Select gloves tested to a relevant standard (e.g. Europe EN dexterity When prolonged or frequently repeated contact may of time greater than 240 minutes according to EN 374, AS/NZS When only brief contact is expected, a glove with a prominutes according to EN 374, AS/NZS 2161.10.1 or national Some glove polymer types are less affected by movem for long-term use.	aration of several substance ecked prior to the applicance ned from the manufacture oves must only be worn of -perfumed moisturiser is a Important factors in the 374, US F739, AS/NZS 2 ccur, a glove with a prote 2161.10.1 or national eco otection class of 3 or high equivalent) is recommer nent and this should be ta	nces, the resistance of the glove material tion. er of the protective gloves and has to be on clean hands. After using gloves, hands recommended. selection of gloves include: 2161.1 or national equivalent). ection class of 5 or higher (breakthrough quivalent) is recommended. her (breakthrough time greater than 60 nded.

	<ul> <li>should also be based on consideration of the task requirements and knowledge of breakthrough times.</li> <li>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</li> <li>Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.</li> <li>Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential</li> <li>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.</li> <li>Protective gloves eq. Leather gloves or gloves with Leather facing</li> <li>Welding gloves conforming to Standards such as EN 12477:2001, ANSI Z49.1, AS/NZS 2161:2008 produced from leather, rubber, treated cotton, or alumininised</li> <li>These gloves which protect against mechanical risk caused by abrasion, blade cut, tear and puncture</li> <li>Other gloves mentioned above.</li> <li>One pair of gloves mentioned above.</li> <li>Done pair of gloves mentioned above.</li> <li>Done pair of gloves mentioned above.</li> <li>Experience indicates that the following polymers are suitable as glove materials for protection agains</li></ul>
	<ul> <li>polyvinyl chloride.</li> <li>Gloves should be examined for wear and/ or degradation constantly.</li> </ul>
Body protection	See Other protection below
Other protection	Before starting; consider that protection should be provided for all personnel within 10 metres of any open arc welding operation. Welding sites must be adequately shielded with screens of non flammable materials. Screens should permit ventilation at floor and ceiling levels.

## **Respiratory protection**

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

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

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	@1@ P2	-	-
	Air-line*	-	-
up to 50 x ES	Air-line**	@1@ P2	@1@ PAPR-P2
	-	Air-line*	-
up to 100 x ES	-	Air-line**	@1@ PAPR-P3

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

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

Welding of powder coated metal requires good general area ventilation, and ventilated mask as local heat causes minor coating decomposition releasing highly discomforting fume which may be harmful if exposure is regular.

Welding or flame cutting of metals with chromate pigmented primers or coatings may result in inhalation of highly toxic chromate fumes. Exposures may be significant in enclosed or poorly ventilated areas

## **SECTION 9** Physical and chemical properties

## Information on basic physical and chemical properties

Appearance	Silver		
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
Chemical stability	Product is considered stable and 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

	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 contro measures be used in an occupational setting.
	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.
Inhaled	Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following remeval from expression
	removal from exposure. Not normally a hazard due to non-volatile nature of product Acute carbon monoxide exposure can mimic acute gastroenteritis or food poisoning with accompanying nausea and vomiting. Rapidly fatal cases of poisoning are characterised by congestion and hemorrhages in all organs. The extent of the tissue and organ damage is related to the duration of the post-hypoxic unconsciousness. Exposure to carbon monoxide can result in immediate effects and, depending on the severity of the exposure, delayed effects. These delayed effects may occur days to

	weeks after the initial exposure. Signs of brain or nerve injury may appear at any time within three weeks following an acute exposure. Characteristically, those patients manifesting delayed neuropathology are middle aged or older. Most of the neurological symptoms associated with carbon monoxide exposure can resolve within a year but memory deficits and gait disturbances may remain Symptoms of poisoning resulting from carbon monoxide exposure include respiratory disorders, diarrhoea and shock. Carbon monoxide competes with oxygen for haemoglobin binding sites and has a 240-fold affinity for these sites compared to oxygen. In addition to oxygen deficiency further disability is produced by the formation of carboxymyoglobin (COHb) in muscles, to produce disturbances in muscle metabolism, particularly that of the heart. The toxygen 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 carebral oedema and increased intracranial pressure resulting from excessive transudate leakage of fluids through the hypoxic capillaries. Carbon monoxide may cause poor concentration, memory and vision problems, vertigo, muscular weakness and loss of muscle cordination, rapid and stretorous breathing, intermittent heart beat, loss of sphincter control and rarely coma and death. At higher levels (200 pm for 2-3 hours), it may cause headaches, fatigue and nausea. At very high levels (400 ppm) the symptoms intensify and will be life-threatening after three hours. Exposure to levels of 1200 ppm or greater are immediately dangerous to life. When carbon monoxide levels in air exceed 3% (30,000 ppm), death occurs almost at once. Carbon monoxide in the blood in sedentary adults is 2-5 hours and the elimination becomes slower as the concentration decreases. Manganese fume is toxic and produces nervous system effects characterised by tiredness. Acute poisoning is rare although acute inflammation of the lungs may occur. A ch
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 <b>NOT</b> been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern. 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 sy

required as a minimum. (Open work spaces exceed 300 cubic meters per welder)

For indoor work, conducted in limited or confined work spaces, use of mechanical ventilation by local exhaust systems is mandatory. (In confined spaces always check that oxygen has not been depleted by excessive rusting of steel or snowflake corrosion of aluminium)

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

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

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

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

Chrome fume, as the chrome VI oxide, is corrosive to the skin and may aggravate pre-existing skin conditions such as dermatitis and eczema. As a potential skin sensitiser, the fume may cause dermatoses to appear suddenly and without warning. Absorption of chrome VI compounds through the skin can cause systemic poisoning effecting the kidneys and liver. 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.

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.

Eve

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.

Long-term exposure to the product is not thought to produce chronic effects adverse to health (as classified by EC Directives using animal models); nevertheless exposure by all routes should be minimised as a matter of course.

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.

Chronic

the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity.

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

There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveolii (sub 5 um) are able to produce pathogenic effects in the lungs. Repeated or prolonged exposure may also damage the liver and may cause a decrease in the heart rate. Systemic poisoning may result from inhalation or chronic ingestion of manganese containing substances. Progressive and permanent disability can occur from chronic manganese poisoning if it is not treated, but it is not fatal.

Chronic exposure has been associated with two major effects; bronchitis/pneumonitis following inhalation of manganese dusts

and "manganism", a neuropsychiatric disorder that may also arise from inhalation exposures. Chronic exposure to low levels may result in the accumulation of toxic concentrations in critical organs. The brain in particular appears to sustain cellular damage to the ganglion. Symptoms appear before any pathology is evident and may include a mask-like facial expression, spastic gait, tremors, slurred speech, sometimes dystonia (disordered muscle tone), fatigue, anorexia, asthenia (loss of strength and energy), apathy and the inability to concentrate. Insomnia may be an early finding. Chronic poisoning may occur over a 6-24 month period depending on exposure levels.

The onset of chronic manganese poisoning is insidious, with apathy, anorexia weakness, headache and spasms. Manganese psychosis follows with certain definitive features: unaccountable laughter, euphoria, impulsive acts, absentmindedness, mental confusion, aggressiveness and hallucinations. The final stage is characterised by speech difficulties, muscular twitching, spastic gait and other nervous system effects. Symptoms resemble those of Parkinson's disease. Rat studies indicate the gradual accumulation of brain manganese to produce lesions mimicking those found in Parkinsonism. If the disease is diagnosed whilst still in the early stages and the patient is removed from exposure, the course may be reversed.

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

Prolonged or repeated eye contact may result in conjunctivitis.

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

Long-term (chronic) exposure to low levels of carbon monoxide may produce heart disease and damage to the nervous system. Exposure of pregnant animals to carbon monoxide may cause low birthweight, increased foetal mortality and nervous system damage to the offspring.

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

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

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

Carbon monoxide gas readily crosses the placenta and CO exposure during pregnancy can be teratogenic.

Carbon dioxide at low levels may initiate or enhance deleterious myocardial alterations in individuals with restricted coronary artery blood flow and decreased myocardial lactate production. - Linde

Chromium(III) is considered an essential trace nutrient serving as a component of the "glucose tolerance factor" and a cofactor for insulin action. High concentrations of chromium are also found in RNA. Trivalent chromium is the most common form found in nature.

Chronic inhalation of trivalent chromium compounds produces irritation of the bronchus and lungs, dystrophic changes to the liver and kidney, pulmonary oedema, and adverse effects on macrophages. Intratracheal administration of chromium(III) oxide, in rats, increased the incidence of sarcomas, and tumors and reticulum cell sarcomas of the lung. There is inadequate evidence of carcinogenicity of chromium(III) compounds in experimental animals and humans (IARC).

Chronic exposure to hexavalent chromium compounds reportedly produces skin, eye and respiratory tract irritation, yellowing of the eyes and skin, allergic skin and respiratory reactions, diminished sense of smell and

taste, blood disorders, liver and kidney damage, digestive disorders and lung damage. There is sufficient evidence of carcinogenicity of chromium(VI) compounds in experimental animals and humans to confirm these as Class 1 carcinogens (IARC).

Exposure to chromium during chrome production and in the chrome pigment industry is associated with cancer of the respiratory tract. A slight increase in gastrointestinal cancer following exposure to chromium compounds has also been reported. The greatest risk is attributed to exposure to acid-soluble, water-insoluble hexavalent chromium which occurs in roasting and refining processes. Animal studies support the idea that the most potent carcinogenic compounds are the slightly soluble hexavalent compounds. The cells are more active in the uptake of the hexavalent forms compared to trivalent forms and this may explain the difference in occupational effect. It is the trivalent form, however, which is metabolically active and binds with nucleic acid within the cell suggesting that chromium mutagenesis first requires biotransformation of the hexavalent form by reduction.

Hexavalent chromes produce chronic ulceration of skin surfaces (quite independent of other hypersensitivity reactions exhibited by the skin). Water-soluble chromium(VI) compounds come close to the top of any published "hit list" of contact allergens (eczematogens) producing positive results in 4 to 10% of tested individuals. On the other hand only chromium(III) compounds can bind to high molecular weight carriers such as proteins to form a complete allergen (such as a hapten). Chromium(VI) compounds cannot. It is assumed that reduction must take place for such compounds to manifest any contact sensitivity. The apparent contradiction that chromium(VI) salts cause allergies to chromium(III) compounds but that allergy to chromium(III) compounds is difficult to demonstrate is accounted for by the different solubilities and skin penetration of these compounds. Water-soluble chromium(VI) salts penetrate the horny layer of the skin more readily than chromium(III) compounds which are bound by cross-linking in the horny layer ("tanning", as for leather) and therefore do not reach the cells involved in antigen processing.

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.

Available ICITY ation(Rat) LC50; >5.14 mg/l4h <sup>[1]</sup> Rat) LD50; >2000 mg/kg <sup>[1]</sup>	Not Available         IRRITATION         Eye (rabbit): 500 mg/24h - mild         Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin (rabbit): 500 mg/24h - mild
ation(Rat) LC50; >5.14 mg/l4h <sup>[1]</sup>	Eye (rabbit): 500 mg/24h - mild Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
Rat) LD50; >2000 mg/kg <sup>[1]</sup>	
	Skin (rabbit): 500 mg/24h - mild
	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
ICITY	IRRITATION
nal (rabbit) LD50: >5000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
Rat) LD50; >5000 mg/kg <sup>[1]</sup>	Skin: no adverse effect observed (not irritating) $^{\left[ 1\right] }$
ICITY	IRRITATION
al (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye (human): 8 ppm irritant
ation(Rat) LC50; >5.43 mg/L4h <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
Rat) LD50; >2000 mg/kg <sup>[1]</sup>	Skin: adverse effect observed (irritating) <sup>[1]</sup>
	Skin: no adverse effect observed (not irritating) $^{\left[ 1\right] }$
ICITY	IRRITATION
ation(Rat) LC50; >5.41 mg/l4h <sup>[1]</sup>	Not Available
Rat) LD50; >5000 mg/kg <sup>[1]</sup>	
ICITY	IRRITATION
Rat) LD50; >9000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Skin: no adverse effect observed (not irritating) $^{\left[ 1\right] }$
ICITY	IRRITATION
Available	Not Available
ICITY	IRRITATION
Available	Not Available
	nal (rabbit) LD50: >5000 mg/kg <sup>[1]</sup> Rat) LD50; >5000 mg/kg <sup>[1]</sup> ICITY         nal (rat) LD50: >2000 mg/kg <sup>[1]</sup> ation(Rat) LC50; >5.43 mg/L4h <sup>[1]</sup> Rat) LD50; >2000 mg/kg <sup>[1]</sup> ICITY         ation(Rat) LC50; >5.41 mg/l4h <sup>[1]</sup> Rat) LD50; >5000 mg/kg <sup>[1]</sup> ICITY         ation(Rat) LC50; >5.41 mg/l4h <sup>[1]</sup> Rat) LD50; >5000 mg/kg <sup>[1]</sup> ICITY         Rat) LD50; >9000 mg/kg <sup>[1]</sup> ICITY         Nailable         ICITY

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.

## BW-308LP, BW-309LP Stainless Steel Flux Cored Mig Wire

SILICON	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 ceases. The disorder is characterised by dyspnea, cough and mucus production. Intraperitoneal injection of silicon produced only minor local trauma and foreign body reaction. Parenterally administered elemental silica is considered biologically inert. 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. Normal human cerebral cortex tissue contains about 3.8 ug/g silicon
СНКОМІИМ	Gastroinestinal tumours, lymphoma, musculoskeletal tumours and tumours at site of application recorded. For chrome(III) and other valence states (except hexavalent): For inhalation exposure, all trivalent and other chromium compounds are treated as particulates, not gases. The mechanisms of chromium toxicity are very complex, and although many studies on chromium are available, there is a great deal of uncertainy about how chromium exercits 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 and use, and chrome plating) conclude that wille occupational exposure to hexavalent chromium compounds is associated with an increased risk of respiratory system cancers (primarily bronchogenic and nasa), results from occupational exposure studies to mixtures that were mainly elemental and trivalent (flerochromium alloy worker) were inconclusive. Studies in leafther tanners, who were exposed to trivalent chromium were consistently negative. In addition to the lack of direct evidence of carcinogenicity 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 thvalent chromium comparison to absorption of hexavalent chromium sompounds. Hexavalent chromium compounds exist as tetrahedral chromate anions, resembling the forms of other natur
NICKEL	Oral (rat) TDLo: 500 mg/kg/5D-I Inhalation (rat) TCLo: 0.1 mg/m3/24H/17W-C The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen

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## BW-308LP, BW-309LP Stainless Steel Flux Cored Mig Wire

	[National Toxicology Program: U.S. Dep. of Health & Human Services 2002]
BW-308LP, BW-309LP Stainless Steel Flux Cored Mig Wire & NICKEL	WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
BW-308LP, BW-309LP Stainless Steel Flux Cored Mig Wire & WELDING FUMES & WELDING FUMES GENERATING ALUMINIUM, COPPER, MANGANESE OR ZINC	Most welding is performed using electric arc processes - manual metal arc, metal inert gas (MG) and tungsten inert gas welding (TIG) - and most welding is on mild steel. In 2017, an IACC working group has determined that *sufficient evidence exists that welding furme is a human lung carcinogen (Group 1). A complicating factor in classifying welding furnes is its complexity. Generally, welding furne is a mixture of metal Humes (i.e., iron, manganese, chromium, nickel, silicon, titanium) and gases (i.e., carton monoxide, czone, argon, carton dioxide). Welding furne can contain vanje over the presence of such metals and the intensity of exposure to each differ significantly according to a number of varianging concentrations of individual components that are of the composition of the base metal and consumable. Nonetheless, IAAC did not differentiate between these variables in its decision. There has been considerable evidence over serveral gas velding phorements of turnes of torium-232, which is used in tungschen welding cartor fields. This association may be due to the presence in some welding environments of furnes of torium-232, which is used in tungschen welding cartor discolar discolar evidence over as everal association may be due to the presence in some welding environments of unes of torium-232, which used short ugaroaction. This widespread consensus is in part based on empirical evidence regarding risks among stalless steal welding, should not carry risk. But it appears that this line of reasoning in not supported by the accompands, which are recognised tung carcor, thisk, the vertexile million evidence is a saryet unoxplane, and welders and in part on the fact that stainless steel welding existent and scenes line (VV) radiation. There has been a necess risk of lung carror risks and parts welding schould not carry risk. But it appears that this line of reasoning in not supported by the accompands, which are proceeds to a range of toxiel and chromium VI compounds, namely mild steel welding, should not carry
MANGANESE & SILICON	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
SILICON & CHROMIUM	No significant acute toxicological data identified in literature search.
WELDING FUMES & WELDING FUMES GENERATING ALUMINIUM,	WARNING: This substance has been classified by the IARC as Group 1: CARCINOGENIC TO HUMANS. Not available. Refer to individual constituents.

COPPER, MANGANESE OR ZINC			
Acute Toxicity	✓	Carcinogenicity	✓
Skin Irritation/Corrosion	×	Reproductivity	×
Serious Eye Damage/Irritation	×	STOT - Single Exposure	×
Respiratory or Skin sensitisation	¥	STOT - Repeated Exposure	~
Mutagenicity	×	Aspiration Hazard	×

✓ – Data available to make classification

## **SECTION 12 Ecological information**

BW-308LP, BW-309LP	Endpoint	Test Duration (hr)		Species		Value	Source
Stainless Steel Flux Cored Mig Wire	Not Available	Not Available		Not Available		Not Available	Not Available
	Endpoint	Test Duration (hr)		Species		Value	Source
	NOEC(ECx)	504h		Algae or other aquatic plants		0.05-3.7mg/l	4
manganese	EC50	72h Algae or othe		Algae or other aquatic plants	or other aquatic plants 2.8mg		2
	LC50	96h		Fish		>3.6mg/l	2
	EC50	48h		Crustacea		>1.6mg/l	2
	Endpoint	Test Duration (hr)		Species		Value	Source
silicon	EC10(ECx)	1.28h		Algae or other aquatic plants		>=66<=88mg/l	2
	EC50	72h		Algae or other aquatic plants		~250mg/l	2
	Endpoint	Test Duration (hr)		Species		Value	Source
sulfur	NOEC(ECx)	504h		Crustacea		>100mg/l	2
	LC50	96h		Fish		>207mg/L	4
	Endpoint	Test Duration (hr)	S	pecies	Va	alue	Source
	EC50(ECx)	48h	С	Crustacea	<(	0.001mg/l	2
	EC50	72h	A	lgae or other aquatic plants	0.	026-0.208mg/L	4
chromium	LC50	96h	F	ïsh	0.	106mg/L	4
	EC50	48h	С	Crustacea	<(	).001mg/l	2
	EC50	96h	A	Algae or other aquatic plants 36mg/L		Smg/L	4
	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
nickel	LC50	96h		Fish		0.168mg/L	4
	EC50	48h		Crustacea		>100mg/l	1
	EC50	96h		Algae or other aquatic plants		0.36mg/l	2
welding fumes	Endpoint	Test Duration (hr)		Species		Value	Source
	Not Available	Not Available		Not Available		Not Available	Not Availabl
welding fumes generating	Endpoint	Test Duration (hr)		Species		Value	Source
aluminium, copper, manganese or zinc	Not Available	Not Available		Not Available		Not Available	Not Available

Legend:

Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5.

ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8.
 Vendor Data

Toxic 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 manginude, 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 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.

carbonate (CO3.2), 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 muscles, and 35 - 930 for coastal fish. Similarly, the BCF of manganese was estimated to be 10,00 -20,000 for marine and freshwater plants, 10,000 - 40,000 for invertebrates, and 10 - 600 for fish. In general, these data indicate that lower organisms such as algae have larger BCFs than higher organisms. In order to protect consumers from the risk of manganese bioaccumulation in marine mollusks, the U.S. EPA has set a criterion for manganese at 0.1 mg/L for marine waters.

Elemental manganese and inorganic manganese compounds have negligible vapor pressures but may exist in air as suspended particulate matter derived from industrial emissions or the erosion of soils. Manganese-containing particles are mainly removed from the atmosphere by gravitational settling, with large particles tending to fall out faster than small particles. The half-life of airborne particles is usually on the order of days, depending on the size of the particle and atmospheric conditions. Some removal by washout mechanisms such as rain may also occur, although it is of minor significance in comparison to dry deposition. **Ecotoxicity:** 

Manganese ion is toxic to aqueous organisms Fish LC50 (28 d): orfe 2490 mg/l, trout 2.91 mg/l Daphnia magna LC50: 50 mg/l Pseudomonas putida LC50: 10.6 mg/l Photobacterium phosphoreum LC50: 14.7 mg/l Turbellarian worms (EC0): Polycelis nigra 660 mg/l (interference threshold); microregma 31 mg/l

Chromium in the oxidation state +3 (the trivalent form) is poorly absorbed by cells found in microorganisms, plants and animals. Chromate anions (CrO4-, oxidation state +6, the hexavalent form) are readily transported into cells and toxicity is closely linked to the higher oxidation state.

Chromium Ecotoxicology:

Chromium is harmful to aquatic organisms in very low concentrations. Fish food organisms are very sensitive to low levels of chromium. Chromium is toxic to fish although less so in warm water. Marked decreases in toxicity are found with increasing pH or water hardness; changes in salinity have little if any effect. Chromium appears to make fish more susceptible to infection. High concentrations can damage and/or accumulate in various fish tissues and in invertebrates such as snails and worms.

Reproduction of Daphnia is affected by exposure to 0.01 mg/kg hexavalent chromium/litre

Toxicity of chromium	in fresh-water organism	s (50% mortality)*
Toxiony of ornorman	in noon mater organion	o (oo /o monunty)

Compound	Category	Exposure	Toxicity Range (mg/litre)	Most sensitive species
hexavalent chrome	invertebrate	acute	0.067-59.9	scud
		long-term	-	-
	vertebrate	acute	17.6-249	fathead minnow
		long-term	0.265-2.0	rainbow trout
trivalent chrome	invertebrate	acute	2.0-64.0	cladoceran
		long-term	0.066	cladoceran
	vertebrate	acute	33.0-71.9	guppy
	invertebrate	long-term	1.0	fathead minnow
	inventebrate	long term	1.0	iuncuu minnow

\* from Environmental Health Criteria 61: WHO Publication.

#### Toxicity in Microorganisms:

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

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

Biological half-life: The elimination curve for chromium, as measured by whole-body counting, has an exponential form. In rats, three different components of the curve have been identified, with half-lives of 0.5, 5.9 and 83.4 days, respectively.

Water Standards: Chromium is identified as a hazardous substance in the Federal (U.S.) Water Pollution Control Act and further regulated by Clean Air Water Act Amendments (US). These regulations apply to discharge. The US Primary drinking water Maximum Contaminant Level (MCL), for chromium, is 0.05 mg/l (total chromium).

Since chromium compounds cannot volatilize from water, transport of chromium from water to the atmosphere is not likely, except by transport in windblown sea sprays. Most of the chromium released into water will ultimately be deposited in the sediment. A very small percentage of chromium can be present in water in both soluble and insoluble forms. Soluble chromium generally accounts for a very small percentage of the total chromium. Most of the soluble chromium is present as chromium(VI) and soluble chromium(III) complexes. In the aquatic phase, chromium(III) occurs mostly as suspended solids adsorbed onto clayish materials, organics, or iron oxide (Fe2O3) present in water. Soluble forms and suspended chromium can undergo intramedia transport. Chromium(VI) in water will eventually be reduced to chromium(III) by organic matter in the water.

The reduction of chromium(VI) and the oxidation of chromium(III) in water has been investigated. The reduction of chromium(VI) by S-2 or Fe+2 ions under anaerobic conditions was fast, and the reduction half-life ranged from instantaneous to a few days. However, the reduction of chromium(VI) by organic sediments and soils was much slower and depended on the type and amount of organic material and on the redox condition of the water. The reaction was generally faster under anaerobic than aerobic conditions. The reduction half-life of chromium(VI) in water with soil and sediment ranged from 4 to 140 day. Dissolved oxygen by itself in natural waters did not cause any measurable oxidation of chromium(III) to chromium(VI) in 128 days. When chromium(III) was added to lake water, a slow oxidation of chromium(III) to chromium(VI) occurred, corresponding to an oxidation half-life of nine years. The oxidation of chromium(III) to chromium(VI) during chlorination of water was highest in the pH range of 5.5?6.0. However, the process would rarely occur during chlorination of drinking water because of the low concentrations of chromium(III) in these waters, and the presence of naturally occurring organics that may protect chromium(III) from oxidation, either by forming strong complexes with chromium(III) or by acting as a reducing agent to free available chlorine.

The bioconcentration factor (BCF) for chromium(VI) in rainbow trout (Salmo gairdneri) is 1. In bottom feeder bivalves, such as the oyster (Crassostrea virginica), blue mussel (Mytilus edulis), and soft shell clam (Mya arenaria), the BCF values for chromium(III) and chromium(VI) may range from 86 to 192. The bioavailability of chromium(III) to freshwater invertebrates (Daphnia pulex) decreased with the addition of humic acid. This decrease in bioavailability was attributed to lower availability of the free form of the metal due to its complexation with humic acid. Based on this information, chromium is not expected to biomagnify in the aquatic food chain. Although higher concentrations of chromium have been reported in plants growing in high chromium-containing soils (e.g., soil near ore deposits or chromium-emitting industries and soil fertilized by sewage sludge) compared with plants growing in normal soils, most of the increased uptake in plants is retained in roots, and only a small fraction is translocated in the aboveground part of edible plants. Therefore, bioaccumulation of chromium from soil

to above-ground parts of plants is unlikely. There is no indication of biomagnification of chromium along the terrestrial food chain (soil-plant-animal). The fate of chromium in soil is greatly dependent upon the speciation of chromium, which is a function of redox potential and the pH of the soil. In most soils, chromium will be present predominantly in the chromium(III) state. This form has very low solubility and low reactivity resulting in low mobility in the environment and low toxicity in living organisms. Under oxidizing conditions chromium(VI) may be present in soil as CrO4?2 and HCrO4-. In this form, chromium is relatively soluble, mobile, and toxic to living organisms. In deeper soil where anaerobic conditions exist, chromium(VI) will be reduced to chromium(III) by S-2 and Fe+2 present in soil. The reduction of chromium(VI) to chromium(III) is possible in aerobic soils that contain appropriate organic energy sources to carry out the redox

reaction. The reduction of chromium(VI) to chromium(III) is facilitated by low pH. From thermodynamic considerations, chromium(VI) may exist in the aerobic zone of some natural soil. The oxidation of chromium(III) to chromium(VI) in soil is facilitated by the presence of low oxidisable organic substances, oxygen, manganese dioxide, and moisture. Organic forms of chromium(III) (e.g., humic acid complexes) are more easily oxidised than insoluble oxides. Because most chromium(III) in soil is immobilized due to adsorption and complexation with soil materials, the barrier to this oxidation process is the lack of availability of mobile chromium(III) to immobile manganese dioxide in soil surfaces. Due to this lack of availability of mobile chromium(III) to manganese dioxide surfaces, a large portion of chromium in soil will not be oxidized to chromium(VI), even in the presence of manganese dioxide and favorable pH conditions.

The microbial reduction of chromium(VI) to chromium(III) has been discussed as a possible remediation technique in heavily contaminated environmental media or wastes. Factors affecting the microbial reduction of chromium(VI) to chromium(III) include biomass concentration, initial chromium(VI) concentration, temperature, pH, carbon source, oxidation-reduction potential and the presence of both oxyanions and metal cations. Although high levels of chromium(VI) are toxic to most microbes, several resistant bacterial species have been identified which could ultimately be employed in remediation strategies

Chromium in soil is present mainly as insoluble oxide Cr2O3. nH2O, and is not very mobile in soil. A leachability study was conducted to study the mobility of chromium in soil. Due to different pH values, a complicated adsorption process was observed and chromium moved only slightly in soil.

Chromium was not found in the leachate from soil, possibly because it formed complexes with organic matter. These results support previous data finding that chromium is not very mobile in soil. These results are supported by leachability investigation in which chromium mobility was studied for a period of 4 years in a sandy loam. The vertical migration pattern of chromium in this soil indicated that after an initial period of mobility, chromium forms insoluble complexes and little leaching is observed. Flooding of soils and the subsequent anaerobic decomposition of plant detritus matters may increase the mobilization of chromium(III) in soils due to formation of soluble complexes. This complexation may be facilitated by a lower soil pH. A smaller percentage of total chromium in soil exists as soluble chromium(VI) and chromium(III), which are more mobile in soil. The mobility of soluble chromium is soil will depend on the sorption characteristics of the soil. The relative retention of metals by soil is in the order of lead > antimony > copper > chromium > zinc > nickel > cobalt > cadmium. The sorption of chromium to soil depends primarily on the clay content of the soil and, to a lesser extent, on Fe2O3 and the organic content of soil. Chromium that is irreversibly sorbed onto soil, for example, in the interstitial lattice of geothite, FeOOH, will not be bioavailable to plants and animals under any condition. Organic matter in soil is expected to convert soluble chromate, chromium(VI), to insoluble chromium(III) oxide, Cr2O3. Chromium in soil may be transported to the atmosphere as an aerosol. Surface runoff from soil can transport both soluble and bulk precipitate of chromium to surface water. Soluble and unadsorbed chromium(VI) and chromium(III) complexes in soil may leach into groundwater. The leachability of chromium(VI) compounds in soil.

Chromium has a low mobility for translocation from roots to aboveground parts of plants. However, depending on the geographical areas where the plants are grown, the concentration of chromium in aerial parts of certain plants may differ by a factor of 2?3.

In the atmosphere, chromium(VI) may be reduced to chromium(III) at a significant rate by vanadium (V2+, V3+, and VO2+), Fe2+, HSO3-, and As3+. Conversely, chromium(III), if present as a salt other than Cr2O3, may be oxidized to chromium(VI) in the atmosphere in the presence of at least 1% manganese oxide.. However, this reaction is unlikely under most environmental conditions. The estimated atmospheric half-life for chromium(VI) reduction to chromium(III) was reported in the range of 16 hours to about 5 days

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.

## Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
sulfur	LOW	LOW

#### **Bioaccumulative potential**

Ingredient	Bioaccumulation
sulfur	LOW (LogKOW = 0.229)

## Mobility in soil

Ingredient	Mobility
sulfur	LOW (KOC = 14.3)

#### **SECTION 13 Disposal considerations**

Waste treatment methods	
Product / Packaging disposal	<ul> <li>Recycle wherever possible or consult manufacturer for recycling options.</li> <li>Consult State Land Waste Management Authority for disposal.</li> <li>Bury residue in an authorised landfill.</li> <li>Recycle containers if possible, or dispose of in an authorised landfill.</li> </ul>

#### **SECTION 14 Transport information**

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
sulfur	Not Available
chromium	Not Available
nickel	Not Available
phosphorus	Not Available
welding fumes	Not Available
welding fumes generating aluminium, copper, manganese or zinc	Not Available

## Transport in bulk in accordance with the ICG Code

Product name	Ship Type
manganese	Not Available
silicon	Not Available
sulfur	Not Available
chromium	Not Available
nickel	Not Available
phosphorus	Not Available
welding fumes	Not Available
welding fumes generating aluminium, copper, manganese or zinc	Not Available

## **SECTION 15 Regulatory information**

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

manganese is found on the following regulatory lists	
Australia Hazardous Chemical Information System (HCIS) - Hazardous	Australian Inventory of Industrial Chemicals (AIIC)
Chemicals	
silicon is found on the following regulatory lists	
Australian Inventory of Industrial Chemicals (AIIC)	
sulfur is found on the following regulatory lists	
Australia Hazardous Chemical Information System (HCIS) - Hazardous	Australian Inventory of Industrial Chemicals (AIIC)
Chemicals	
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

nickel is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous	International Agency for Research on Cancer (IARC) - Agents Classified by
Chemicals	the IARC Monographs
Australian Inventory of Industrial Chemicals (AIIC)	International Agency for Research on Cancer (IARC) - Agents Classified by
Chemical Footprint Project - Chemicals of High Concern List	the IARC Monographs - Group 2B: Possibly carcinogenic to humans
welding fumes is found on the following regulatory lists	
Internetional Anomaly for Dessarsh on Conserv(IADO) Anomate Classified by	Internetional Assess for December (IADC) Assets Classified by

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

#### **National Inventory Status**

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (manganese; silicon; sulfur; chromium; nickel; phosphorus)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (manganese; silicon; sulfur; chromium; nickel; phosphorus)
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:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

## **SECTION 16 Other information**

Revision Date	18/08/2021
Initial Date	18/08/2021

## 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

Classification of the preparation and its individual components has drawn on official and authoritative sources using available literature references.

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

#### **Definitions and abbreviations**

PC-TWA: Permissible Concentration-Time Weighted Average PC-STEL: Permissible Concentration-Short Term Exposure Limit IARC: International Agency for Research on Cancer ACGIH: American Conference of Governmental Industrial Hygienists STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit。 IDLH: Immediately Dangerous to Life or Health Concentrations ES: Exposure Standard OSF: Odour Safety Factor NOAEL :No Observed Adverse Effect Level LOAEL: Lowest Observed Adverse Effect Level TLV: Threshold Limit Value LOD: Limit Of Detection OTV: Odour Threshold Value **BCF: BioConcentration Factors** BEI: Biological Exposure Index AIIC: Australian Inventory of Industrial Chemicals **DSL:** Domestic Substances List NDSL: Non-Domestic Substances List IECSC: Inventory of Existing Chemical Substance in China EINECS: European INventory of Existing Commercial chemical Substances ELINCS: European List of Notified Chemical Substances NLP: No-Longer Polymers ENCS: Existing and New Chemical Substances Inventory KECI: Korea Existing Chemicals Inventory NZIoC: New Zealand Inventory of Chemicals PICCS: Philippine Inventory of Chemicals and Chemical Substances TSCA: Toxic Substances Control Act TCSI: Taiwan Chemical Substance Inventory INSQ: Inventario Nacional de Sustancias Químicas NCI: National Chemical Inventory FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances