STAINLESS STEEL STICK ELECTRODES– INOX 309l and EI 309L

Welding Guns of Australia Pty Ltd

Chemwatch Hazard Alert Code: **2**

Chemwatch: **5236-37**
Version No: **2.1.1.1**
Safety Data Sheet according to WHS and ADG requirements

Issue Date: **19/12/2016**
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L.GHS.AUS.EN

SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	STAINLESS STEEL STICK ELECTRODES– INOX 309l and EI 309L
Synonyms	AWS/ASME SFA 5.4 : E309L-16, Arc Welding / STAINLESS STEEL STICK ELECTRODES– INOX 309l and EI 309L, EN ISO 3581 A : E 23 12 L R 12
Other means of identification	Not Available

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

Relevant identified uses	Arc Welding.

Details of the supplier of the safety data sheet

Registered company name	Welding Guns of Australia Pty Ltd
Address	112 Christina Road Villawood NSW 2163 Australia
Telephone	+61 2 9780 4200
Fax	Not Available
Website	Not Available
Email	sales@unimig.com.au

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	1800 039 008 (24 hours)
Other emergency telephone numbers	+61 3 9573 3112 (24 hours)

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Carcinogenicity Category 2, Acute Aquatic Hazard Category 3
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI*

Label elements

GHS label elements	
SIGNAL WORD	**WARNING**

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Hazard statement(s)

H351	Suspected of causing cancer.
H402	Harmful to aquatic life

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P281	Use personal protective equipment as required.
P273	Avoid release to the environment.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/attention.

Precautionary statement(s) Storage

P405	Store locked up.

Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
		stainless steel stick electrode, containing
7439-89-6	60-75	iron
7439-96-5	1-4	manganese
7429-90-5	<1	aluminium
7440-02-0	9-12	nickel
7440-47-3	23-27	chromium
7789-75-5	1-3	calcium fluoride
9004-34-6	<1	cellulose
1317-60-8	<1	haematite
1312-76-1	2-4	potassium silicate
1344-09-8	2-4	sodium metasilicate
471-34-1	3-6	calcium carbonate
14808-60-7	3-6	silica crystalline - quartz
12001-26-2	5-8	mica
13463-67-7	10-15	titanium dioxide
		which upon use produces
Not avail.		welding fumes
		as
1309-37-1.		iron oxide fume
7439-96-5.		manganese fume
7440-02-0		nickel fume
7440-47-3		chromium fume
		action of arc on air may generate
10028-15-6		ozone
Mixture		nitrogen oxides

SECTION 4 FIRST AID MEASURES

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Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Wash out immediately with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. ▸ Particulate bodies from welding spatter may be removed carefully. ▸ **DO NOT** attempt to remove particles attached to or embedded in eye. ▸ Lay victim down, on stretcher if available and pad **BOTH** eyes, make sure dressing does not press on the injured eye by placing thick pads under dressing, above and below the eye. ▸ Seek urgent medical assistance, or transport to hospital. ▸ For "arc eye", i.e. welding flash or UV light burns to the eye: ▸ Place eye pads or light clean dressings over both eyes. ▸ Seek medical assistance. For THERMAL burns: ▸ Do NOT remove contact lens ▸ Lay victim down, on stretcher if available and pad **BOTH** eyes, make sure dressing does not press on the injured eye by placing thick pads under dressing, above and below the eye. ▸ Seek urgent medical assistance, or transport to hospital.
Skin Contact	If skin or hair contact occurs: ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation. For thermal burns: ▸ Decontaminate area around burn. ▸ Consider the use of cold packs and topical antibiotics. For first-degree burns (affecting top layer of skin) ▸ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides. ▸ Use compresses if running water is not available. ▸ Cover with sterile non-adhesive bandage or clean cloth. ▸ Do NOT apply butter or ointments; this may cause infection. ▸ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur. For second-degree burns (affecting top two layers of skin) ▸ Cool the burn by immerse in cold running water for 10-15 minutes. ▸ Use compresses if running water is not available. ▸ Do NOT apply ice as this may lower body temperature and cause further damage. ▸ Do NOT break blisters or apply butter or ointments; this may cause infection. ▸ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape. To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort): ▸ Lay the person flat. ▸ Elevate feet about 12 inches. ▸ Elevate burn area above heart level, if possible. ▸ Cover the person with coat or blanket. ▸ Seek medical assistance. For third-degree burns Seek immediate medical or emergency assistance. In the mean time: ▸ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound. ▸ Separate burned toes and fingers with dry, sterile dressings. ▸ Do not soak burn in water or apply ointments or butter; this may cause infection. ▸ To prevent shock see above. ▸ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. ▸ Have a person with a facial burn sit up. ▸ Check pulse and breathing to monitor for shock until emergency help arrives. ▸ Generally not applicable.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor. ▸ Generally not applicable.
Ingestion	▸ Generally not applicable.

Indication of any immediate medical attention and special treatment needed

Copper, magnesium, aluminium, antimony, iron, manganese, nickel, zinc (and their compounds) in welding, brazing, galvanising or smelting operations all

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give rise to thermally produced particulates of smaller dimension than may be produced if the metals are divided mechanically. Where insufficient ventilation or respiratory protection is available these particulates may produce "metal fume fever" in workers from an acute or long term exposure.

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

[Ellenhorn and Barceloux: Medical Toxicology]

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

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

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

- There is no restriction on the type of extinguisher which may be used.

Special hazards arising from the substrate or mixture

Fire Incompatibility	Welding electrodes should not be allowed to come into contact with strong acids or other substances which are corrosive to metals. Welding arc and metal sparks can ignite combustibles.

Advice for firefighters

Fire Fighting	- Alert Fire Brigade and tell them location and nature of hazard. - Wear breathing apparatus plus protective gloves in the event of a fire. - Prevent, by any means available, spillage from entering drains or water courses. - Use fire fighting procedures suitable for surrounding area. - **DO NOT** approach containers suspected to be hot. - Cool fire exposed containers with water spray from a protected location. - If safe to do so, remove containers from path of fire. - Equipment should be thoroughly decontaminated after use. Slight hazard when exposed to heat, flame and oxidisers.
Fire/Explosion Hazard	- Non combustible. - Not considered to be a significant fire risk, however containers may burn. - In a fire may decompose on heating and produce toxic / corrosive fumes. Welding arc and metal sparks can ignite combustibles.
HAZCHEM	Not Applicable

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	- Clean up all spills immediately. - Secure load if safe to do so. - Bundle/collect recoverable product. - Collect remaining material in containers with covers for disposal.

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Major Spills	▸ Clean up all spills immediately. ▸ Wear protective clothing, safety glasses, dust mask, gloves. ▸ Secure load if safe to do so. Bundle/collect recoverable product. ▸ Use dry clean up procedures and avoid generating dust. ▸ Vacuum up (consider explosion-proof machines designed to be grounded during storage and use). ▸ Water may be used to prevent dusting. ▸ Collect remaining material in containers with covers for disposal. ▸ Flush spill area with water.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	▸ Limit all unnecessary personal contact. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	▸ Keep dry. ▸ Store under cover. ▸ Protect containers against physical damage. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Packaging as recommended by manufacturer. ▸ Check that containers are clearly labelled
Storage incompatibility	Welding electrodes should not be allowed to come into contact with strong acids or other substances which are corrosive to metals. ▸ Avoid reaction with oxidising agents

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	iron	Fume (thermally generated) (respirable dust)	2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	manganese	Manganese, fume (as Mn)	1 mg/m3	3 mg/m3	Not Available	Not Available
Australia Exposure Standards	aluminium	Aluminium (metal dust) / Aluminium (welding fumes) (as Al) / Aluminium, pyro powders (as Al)	10 mg/m3 / 5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	nickel	Nickel, metal	1 mg/m3	Not Available	Not Available	Sen
Australia Exposure Standards	chromium	Chromium (metal)	0.5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	calcium fluoride	Fluorides (as F)	2.5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	cellulose	Cellulose (paper fibre)	10 mg/m3	Not Available	Not Available	Not Available

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Australia Exposure Standards	calcium carbonate	Calcium carbonate	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silica crystalline - quartz	Silica - Crystalline: Quartz (respirable dust) / Quartz (respirable dust)	0.1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	mica	Mica	2.5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	titanium dioxide	Titanium dioxide	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	welding fumes	Welding fumes (not otherwise classified)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	iron oxide fume	Iron oxide fume (Fe2O3) (as Fe)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	manganese fume	Manganese, fume (as Mn)	1 mg/m3	3 mg/m3	Not Available	Not Available
Australia Exposure Standards	nickel fume	Nickel, metal	1 mg/m3	Not Available	Not Available	Sen
Australia Exposure Standards	chromium fume	Chromium (metal)	0.5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	ozone	Ozone	Not Available	Not Available	0.2 mg/m3 / 0.1 ppm	Not Available
Australia Exposure Standards	nitrogen oxides	Nitrous oxide	45 mg/m3 / 25 ppm	Not Available	Not Available	Not Available
Australia Exposure Standards	nitrogen oxides	Nitric oxide	31 mg/m3 / 25 ppm	Not Available	Not Available	Not Available
Australia Exposure Standards	nitrogen oxides	Nitrogen dioxide	5.6 mg/m3 / 3 ppm	9.4 mg/m3 / 5 ppm	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
iron	Iron	3.2 mg/m3	35 mg/m3	150 mg/m3
manganese	Manganese	3 mg/m3	5 mg/m3	1,800 mg/m3
nickel	Nickel	4.5 mg/m3	50 mg/m3	99 mg/m3
chromium	Chromium	1.5 mg/m3	17 mg/m3	99 mg/m3
calcium fluoride	Calcium fluoride	15 mg/m3	170 mg/m3	1,000 mg/m3
potassium silicate	Potassium silicate; (Silicic acid, potassium salt)	30 mg/m3	330 mg/m3	2,000 mg/m3
sodium metasilicate	Silicic acid, sodium salt; (Sodium silicate)	5.9 mg/m3	65 mg/m3	390 mg/m3
calcium carbonate	Limestone; (Calcium carbonate; Dolomite)	45 mg/m3	500 mg/m3	3,000 mg/m3
calcium carbonate	Carbonic acid, calcium salt	45 mg/m3	210 mg/m3	1,300 mg/m3
silica crystalline - quartz	Silica, crystalline-quartz; (Silicon dioxide)	0.075 mg/m3	33 mg/m3	200 mg/m3
mica	Mica; (mica silicates)	9 mg/m3	99 mg/m3	590 mg/m3
titanium dioxide	Titanium oxide; (Titanium dioxide)	30 mg/m3	330 mg/m3	2,000 mg/m3
iron oxide fume	Iron oxide; (Ferric oxide)	15 mg/m3	360 mg/m3	2,200 mg/m3
manganese fume	Manganese	3 mg/m3	5 mg/m3	1,800 mg/m3
nickel fume	Nickel	4.5 mg/m3	50 mg/m3	99 mg/m3
chromium fume	Chromium	1.5 mg/m3	17 mg/m3	99 mg/m3
ozone	Ozone	0.24 ppm	1 ppm	10 ppm

Ingredient	Original IDLH	Revised IDLH
iron	Not Available	Not Available
manganese	N.E. mg/m3 / N.E. ppm	500 mg/m3
aluminium	Not Available	Not Available
nickel	N.E. mg/m3 / N.E. ppm	10 mg/m3
chromium	N.E. mg/m3 / N.E. ppm	250 mg/m3
calcium fluoride	500 mg/m3	250 mg/m3
cellulose	Not Available	Not Available
haematite	Not Available	Not Available

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potassium silicate	Not Available	Not Available
sodium metasilicate	Not Available	Not Available
calcium carbonate	Not Available	Not Available
silica crystalline - quartz	N.E. mg/m3 / N.E. ppm	50 mg/m3
mica	N.E. mg/m3 / N.E. ppm	1,500 mg/m3
titanium dioxide	N.E. mg/m3 / N.E. ppm	5,000 mg/m3
welding fumes	Not Available	Not Available
iron oxide fume	N.E. mg/m3 / N.E. ppm	2,500 mg/m3
manganese fume	N.E. mg/m3 / N.E. ppm	500 mg/m3
nickel fume	N.E. mg/m3 / N.E. ppm	10 mg/m3
chromium fume	N.E. mg/m3 / N.E. ppm	250 mg/m3
ozone	10 ppm	5 ppm
nitrogen oxides	Not Available	Not Available

MATERIAL DATA

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.
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)

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. Special ventilation requirements apply for processes which result in the generation of barium, chromium, lead, or nickel fume and in those processes which generate ozone. The use of mechanical ventilation by local exhaust systems is required as a minimum in all circumstances (including outdoor work). (In confined spaces always check that oxygen has not been depleted by excessive rusting of steel or snowflake corrosion of aluminium) Local exhaust systems must be designed to provide a minimum capture velocity at the fume source, away from the worker,

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	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. If risk of inhalation or overexposure exists, wear SAA approved respirator or work in fume hood.
Personal protection	
Eye and face protection	‣ Goggles or other suitable eye protection shall be used during all gas welding or oxygen cutting operations. Spectacles without side shields, with suitable filter lenses are permitted for use during gas welding operations on light work, for torch brazing or for inspection. ‣ For most open welding/brazing operations, goggles, even with appropriate filters, will not afford sufficient facial protection for operators. Where possible use welding helmets or handshields corresponding to EN 175, ANSI Z49:12005, AS 1336 and AS 1338 which provide the maximum possible facial protection from flying particles and fragments. [WRIA-WTIA Technical Note 7] ‣ An approved face shield or welding helmet can also have filters for optical radiation protection, and offer additional protection against debris and sparks. ‣ UV blocking protective spectacles with side shields or welding goggles are considered primary protection, with the face shield or welding helmet considered secondary protection. ‣ The optical filter in welding goggles, face mask or helmet must be a type which is suitable for the sort of work being done.A filter suitable for gas welding, for instance, should not be used for arc welding. ‣ Face masks which are self dimming are available for arc welding, MIG, TIG and plasma cutting, and allow better vision before the arc is struck and after it is extinguished. For submerged arc welding use a lens shade which gives just sufficient arc brightness to allow weld pool control.
Skin protection	See Hand protection below
Hands/feet protection	‣ Welding gloves conforming to Standards such as EN 12477:2001, ANSI Z49.1, AS/NZS 2161:2008 produced from leather, rubber, treated cotton,or alumininised ‣ These gloves protect against mechanical risk caused by abrasion, blade cut, tear and puncture ‣ Other gloves which protect against thermal risks (heat and fire) might also be considered - these comply with different standards to those mentioned above. ‣ One pair of gloves may not be suitable for all processes. For example, gloves that are suitable for low current Gas Tungsten Arc Welding (GTAW) (thin and flexible) would not be proper for high-current Air Carbon Arc Cutting (CAC-A) (insulated, tough, and durable) ‣ Protective gloves eg. Leather gloves or gloves with Leather facing
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.
Thermal hazards	Not Available

Respiratory protection

Type NO Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.
Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

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Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	NO-AUS	-	NO-PAPR-AUS / Class 1
up to 50 x ES	-	NO-AUS / Class 1	-
up to 100 x ES	-	NO-2	NO-PAPR-2 ^

^ - Full-face
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	Odourless solid, non-volatile with varying colour; insoluble in water.

Physical state	Manufactured	**Relative density (Water = 1)**	7.8
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Applicable
pH (as supplied)	Not Applicable	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	1500 ca.	**Viscosity (cSt)**	Not Applicable
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Applicable	**Taste**	Not Available
Evaporation rate	Not Applicable	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Applicable	**Surface Tension (dyn/cm or mN/m)**	Not Applicable
Lower Explosive Limit (%)	Not Applicable	**Volatile Component (%vol)**	Not Applicable
Vapour pressure (kPa)	Not Applicable	**Gas group**	Not Available
Solubility in water (g/L)	Immiscible	**pH as a solution (1%)**	Not Applicable
Vapour density (Air = 1)	Not Applicable	**VOC g/L**	Not Applicable

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

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Information on toxicological effects

Inhaled	Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Fumes evolved during welding operations may be irritating to the upper-respiratory tract and may be harmful if inhaled. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure.
Ingestion	The material has **NOT** been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.
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. 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. Irritation and skin reactions are possible with sensitive skin Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Limited evidence exists, or practical experience suggests, that the material may cause eye irritation in a substantial number of individuals and/or is expected to produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. Ultraviolet (UV) radiation can also damage the lens of the eye. Many arc welders are aware of the condition known as "arc-eye," a sensation of sand in the eyes. This condition is caused by excessive eye exposure to UV. Exposure to ultraviolet rays may also increase the skin effects of some industrial chemicals (coal tar and cresol compounds, for example). Exposure of the human eye to intense visible light can produce adaptation, pupillary reflex, and shading of the eyes. Such actions are protective mechanisms to prevent excessive light from being focused on the retina. In the arc welding process, eye exposure to intense visible light is prevented for the most part by the welder's helmet. However, some individuals have sustained retinal damage due to careless "viewing" of the arc. At no time should the arc be observed without eye protection. Contact with the eye, by metal dusts, may produce mechanical abrasion or foreign body penetration of the eyeball. Iron particles embedded in the eye may produce a condition known as ocular siderosis; effects include discolouration of the cornea and iris and pupillary effects such as poor reaction to light and accommodation. Particles entering the lens may produce cataracts. A rare consequence of ocular siderosis is glaucoma.
Chronic	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. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. 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

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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.
There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of:
- clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.
Chronic excessive iron exposure has been associated with haemosiderosis and consequent possible damage to the liver and pancreas. Haemosiderin is a golden-brown insoluble protein produced by phagocytic digestion of haematin (an iron-based pigment). Haemosiderin is found in most tissues, especially in the liver, in the form of granules. Other sites of haemosiderin deposition include the pancreas and skin. A related condition, haemochromatosis, which involves a disorder of metabolism of these deposits, may produce cirrhosis of the liver, diabetes, and bronze pigmentation of the skin - heart failure may eventually occur.
Such exposure may also produce conjunctivitis, choroiditis, retinitis (both inflammatory conditions involving the eye) and siderosis of tissues if iron remains in these tissues. Siderosis is a form of pneumoconiosis produced by iron dusts. Siderosis also includes discoloration of organs, excess circulating iron and degeneration of the retina, lens and uvea as a result of the deposition of intraocular iron. Siderosis might also involve the lungs - involvement rarely develops before ten years of regular exposure. Often there is an accompanying inflammatory reaction of the bronchi. Permanent scarring of the lungs does not normally occur.
High levels of iron may raise the risk of cancer. This concern stems from the theory that iron causes oxidative damage to tissues and organs by generating highly reactive chemicals, called free radicals, which subsequently react with DNA. Cells may be disrupted and may be become cancerous. People whose genetic disposition prevents them from keeping tight control over iron (e.g. those with the inherited disorder, haemochromatosis) may be at increased risk.
Iron overload in men may lead to diabetes, arthritis, liver cancer, heart irregularities and problems with other organs as iron builds up.
[K. Schmidt, New Scientist, No. 1919 pp.11-12, 2nd April, 1994]

	TOXICITY	IRRITATION
STAINLESS STEEL STICK ELECTRODES– INOX 309l and El 309L	Not Available	Not Available
iron	TOXICITY	IRRITATION
	Oral (rat) LD50: 7500 mg/kg[1]	Not Available
manganese	TOXICITY	IRRITATION
	Oral (rat) LD50: >2000 mg/kg[1]	Eye (rabbit): 500 mg/24h - mild
		Skin (rabbit): 500 mg/24h - mild
aluminium	TOXICITY	IRRITATION
	Oral (rat) LD50: >2000 mg/kg[1]	Not Available
nickel	TOXICITY	IRRITATION
	Oral (rat) LD50: 5000 mg/kg[2]	Not Available
chromium	TOXICITY	IRRITATION
	Not Available	Not Available
calcium fluoride	TOXICITY	IRRITATION
	Oral (rat) LD50: 4250 mg/kg[2]	Not Available

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cellulose	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Not Available
	Inhalation (rat) LC50: >5.8 mg/L/4hr[2]	
	Oral (rat) LD50: >5000 mg/kg[2]	
haematite	**TOXICITY**	**IRRITATION**
	Inhalation (rat) LC50: >2.2 mg/l1 hr[1]	Not Available
	Oral (rat) LD50: 14.6 mg/kg[1]	
potassium silicate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >5000 mg/kg[1]	Not Available
	Oral (rat) LD50: >5000 mg/kg[1]	
sodium metasilicate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >5000 mg/kg[1]	Skin (human): 250 mg/24h SEVERE
	Oral (rat) LD50: 500 mg/kg[1]	Skin (rabbit): 250 mg/24h SEVERE
calcium carbonate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit): 0.75 mg/24h - SEVERE
	Oral (rat) LD50: >2000 mg/kg[1]	Skin (rabbit): 500 mg/24h-moderate
silica crystalline - quartz	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
mica	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
titanium dioxide	**TOXICITY**	**IRRITATION**
	Inhalation (rat) LC50: >2.28 mg/l/4hr[1]	Skin (human): 0.3 mg /3D (int)-mild *
	Inhalation (rat) LC50: >3.56 mg/l/4hr[1]	
	Inhalation (rat) LC50: >6.82 mg/l/4hr[1]	
	Inhalation (rat) LC50: 3.43 mg/l/4hr[1]	
	Inhalation (rat) LC50: 5.09 mg/l/4hr[1]	
	Oral (rat) LD50: >2000 mg/kg[1]	
welding fumes	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
iron oxide fume	**TOXICITY**	**IRRITATION**
	Oral (rat) LD50: >5000 mg/kg[1]	Not Available
manganese fume	**TOXICITY**	**IRRITATION**
	Oral (rat) LD50: >2000 mg/kg[1]	Eye (rabbit) 500mg/24H Mild
		Skin (rabbit) 500mg/24H Mild
nickel fume	**TOXICITY**	**IRRITATION**
	Oral (rat) LD50: 5000 mg/kg[2]	Not Available
chromium fume	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
ozone	**TOXICITY**	**IRRITATION**
	Inhalation (rat) LC50: 0.001 mg/L/44hr[2]	Not Available
	Inhalation (rat) LC50: 4.8 ppm/4hr[2]	

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nitrogen oxides	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*	

NICKEL	Oral (rat) TDLo: 500 mg/kg/5D-I Inhalation (rat) TCLo: 0.1 mg/m3/24H/17W-C
CHROMIUM	Tenth Annual Report on Carcinogens: Substance known to be Carcinogenic [*National Toxicology Program: U.S. Dep. of Health and Human Services 2002*] Gastrointestinal tumours, lymphoma, musculoskeletal tumours and tumours at site of application recorded.
CALCIUM CARBONATE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. No evidence of carcinogenic properties. No evidence of mutagenic or teratogenic effects.
SILICA CRYSTALLINE - QUARTZ	**WARNING:** For inhalation exposure ONLY: This substance has been classified by the IARC as Group 1: **CARCINOGENIC TO HUMANS** The International Agency for Research on Cancer (IARC) has classified occupational exposures to **respirable** (<5 um) crystalline silica as being carcinogenic to humans . This classification is based on what IARC considered sufficient evidence from epidemiological studies of humans for the carcinogenicity of inhaled silica in the forms of quartz and cristobalite. Crystalline silica is also known to cause silicosis, a non-cancerous lung disease. Intermittent exposure produces; focal fibrosis, (pneumoconiosis), cough, dyspnoea, liver tumours. * Millions of particles per cubic foot (based on impinger samples counted by light field techniques). NOTE : the physical nature of quartz in the product determines whether it is likely to present a chronic health problem. To be a hazard the material must enter the breathing zone as respirable particles.
TITANIUM DIOXIDE	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. For titanium dioxide: Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin. Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica. No data were available on genotoxic effects in titanium dioxide-exposed humans. Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts. Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium. Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light. **Animal carcinogenicity data**

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	Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats. In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative. Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice. In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative. * IUCLID
WELDING FUMES	Not available. Refer to individual constituents.
OZONE	NOTE: Ozone aggravates chronic obstructive pulmonary diseases. Ozone is suspected also of increasing the risk of acute and chronic respiratory disease, mutagenesis and foetotoxicity. In animals short-term exposure to ambient concentrations of less than 1 ppm results in reduced capacity to kill intrapulmonary organisms and allows purulent bacteria to proliferate [Ellenhorn etal].
nitrogen oxides	Data for nitrogen dioxide: Substance has been investigated as a mutagen and reproductive effector. NOTE: Interstitial edema, epithelial proliferation and, in high concentrations, fibrosis and emphysema develop after repeated exposure.
STAINLESS STEEL STICK ELECTRODES– INOX 309l and El 309L & NICKEL & TITANIUM DIOXIDE & WELDING FUMES & NICKEL FUME	**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
STAINLESS STEEL STICK ELECTRODES– INOX 309l and El 309L & WELDING FUMES	Most welding is performed using electric arc processes - manual metal arc, metal inert gas (MIG) and tungsten inert gas welding (TIG) – and most welding is on mild steel. There has been considerable evidence over several decades regarding cancer risks in relation to welding activities. Several case-control studies reported excess risks of ocular melanoma in welders. This association may be due to the presence in some welding environments of fumes of thorium-232, which is used in tungsten welding rods. Different welding environments may present different and complex profiles of exposures. In one study to characterise welding fume aerosol nanoparticles in mild steel metal active gas welding showed a mass median diameter (MMMD) of 200-300 nm. A widespread consensus seems to have formed to the effect that some welding environments, notably in stainless steel welding, do carry risks of lung cancer. This widespread consensus is in part based on empirical evidence regarding risks among stainless steel welders and in part on the fact that stainless steel welding entails moderately high exposure to nickel and chromium VI compounds, which are recognised lung carcinogens. The corollary is that welding without the presence of nickel and chromium VI compounds, namely mild-steel welding, should not carry risk. But it appears that this line of reasoning in not supported by the accumulated body of epidemiologic evidence. While there remained some uncertainty about possible confounding by smoking and by asbestos, and some possible publication bias, the overwhelming evidence is that there has been an excess risk of lung cancer among welders as a whole in the order of 20%-40%.The most begrudging explanation is that there is an as-yet unexplained common reason for excess lung cancer risks that applies to all types of welders. It has been have proposed that iron fumes may play such a role, and some Finnish data appear to support this hypothesis, though not conclusively. This hypothesis would also imply that excess lung cancer risks among welders are not unique to welders, but rather may be shared among many types of metal working occupations. Welders are exposed to a range of fumes and gases (evaporated metal, metal oxides, hydrocarbons, nanoparticles, ozone, oxides of nitrogen (NOx)) depending on the electrodes, filler wire and flux materials used in the process, but also physical exposures such as electric and magnetic fields (EMF) and ultraviolet (UV) radiation. Fume particles contain a wide variety of oxides and salts of metals and other compounds, which are produced mainly from electrodes, filler wire and flux materials. Fumes from the welding of stainless-steel and other alloys contain nickel compounds and chromium[VI] and [III]. Ozone is formed during most electric arc welding, and exposures can be high in comparison to the exposure limit, particularly during metal inert gas welding of aluminium. Oxides of nitrogen are found during manual metal arc welding and particularly during gas welding. Welders who weld painted mild steel can also be exposed to a range of organic compounds produced by pyrolysis. In one study particle elemental composition was mainly iron and manganese. Ni and Cr exposures were very low in the vicinity of mild steel welders, but much higher in the background in the workshop where there presumably was some stainless steel welding. Personal exposures to manganese ranged from 0.01-4.93 mg/m3 and to iron ranged from 0.04-16.29 mg/m3 in eight Canadian welding companies. Types of welding identified were mostly (90%) MIG mild steel, MIG stainless steel, and TIG aluminum. Carbon monoxide levels were less than 5.0 ppm (at source) and ozone levels varied from 0.4-0.6 ppm (at source). Welders, especially in shipyards, may also be exposed to asbestos dust. Physical exposures such as electric and magnetic fields (EMF) and ultraviolet (UV) radiation are also common. In all, the in vivo studies suggest that different welding fumes cause varied responses in rat lungs in vivo , and the toxic effects typically correlate with the metal composition of the fumes and their ability to produce free radicals. In many studies both soluble and insoluble fractions of the stainless steel welding fumes were required to produce most types of effects,

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	indicating that the responses are not dependent exclusively on the soluble metals. Lung tumourigenicity of welding fumes was investigated in lung tumour susceptible (A/J) strain of mice. Male mice were exposed by pharyngeal aspiration four times (once every 3 days) to 85 ug of gas metal arc-mild steel (GMA-MS), GMA-SS, or manual metal arc-SS (MMA-SS) fume. At 48 weeks post-exposure, GMA-SS caused the greatest increase in tumour multiplicity and incidence, but did not differ from sham exposure. Tumour incidence in the GMA-SS group versus sham control was close to significance at 78 weeks post exposure. Histopathological analysis of the lungs of these mice showed the GMA-SS group having an increase in preneoplasia/tumour multiplicity and incidence compared to the GMA-MS and sham groups at 48 weeks. The increase in incidence in the GMA-SS exposed mice was significant compared to the GMA-MS group but not to the sham-exposed animals, and the difference in incidence between the GMA-SS and MMA-SS groups was of border-line significance (p = 0.06). At 78 week s post-exposure, no statistically significant differences. A significantly higher frequency of micronuclei in peripheral blood lymphocytes (binucleated cell assay) and higher mean levels of both centromere-positive and centromere-negative micronuclei was observed in welders (n=27) who worked without protective device compared to controls (n=30).The rate of micronucleated cells did not correlate with the duration of exposure.
MANGANESE & SODIUM METASILICATE	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
MANGANESE & SODIUM METASILICATE & TITANIUM DIOXIDE	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.
ALUMINIUM & CHROMIUM & HAEMATITE & POTASSIUM SILICATE & MICA & CHROMIUM FUME	No significant acute toxicological data identified in literature search.
NICKEL & NICKEL FUME	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.
NICKEL & NICKEL FUME	Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [*National Toxicology Program: U.S. Dep. of Health & Human Services 2002*]
CHROMIUM & CHROMIUM FUME	For chrome(III) and other valence states (except hexavalent): For inhalation exposure, all trivalent and other chromium compounds are treated as particulates, not gases. The mechanisms of chromium toxicity are very complex, and although many studies on chromium are available, there is a great deal of uncertainty about how chromium exerts its toxic influence. Much more is known about the mechanisms of hexavalent chromium toxicity than trivalent chromium toxicity. There is an abundance of information available on the carcinogenic potential of chromium compounds and on the genotoxicity and mutagenicity of chromium compounds in experimental systems. The consensus from various reviews and agencies is that evidence of carcinogenicity of elemental, divalent, or trivalent chromium compounds is lacking. Epidemiological studies of workers in a number of industries (chromate production, chromate pigment production and use, and chrome plating) conclude that while occupational exposure to hexavalent chromium compounds is associated with an increased risk of respiratory system cancers (primarily bronchogenic and nasal), results from occupational exposure studies to mixtures that were mainly elemental and trivalent (ferrochromium alloy worker) were inconclusive. Studies in leather tanners, who were exposed to trivalent chromium were consistently negative. In addition to the lack of direct evidence of carcinogenicity of trivalent or elemental chromium and its compounds, the genotoxic evidence is overwhelmingly negative. The lesser potency of trivalent chromium relative to hexavalent chromium is likely related to the higher redox potential of hexavalent chromium and its greater ability to enter cells. enter cells The general inability of trivalent chromium to traverse membranes and thus be absorbed or reach peripheral tissue in significant amounts is generally accepted as a probable explanation for the overall absence of systemic trivalent chromium toxicity. Elemental and divalent forms of chromium are not able to traverse membranes readily either. This is not to say that elemental, divalent, or trivalent chromium compounds cannot traverse membranes and reach peripheral tissue, the mechanism of absorption is simply less efficient in comparison to absorption of hexavalent chromium compounds. Hexavalent chromium compounds exist as tetrahedral chromate anions, resembling the forms of other natural anions like sulfate and phosphate which are permeable across nonselective membranes. Trivalent chromium forms octahedral complexes which cannot easily enter though these channels, instead being absorbed via passive diffusion and phagocytosis. Although trivalent chromium is less well absorbed than hexavalent chromium, workers exposed to trivalent compounds have had detectable levels of chromium in the urine at the end of a workday. Absorbed chromium is widely distributed throughout the body via the bloodstream, and can reach the foetus. Although there is ample in vivo evidence that hexavalent chromium is efficiently reduced to trivalent chromium in the gastrointestinal tract and can be reduced to the trivalent form by ascorbate and glutathione in the lungs, there is no evidence that trivalent chromium is converted to hexavalent chromium in biological systems. In general, trivalent chromium compounds are cleared rapidly from the blood and more slowly from the tissues. Although not fully characterized, the biologically active trivalent chromium molecule appears to be chromodulin, also referred to as (GTF). Chromodulin is an oligopeptide complex containing four chromic ions. Chromodulin may facilitate interactions of insulin with its receptor site, influencing protein, glucose, and lipid metabolism. Inorganic trivalent chromium compounds, which do not appear to have insulin-potentiating properties, are capable of being converted into biologically active forms by humans and animals Chromium can be a potent sensitiser in a small minority of humans, both from dermal and inhalation exposures.

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	The most sensitive endpoint identified in animal studies of acute exposure to trivalent chromium appears to involve the respiratory system. Specifically, acute exposure to trivalent chromium is associated with impaired lung function and lung damage. Based on what is known about absorption of chromium in the human body, its potential mechanism of action in cells, and occupational data indicating that valence states other than hexavalent exhibit a relative lack of toxicity the toxicity of elemental and divalent chromium compounds is expected to be similar to or less than common trivalent forms.
CHROMIUM & HAEMATITE & CHROMIUM FUME	The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
CALCIUM FLUORIDE & CELLULOSE & POTASSIUM SILICATE & SODIUM METASILICATE & CALCIUM CARBONATE & OZONE & nitrogen oxides	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

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

Legend: ✘ *– Data available but does not fill the criteria for classification*
✔ *– Data required to make classification available*
⊘ *– Data Not Available to make classification*

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
iron	LC50	96	Fish	0.05mg/L	2
iron	EC50	96	Algae or other aquatic plants	3.7mg/L	4
iron	BCF	24	Crustacea	0.0000002mg/L	4
iron	EC50	504	Crustacea	4.49mg/L	2
iron	NOEC	504	Fish	0.52mg/L	2
manganese	LC50	96	Fish	>3.6mg/L	2
manganese	EC50	48	Crustacea	>1.6mg/L	2
manganese	EC50	72	Algae or other aquatic plants	2.8mg/L	2
manganese	BCFD	37	Algae or other aquatic plants	2.2mg/L	4
manganese	EC50	72	Algae or other aquatic plants	4.5mg/L	2
manganese	NOEC	48	Crustacea	1.6mg/L	2
aluminium	LC50	96	Fish	0.078-0.108mg/L	2
aluminium	EC50	48	Crustacea	0.7364mg/L	2
aluminium	EC50	96	Algae or other aquatic plants	0.0054mg/L	2
aluminium	BCF	360	Algae or other aquatic plants	9mg/L	4
aluminium	EC50	120	Fish	0.000051mg/L	5
aluminium	NOEC	72	Algae or other aquatic plants	>=0.004mg/L	2
nickel	LC50	96	Fish	0.0000475mg/L	4
nickel	EC50	48	Crustacea	0.013mg/L	5
nickel	EC50	72	Algae or other aquatic plants	0.0407mg/L	2
nickel	BCF	1440	Algae or other aquatic plants	0.47mg/L	4

Continued...

nickel	EC50	720	Crustacea	0.0062mg/L	2
nickel	NOEC	72	Algae or other aquatic plants	0.0035mg/L	2
chromium	LC50	96	Fish	13.9mg/L	4
chromium	EC50	48	Crustacea	0.0225mg/L	5
chromium	EC50	72	Algae or other aquatic plants	0.104mg/L	4
chromium	BCF	1440	Algae or other aquatic plants	0.0495mg/L	4
chromium	EC50	48	Crustacea	0.0245mg/L	5
chromium	NOEC	672	Fish	0.00019mg/L	4
calcium fluoride	LC50	96	Fish	51mg/L	2
calcium fluoride	EC50	96	Crustacea	26-48mg/L	2
calcium fluoride	NOEC	504	Fish	4mg/L	2
cellulose	LC50	96	Fish	7.45058mg/L	3
cellulose	EC50	96	Algae or other aquatic plants	17857.93905mg/L	3
cellulose	EC50	384	Crustacea	42.76118mg/L	3
potassium silicate	EC50	96	Crustacea	160mg/L	1
potassium silicate	NOEC	96	Fish	>=1000mg/L	1
sodium metasilicate	LC50	96	Fish	1800mg/L	4
sodium metasilicate	EC50	96	Crustacea	160mg/L	1
sodium metasilicate	NOEC	96	Fish	>=1000mg/L	1
calcium carbonate	LC50	96	Fish	>56000mg/L	4
calcium carbonate	EC50	72	Algae or other aquatic plants	>14mg/L	2
calcium carbonate	NOEC	72	Algae or other aquatic plants	14mg/L	2
titanium dioxide	LC50	96	Fish	9.214mg/L	3
titanium dioxide	EC50	48	Crustacea	>10mg/L	2
titanium dioxide	EC50	72	Algae or other aquatic plants	5.83mg/L	4
titanium dioxide	EC20	72	Algae or other aquatic plants	1.81mg/L	4
titanium dioxide	NOEC	336	Fish	0.089mg/L	4
iron oxide fume	LC50	96	Fish	0.05mg/L	2
iron oxide fume	EC50	72	Algae or other aquatic plants	18mg/L	2
iron oxide fume	EC50	504	Crustacea	4.49mg/L	2
iron oxide fume	NOEC	504	Fish	0.52mg/L	2
manganese fume	LC50	96	Fish	>3.6mg/L	2
manganese fume	EC50	48	Crustacea	>1.6mg/L	2
manganese fume	EC50	72	Algae or other aquatic plants	2.8mg/L	2
manganese fume	BCFD	37	Algae or other aquatic plants	2.2mg/L	4
manganese fume	EC50	72	Algae or other aquatic plants	4.5mg/L	2
manganese fume	NOEC	48	Crustacea	1.6mg/L	2
nickel fume	LC50	96	Fish	0.0000475mg/L	4
nickel fume	EC50	48	Crustacea	0.013mg/L	5
nickel fume	EC50	72	Algae or other aquatic plants	0.0407mg/L	2
nickel fume	BCF	1440	Algae or other aquatic plants	0.47mg/L	4
nickel fume	EC50	720	Crustacea	0.0062mg/L	2
nickel fume	NOEC	72	Algae or other aquatic plants	0.0035mg/L	2
chromium fume	LC50	96	Fish	13.9mg/L	4
chromium fume	EC50	48	Crustacea	0.0225mg/L	5
chromium fume	EC50	72	Algae or other aquatic plants	0.104mg/L	4
chromium fume	BCF	1440	Algae or other aquatic plants	0.0495mg/L	4
chromium fume	EC50	48	Crustacea	0.0245mg/L	5
chromium fume	NOEC	672	Fish	0.00019mg/L	4
ozone	LC50	96	Fish	0.0093mg/L	4
ozone	EC50	96	Fish	0.0093mg/L	5

Continued...

ozone	NOEC	2160	Fish	0.002mg/L	5
Legend:	*Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*				

Harmful to aquatic organisms.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
cellulose	LOW	LOW
titanium dioxide	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
cellulose	LOW (LogKOW = -5.1249)
titanium dioxide	LOW (BCF = 10)

Mobility in soil

Ingredient	Mobility
cellulose	LOW (KOC = 10)
titanium dioxide	LOW (KOC = 23.74)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	· Recycle wherever possible or consult manufacturer for recycling options. · Consult State Land Waste Management Authority for disposal. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority.

SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

IRON(7439-89-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Inventory of Chemical Substances (AICS)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

MANGANESE(7439-96-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Continued...

Australia Exposure Standards
Australia Hazardous Substances Information System - Consolidated Lists
Australia Inventory of Chemical Substances (AICS)
International Air Transport Association (IATA) Dangerous Goods Regulations - Prohibited List Passenger and Cargo Aircraft

ALUMINIUM(7429-90-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Hazardous Substances Information System - Consolidated Lists
Australia Inventory of Chemical Substances (AICS)

NICKEL(7440-02-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Hazardous Substances Information System - Consolidated Lists
Australia Inventory of Chemical Substances (AICS)

CHROMIUM(7440-47-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Hazardous Substances Information System - Consolidated Lists
Australia Inventory of Chemical Substances (AICS)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

CALCIUM FLUORIDE(7789-75-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Inventory of Chemical Substances (AICS)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

CELLULOSE(9004-34-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Inventory of Chemical Substances (AICS)

HAEMATITE(1317-60-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

POTASSIUM SILICATE(1312-76-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

SODIUM METASILICATE(1344-09-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

CALCIUM CARBONATE(471-34-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Inventory of Chemical Substances (AICS)

SILICA CRYSTALLINE - QUARTZ(14808-60-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Hazardous Substances Information System - Consolidated Lists
Australia Inventory of Chemical Substances (AICS)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

MICA(12001-26-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Hazardous Substances Information System - Consolidated Lists
Australia Inventory of Chemical Substances (AICS)

TITANIUM DIOXIDE(13463-67-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Inventory of Chemical Substances (AICS)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

WELDING FUMES(NOT AVAIL.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Hazardous Substances Information System - Consolidated Lists
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

IRON OXIDE FUME(1309-37-1.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Hazardous Substances Information System - Consolidated Lists
Australia Inventory of Chemical Substances (AICS)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

MANGANESE FUME(7439-96-5.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Hazardous Substances Information System - Consolidated Lists
Australia Inventory of Chemical Substances (AICS)

NICKEL FUME(7440-02-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Hazardous Substances Information System - Consolidated Lists
Australia Inventory of Chemical Substances (AICS)

Continued...

CHROMIUM FUME(7440-47-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Hazardous Substances Information System - Consolidated Lists
Australia Inventory of Chemical Substances (AICS)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

OZONE(10028-15-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Hazardous Substances Information System - Consolidated Lists

NITROGEN OXIDES(MIXTURE) IS FOUND ON THE FOLLOWING REGULATORY LISTS

International Air Transport Association (IATA) Dangerous Goods Regulations - Prohibited List Passenger and Cargo Aircraft

National Inventory	Status
Australia - AICS	N (ozone; welding fumes; nitrogen oxides)
Canada - DSL	N (haematite; ozone; welding fumes; nitrogen oxides)
Canada - NDSL	N (manganese fume; nickel fume; chromium fume; sodium metasilicate; manganese; silica crystalline - quartz; welding fumes; iron oxide fume; nitrogen oxides; aluminium; nickel; potassium silicate; iron; mica; chromium)
China - IECSC	N (welding fumes; nitrogen oxides)
Europe - EINEC / ELINCS / NLP	N (welding fumes; nitrogen oxides; mica)
Japan - ENCS	N (manganese fume; nickel fume; haematite; chromium fume; ozone; manganese; welding fumes; nitrogen oxides; aluminium; nickel; iron; cellulose; mica; chromium)
Korea - KECI	N (welding fumes; nitrogen oxides)
New Zealand - NZIoC	N (welding fumes; nitrogen oxides)
Philippines - PICCS	N (ozone; welding fumes; nitrogen oxides)
USA - TSCA	N (welding fumes; nitrogen oxides; mica)
Legend:	*Y = All ingredients are on the inventory* *N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 OTHER INFORMATION

Other information

Ingredients with multiple cas numbers

Name	CAS No
aluminium	7429-90-5, 91728-14-2
calcium fluoride	7789-75-5, 14542-23-5
cellulose	9004-34-6, 68442-85-3
potassium silicate	1312-76-1, 11116-04-4, 12698-85-0, 61869-46-3
calcium carbonate	471-34-1, 13397-26-7, 15634-14-7, 1317-65-3, 72608-12-9, 878759-26-3, 63660-97-9, 459411-10-0, 198352-33-9, 146358-95-4
silica crystalline - quartz	14808-60-7, 122304-48-7, 122304-49-8, 12425-26-2, 1317-79-9, 70594-95-5, 87347-84-0
mica	12001-26-2, 129899-84-9, 61076-94-6
titanium dioxide	13463-67-7, 1317-70-0, 1317-80-2, 12188-41-9, 1309-63-3, 100292-32-8, 101239-53-6, 116788-85-3, 12000-59-8, 12701-76-7, 12767-65-6, 12789-63-8, 1344-29-2, 185323-71-1, 185828-91-5, 188357-76-8, 188357-79-1, 195740-11-5, 221548-98-7, 224963-00-2, 246178-32-5, 252962-41-7, 37230-92-5, 37230-94-7, 37230-95-8, 37230-96-9, 39320-58-6, 39360-64-0, 39379-02-7, 416845-43-7, 494848-07-6, 494848-23-6, 494851-77-3, 494851-98-8, 55068-84-3, 55068-85-4, 552316-51-5, 62338-64-1, 767341-00-4, 97929-50-5, 98084-96-9

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.
A list of reference resources used to assist the committee may be found at:
www.chemwatch.net

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

Continued...

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
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