Talweld 80Ni1, 80Ni2, D2, Cu, 110, B2, B3, B6, B9

TALARC

Chemwatch Hazard Alert Code: **2**

Chemwatch: **5189-85**
Version No: **3.1.1.1**
Safety Data Sheet according to WHS and ADG requirements

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Initial Date: **Not Available**
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SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	Talweld 80Ni1, 80Ni2, D2, Cu, 110, B2, B3, B6, B9
Synonyms	Rods for Tungsten Inert Gas (TIG) Welding, Solid low alloy welding wire for gas-metal arc welding (GMAW/MIG)
Other means of identification	Not Available

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

Relevant identified uses	Welding and filler metal.

Details of the supplier of the safety data sheet

Registered company name	TALARC
Address	10-16 Syme Street Brunswick 3056 VIC Australia
Telephone	+61 3 9388 0588
Fax	+61 3 9388 0710
Website	http://talarc.com
Email	sales@talarc.com

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	+61 3 9388 0588 (Hours 9am-5pm AEST)
Other emergency telephone numbers	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

CHEMWATCH HAZARD RATINGS

		Min Max
Flammability	0	
Toxicity	2	
Body Contact	1	
Reactivity	0	
Chronic	2	

0 = Minimum
1 = Low
2 = Moderate
3 = High
4 = Extreme

Poisons Schedule	Not Applicable
GHS Classification [1]	Acute Toxicity (Inhalation) Category 4, Carcinogen Category 2
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI*

Label elements

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GHS label elements	
SIGNAL WORD	WARNING

Hazard statement(s)

H332	Harmful if inhaled
H351	Suspected of causing cancer

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P271	Use only outdoors or in a well-ventilated area.
P281	Use personal protective equipment as required.
P261	Avoid breathing dust/fume/gas/mist/vapours/spray.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/attention.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised chemical landfill or if organic to high temperature incineration

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
		metal arc solid wire
		which upon use generates:
Not avail.	>60	welding fumes
		as
1309-37-1.		iron oxide fume
7440-47-3		chromium fume
7440-50-8		copper fume
7439-96-5.		manganese fume
7440-02-0		nickel fume
69012-64-2		silica welding fumes
7439-98-7		molybdenum fume

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	▸ 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.

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	Arc rays can injure eyes
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. Arc rays can burn skin
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.
Ingestion	Not normally a hazard due to physical form of product.

Indication of any immediate medical attention and special treatment needed

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

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

[Ellenhorn and Barceloux: Medical Toxicology]

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.
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.

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Minor Spills	Clean up all spills immediately. Avoid contact with skin and eyes. Wear impervious gloves and safety glasses. Use dry clean up procedures and avoid generating dust. Place in suitable containers for disposal.
Major Spills	Minor hazard. ‣ Clear area of personnel. ‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Control personal contact with the substance, by using protective equipment if risk of overexposure exists. ‣ Prevent, by any means available, spillage from entering drains or water courses.

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	▸ Contain spill/secure load if safe to do so. ▸ Bundle/collect recoverable product and label for recycling. ▸ Collect remaining product and place in appropriate containers for disposal. ▸ Clean up/sweep up area. Water may be required. ▸ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	▸ 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 MSDS. ▸ 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 MSDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Packaging as recommended by manufacturer. ▸ Check that containers are clearly labelled
Storage incompatibility	▸ Avoid strong acids, bases.

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 oxide fume	Iron oxide fume (Fe2O3) (as Fe)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	chromium fume	Chromium (metal)	0.5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	copper fume	Copper (fume) / Copper, dusts & mists (as Cu)	0.2 mg/m3 / 1 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	silica welding fumes	Silica - Amorphous Fume (thermally generated)(respirable dust) (g)	2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	molybdenum fume	Fume (thermally generated) (respirable dust)(g)	2 mg/m3	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
iron oxide fume	Iron oxide; (Ferric oxide)	15 mg/m3	360 mg/m3	2200 mg/m3
chromium fume	Chromium	1.5 mg/m3	17 mg/m3	99 mg/m3
copper fume	Copper	1 mg/m3	1 mg/m3	45 mg/m3

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manganese fume	Manganese	3 mg/m3	5 mg/m3	1800 mg/m3
nickel fume	Nickel	4.5 mg/m3	50 mg/m3	99 mg/m3
silica welding fumes	Silica, amorphous fume	0.3 mg/m3	0.3 mg/m3	1.6 mg/m3
molybdenum fume	Molybdenum	10 mg/m3	10 mg/m3	17 mg/m3

Ingredient	Original IDLH	Revised IDLH
welding fumes	Not Available	Not Available
iron oxide fume	N.E. mg/m3 / N.E. ppm	2,500 mg/m3
chromium fume	N.E. mg/m3 / N.E. ppm	250 mg/m3
copper fume	N.E. mg/m3 / N.E. ppm	100 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
silica welding fumes	Not Available	Not Available
molybdenum fume	N.E. mg/m3 / N.E. ppm	5,000 mg/m3

MATERIAL DATA

Exposure controls

Appropriate engineering controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.
Special ventilation requirements apply for processes which result in the generation of barium, chromium, lead, or nickel fume and in those processes which generate ozone.
The use of mechanical ventilation by local exhaust systems is required as a minimum in all circumstances (including outdoor work). (In confined spaces always check that oxygen has not been depleted by excessive rusting of steel or snowflake corrosion of aluminium)
Local exhaust systems must be designed to provide a minimum capture velocity at the fume source, away from the worker, of 0.5 metre/sec. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

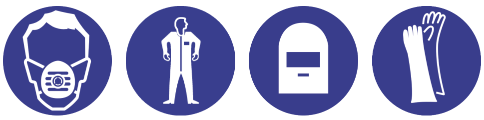
Type of Contaminant:	Air Speed:
welding, brazing fumes (released at relatively low velocity into moderately still air)	0.5-1.0 m/s (100-200 f/min.)

Within each range the appropriate value depends on:

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

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

Personal protection



Eye and face protection

Welding helmet with suitable filter. Welding hand shield with suitable filter.

- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lens or restrictions on use, should be created for each workplace or task. This should

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	- include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] - 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.
Skin protection	See Hand protection below
Hands/feet protection	Welding Gloves Safety footwear
Body protection	See Other protection below
Other protection	Overalls - Eyewash unit. Aprons, sleeves, shoulder covers, leggings or spats of pliable flame resistant leather or other suitable materials may also be required in positions where these areas of the body will encounter hot metal.
Thermal hazards	Not Available

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
Talweld 80Ni1, 80Ni2, D2, Cu, 110, B2, B3, B6, B9 Not Available

Material	CPI

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

Not Available

Not Applicable

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Solid metal welding wire with no odour, shaped as wire of various diameters, 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

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Melting point / freezing point (°C)	~1500	**Viscosity (cSt)**	Not Applicable
Initial boiling point and boiling range (°C)	Not Applicable	**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 Available	**VOC g/L**	Not Available

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	Manganese fume is toxic and produces nervous system effects characterised by tiredness. Acute poisoning is rare although acute inflammation of the lungs may occur. A chemical pneumonia may also result from frequent exposure. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure. Harmful levels of ozone may be found when working in confined spaces. Symptoms of exposure include irritation of the upper membranes of the respiratory tract and lungs as well as pulmonary (lung) changes including irritation, accumulation of fluid (congestion and oedema) and in some cases haemorrhage. Exposure may aggravate any pre-existing lung condition such as bronchitis, asthma or emphysema. Shielding gases may act as simple asphyxiants if significant levels are allowed to accumulate. Oxygen monitoring may be necessary. Copper poisoning following exposure to copper dusts and fume may result in headache, cold sweat and weak pulse. Capillary, kidney, liver and brain damage are the longer term manifestations of such poisoning. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure. Bronchial and alveolar exudate are apparent in animals exposed to molybdenum by inhalation. Molybdenum fume may produce bronchial irritation and moderate fatty changes in liver and kidney.
Ingestion	Not normally a hazard due to physical form of product.
Skin Contact	Nickel dusts, fumes and salts are potent contact allergens and sensitisers producing a dermatitis known as "nickel" rash. In the absence of properly designed ventilation systems or where respiratory protective devises are inadequate, up to 10%

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	of exposed workers are expected to be symptomatic. 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.
Eye	Fumes from welding/brazing operations may be irritating to the eyes.
Chronic	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. 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. Ozone is suspected to produce lung cancer in laboratory animals; no reports of this effect have been documented in exposed human populations. 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 Cr_2O_3 or double oxides with iron. These chromium (III) compounds are generally biologically inert. 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. Regular exposure to nickel fume, as the oxide, may result in "metal fume fever" a sometimes debilitating upper respiratory tract condition resembling influenza. Symptoms include malaise, fever, weakness, nausea and may appear quickly if operations occur in closed or poorly ventilated areas. Pulmonary oedema, pulmonary fibrosis and asthma has been reported in welders using nickel alloys; level of exposure are generally not available and case reports are often confounded by mixed exposures to other agents. 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.

	TOXICITY	IRRITATION
Talweld 80Ni1, 80Ni2, D2, Cu, 110, B2, B3, B6, B9	Not Available	Not Available
welding fumes	TOXICITY	IRRITATION
	Not Available	Not Available
iron oxide fume	TOXICITY	IRRITATION
	Oral (rat) LD50: >5000 mg/kg[1]	Not Available
chromium fume	TOXICITY	IRRITATION
	Not Available	Not Available
copper fume	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg[1]	Not Available
	Inhalation (rat) LC50: 0.733 mg/l4 h[1]	
	Inhalation (rat) LC50: 1.03 mg/l4 h[1]	
	Inhalation (rat) LC50: 1.67 mg/l4 h[1]	
	Oral (rat) LD50: 300500 mg/kg[1]	
manganese fume	TOXICITY	IRRITATION
	Oral (rat) LD50: >2000 mg/kg[1]	Eye (rabbit) 500mg/24H Mild
		Skin (rabbit) 500mg/24H Mild

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	TOXICITY	IRRITATION
nickel fume	Oral (rat) LD50: 5000 mg/kg[2]	Not Available
silica welding fumes	Dermal (rabbit) LD50: >5000 mg/kg[1]	No data [RTECS]
	Oral (rat) LD50: 3160 mg/kg[2]	
molybdenum fume	dermal (rat) LD50: >2000 mg/kg[1]	Not Available
	Oral (rat) LD50: >2000 mg/kg[1]	
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*	

WELDING FUMES

WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
Most welding is performed using electric arc processes - manual metal arc, metal inert gas (MIG) and tungsten inert gas welding (TIG) – and most welding is on mild steel.
There has been considerable evidence over several decades regarding cancer risks in relation to welding activities. Several case-control studies reported excess risks of ocular melanoma in welders. This association may be due to the presence in some welding environments of fumes of thorium-232, which is used in tungsten welding rods.
Different welding environments may present different and complex profiles of exposures. In one study to characterise welding fume aerosol nanoparticles in mild steel metal active gas welding showed a mass median diameter (MMMD) of 200-300 nm. A widespread consensus seems to have formed to the effect that some welding environments, notably in stainless steel welding, do carry risks of lung cancer. This widespread consensus is in part based on empirical evidence regarding risks among stainless steel welders and in part on the fact that stainless steel welding entails moderately high exposure to nickel and chromium VI compounds, which are recognised lung carcinogens. The corollary is that welding without the presence of nickel and chromium VI compounds, namely mild-steel welding, should not carry risk. But it appears that this line of reasoning in not supported by the accumulated body of epidemiologic evidence. While there remained some uncertainty about possible confounding by smoking and by asbestos, and some possible publication bias, the overwhelming evidence is that there has been an excess risk of lung cancer among welders as a whole in the order of 20%-40%.The most begrudging explanation is that there is an as-yet unexplained common reason for excess lung cancer risks that applies to all types of welders. It has been have proposed that iron fumes may play such a role, and some Finnish data appear to support this hypothesis, though not conclusively. This hypothesis would also imply that excess lung cancer risks among welders are not unique to welders, but rather may be shared among many types of metal working occupations.
Welders are exposed to a range of fumes and gases (evaporated metal, metal oxides, hydrocarbons, nanoparticles, ozone, oxides of nitrogen (NOx)) depending on the electrodes, filler wire and flux materials used in the process, but also physical exposures such as electric and magnetic fields (EMF) and ultraviolet (UV) radiation. Fume particles contain a wide variety of oxides and salts of metals and other compounds, which are produced mainly from electrodes, filler wire and flux materials. Fumes from the welding of stainless-steel and other alloys contain nickel compounds and chromium[VI] and [III].
Ozone is formed during most electric arc welding, and exposures can be high in comparison to the exposure limit, particularly during metal inert gas welding of aluminium. Oxides of nitrogen are found during manual metal arc welding and particularly during gas welding. Welders who weld painted mild steel can also be exposed to a range of organic compounds produced by pyrolysis.
In one study particle elemental composition was mainly iron and manganese. Ni and Cr exposures were very low in the vicinity of mild steel welders, but much higher in the background in the workshop where there presumably was some stainless steel welding.
Personal exposures to manganese ranged from 0.01-4.93 mg/m3 and to iron ranged from 0.04-16.29 mg/m3 in eight Canadian welding companies. Types of welding identified were mostly (90%) MIG mild steel, MIG stainless steel, and TIG aluminum. Carbon monoxide levels were less than 5.0 ppm (at source) and ozone levels varied from 0.4-0.6 ppm (at source).
Welders, especially in shipyards, may also be exposed to asbestos dust. Physical exposures such as electric and magnetic fields (EMF) and ultraviolet (UV) radiation are also common.
In all, the in vivo studies suggest that different welding fumes cause varied responses in rat lungs in vivo , and the toxic effects typically correlate with the metal composition of the fumes and their ability to produce free radicals. In many studies both soluble and insoluble fractions of the stainless steel welding fumes were required to produce most types of effects, indicating that the responses are not dependent exclusively on the soluble metals.
Lung tumourigenicity of welding fumes was investigated in lung tumour susceptible (A/J) strain of mice. Male mice were exposed by pharyngeal aspiration four times (once every 3 days) to 85 ug of gas metal arc-mild steel (GMA-MS), GMA-SS, or manual metal arc-SS (MMA-SS) fume. At 48 weeks post-exposure, GMA-SS caused the greatest increase in tumour multiplicity and incidence, but did not differ from sham exposure. Tumour incidence in the GMA-SS group versus sham control was close to significance at 78 weeks post exposure. Histopathological analysis of the lungs of these mice showed the GMA-SS group having an increase in preneoplasia/tumour multiplicity and incidence compared to the GMA-MS and sham groups at 48 weeks. The increase in incidence in the GMA-SS exposed mice was significant compared to the GMA-MS group but not to the sham-exposed animals, and the difference in incidence between the GMA-SS and MMA-SS groups was of border-line significance ($p = 0.06$). At 78 week s post-exposure, no statistically significant differences.

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	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. Not available. Refer to individual constituents.
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. 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. No significant acute toxicological data identified in literature search. 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.
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. **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [*National Toxicology Program: U.S. Dep. of Health & Human Services 2002*]
SILICA WELDING FUMES	For silica amorphous: When experimental animals inhale synthetic amorphous silica (SAS) dust, it dissolves in the lung fluid and is rapidly eliminated. If swallowed, the vast majority of SAS is excreted in the faeces and there is little accumulation in the body. Following absorption across the gut, SAS is eliminated via urine without modification in animals and humans. SAS is not

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	expected to be broken down (metabolised) in mammals. After ingestion, there is limited accumulation of SAS in body tissues and rapid elimination occurs. Intestinal absorption has not been calculated, but appears to be insignificant in animals and humans. SASs injected subcutaneously are subjected to rapid dissolution and removal. There is no indication of metabolism of SAS in animals or humans based on chemical structure and available data. In contrast to crystalline silica, SAS is soluble in physiological media and the soluble chemical species that are formed are eliminated via the urinary tract without modification. Both the mammalian and environmental toxicology of SASs are significantly influenced by the physical and chemical properties, particularly those of solubility and particle size. SAS has no acute intrinsic toxicity by inhalation. Adverse effects, including suffocation, that have been reported were caused by the presence of high numbers of respirable particles generated to meet the required test atmosphere. These results are not representative of exposure to commercial SASs and should not be used for human risk assessment. Though repeated exposure of the skin may cause dryness and cracking, SAS is not a skin or eye irritant, and it is not a sensitiser. Repeated-dose and chronic toxicity studies confirm the absence of toxicity when SAS is swallowed or upon skin contact. Long-term inhalation of SAS caused some adverse effects in animals (increases in lung inflammation, cell injury and lung collagen content), all of which subsided after exposure. Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted with SAS in a number of species, at airborne concentrations ranging from 0.5 mg/m3 to 150 mg/m3. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m3. When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m3. The difference in values may be explained by different particle size, and therefore the number of particles administered per unit dose. In general, as particle size decreases so does the NOAEL/LOAEL. Neither inhalation nor oral administration caused neoplasms (tumours). SAS is not mutagenic in vitro. No genotoxicity was detected in in vivo assays. SAS does not impair development of the foetus. Fertility was not specifically studied, but the reproductive organs in long-term studies were not affected. In humans, SAS is essentially non-toxic by mouth, skin or eyes, and by inhalation. Epidemiology studies show little evidence of adverse health effects due to SAS. Repeated exposure (without personal protection) may cause mechanical irritation of the eye and drying/cracking of the skin. There is no evidence of cancer or other long-term respiratory health effects (for example, silicosis) in workers employed in the manufacture of SAS. Respiratory symptoms in SAS workers have been shown to correlate with smoking but not with SAS exposure, while serial pulmonary function values and chest radiographs are not adversely affected by long-term exposure to SAS. 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. Reports indicate high/prolonged exposures to amorphous silicas induced lung fibrosis in experimental animals; in some experiments these effects were reversible. [PATTYS]
MOLYBDENUM FUME	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. No significant acute toxicological data identified in literature search.

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 required to make classification available*
✖ *– Data available but does not fill the criteria for classification*
⦸ *– Data Not Available to make classification*

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

NOT AVAILABLE

Ingredient	Endpoint	Test Duration	Effect	Value	Species	BCF
welding fumes	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
iron oxide fume	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
chromium fume	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
copper fume	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
manganese fume	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
nickel fume	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
silica welding fumes	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
molybdenum fume	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available

Persistence and degradability

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

Bioaccumulative potential

Ingredient	Bioaccumulation
	No Data available for all ingredients

Mobility in soil

Ingredient	Mobility
	No Data available for all ingredients

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

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

SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

SECTION 15 REGULATORY INFORMATION

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

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

Not Applicable

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

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

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

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

COPPER FUME(7440-50-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

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

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

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

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

SILICA WELDING FUMES(69012-64-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)

MOLYBDENUM FUME(7439-98-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (manganese fume; nickel fume; copper fume; chromium fume; silica welding fumes; iron oxide fume; molybdenum fume)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	N (manganese fume; nickel fume; copper fume; chromium fume; silica welding fumes; molybdenum fume)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
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

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 (M)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.

end of SDS