Hobart Filler Gas Shielded Carbon and Low Alloy Steel

Weldwell

Chemwatch Hazard Alert Code: **4**

Chemwatch: **5314-28**
Version No: **3.1.1.1**
Safety Data Sheet according to HSNO Regulations

Issue Date: **09/05/2018**
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SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	Hobart Filler Gas Shielded Carbon and Low Alloy Steel
Synonyms	FABCO HORNET, FABCOR 86R
Other means of identification	Not Available

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

Relevant identified uses	Tubular arc welding electrodes for flux cored, metal cored and composite submerged arc welding.

Details of the supplier of the safety data sheet

Registered company name	**Weldwell**
Address	59 Thames Street Pandora Napier 4110 New Zealand
Telephone	+64 6 834 1600
Fax	+64 6 835 4568
Website	http://www.weldwell.co.nz
Email	info@weldwell.co.nz

Emergency telephone number

Association / Organisation	**Not Available**
Emergency telephone numbers	0800 POISON (0800 764 766) National Poisons Centre
Other emergency telephone numbers	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

Considered a Hazardous Substance according to the criteria of the New Zealand Hazardous Substances New Organisms legislation. Not regulated for transport of Dangerous Goods.

CHEMWATCH HAZARD RATINGS

		Min / Max
Flammability	0	
Toxicity	2	
Body Contact	0	
Reactivity	0	
Chronic	4	

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

Classification [1]	Acute Toxicity (Inhalation) Category 4, Carcinogenicity Category 1
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*
Determined by Chemwatch using GHS/HSNO criteria	6.1D (inhalation), 6.7A

Label elements

Hazard pictogram(s)	
SIGNAL WORD	**DANGER**

Hazard statement(s)

H332	Harmful if inhaled.
H350	May cause cancer.

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Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P261	Avoid breathing dust/fumes.

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 in accordance with local regulations.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
		tubular arc welding electrode, in use generates
Not Available	>60	welding fumes
		as
7429-90-5.		aluminium fumes
7440-50-8.		copper fume
1309-37-1.		iron oxide fume
7439-96-5.		manganese fume
7439-98-7		molybdenum fume
69012-64-2		silica, fumes

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. ▸ 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.

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	▶ 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.
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 the physical form of product. The material is a physical irritant to the gastro-intestinal tract

Indication of any immediate medical attention and special treatment needed

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

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

[Ellenhorn and Barceloux: Medical Toxicology]

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

[*Ellenhorn and Barceloux: Medical Toxicology*]

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

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

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	Welding electrodes should not be allowed to come into contact with strong acids or other substances which are corrosive to metals.

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.

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

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Minor Spills	Clean up all spills immediately. Wear impervious gloves and safety glasses. Use dry clean up procedures and avoid generating dust. Place in suitable containers for disposal. Place spilled material in clean, dry, sealable, labelled container.
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. ▸ 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	▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ **DO NOT** allow material to contact humans, exposed food or food utensils. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
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.

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
New Zealand Workplace Exposure Standards (WES)	aluminium fumes	Aluminium, as Al: Welding fumes	5 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	aluminium fumes	Aluminium, as Al: Metal dust	10 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	aluminium fumes	Aluminium, as Al: Pyro powders	5 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	copper fume	Copper fume Dusts and mists, as Cu	0.2; 1 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	iron oxide fume	Iron oxide dust and fume (Fe2O3), as Fe	5 mg/m3	Not Available	Not Available	(w) - A range of airborne contaminants are associated with gas and arc welding. The type of metal being welded, the electrode employed and the welding process will all influence the composition and amount of fume. Gaseous products such as oxides of nitrogen, carbon monoxide and ozone may also be produced. In the absence of specific substances such as chromium, and where conditions do not support the generation of toxic gases, the fume concentration inside the welder's helmet should not exceed 5 mg/m3.
New Zealand Workplace Exposure Standards (WES)	iron oxide fume	Rouge	10 mg/m3	Not Available	Not Available	(w) - A range of airborne contaminants are associated with gas and arc welding. The type of metal being welded, the electrode employed and the welding process will all influence the composition and amount of fume. Gaseous products such as oxides of nitrogen, carbon monoxide and ozone may also be produced. In the absence of specific substances such as chromium, and where conditions do not support the generation of toxic

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						gases, the fume concentration inside the welder's helmet should not exceed 5 mg/m3.
New Zealand Workplace Exposure Standards (WES)	manganese fume	Fume, as Mn	1 mg/m3	3 mg/m3	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	molybdenum fume	Molybdenum, as Mo: Insoluble compounds	10 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	silica, fumes	Silica fume	2 mg/m3	Not Available	Not Available	(r) - The value for respirable dust.

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
copper fume	Copper	3 mg/m3	33 mg/m3	200 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
molybdenum fume	Molybdenum	30 mg/m3	330 mg/m3	2,000 mg/m3
silica, fumes	Silica, amorphous fume	45 mg/m3	500 mg/m3	3,000 mg/m3

Ingredient	Original IDLH	Revised IDLH
welding fumes	Not Available	Not Available
aluminium fumes	Not Available	Not Available
copper fume	100 mg/m3	Not Available
iron oxide fume	2,500 mg/m3	Not Available
manganese fume	Not Available	Not Available
molybdenum fume	Not Available	Not Available
silica, fumes	Not Available	Not Available

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 aluminium, copper, fluoride, manganese or zinc fume.

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

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

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

Within each range the appropriate value depends on:

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

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

For manual arc welding operations the nature of ventilation is determined by the location of the work.

- For outdoor work, natural ventilation is generally sufficient.
- For indoor work, conducted in open spaces, use mechanical (general exhaust or plenum) ventilation. (Open work spaces exceed 300 cubic metres per welder)
- For work conducted in limited or confined spaces, mechanical ventilation, using local exhaust systems, is required. (In confined spaces always check that oxygen has not been depleted by excessive rusting of steel or snowflake corrosion of aluminium)

Mechanical or local exhaust ventilation may not be required where the process working time does not exceed 24 mins. (in an 8 hr. shift) provided the work is intermittent (a maximum of 5 mins. every hour). Local exhaust systems must be designed to provide a minimum capture velocity at the fume source, away

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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 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection.
Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.
An approved self contained breathing apparatus (SCBA) may be required in some situations.
Provide adequate ventilation in warehouse or closed storage area. 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.

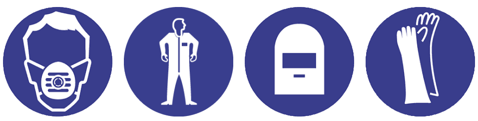
Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 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 solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Personal protection



Eye and face protection

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

For submerged arc welding use a lens shade which gives just sufficient arc brightness to allow weld pool control.
Welding helmet with suitable filter. Welding hand shield with suitable filter.

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) Welding Gloves Safety footwear
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. 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.

Respiratory protection

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	Shiny metallic grey or copper solid.

Physical state	Manufactured	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Applicable	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**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)	Not Applicable	**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	Product is considered stable and hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	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. 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.

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	Acute carbon monoxide exposure can mimic acute gastroenteritis or food poisoning with accompanying nausea and vomiting. Rapidly fatal cases of poisoning are characterised by congestion and hemorrhages in all organs. The extent of the tissue and organ damage is related to the duration of the post-hypoxic unconsciousness. Exposure to carbon monoxide can result in immediate effects and, depending on the severity of the exposure, delayed effects. These delayed effects may occur days to weeks after the initial exposure. Signs of brain or nerve injury may appear at any time within three weeks following an acute exposure. Characteristically, those patients manifesting delayed neuropathology are middle aged or older. Most of the neurological symptoms associated with carbon monoxide exposure can resolve within a year but memory deficits and gait disturbances may remain Symptoms of poisoning resulting from carbon monoxide exposure include respiratory disorders, diarrhoea and shock. Carbon monoxide competes with oxygen for haemoglobin binding sites and has a 240-fold affinity for these sites compared to oxygen. In addition to oxygen deficiency further disability is produced by the formation of carboxymyoglobin (COHb) in muscles, to produce disturbances in muscle metabolism, particularly that of the heart. The tissues most affected by carbon monoxide are those which are most sensitive to oxygen deprivation such as the brain and the heart. The overt lesion in these tissues is mostly haemorrhage. The severe headache associated with exposure is believed to be caused by cerebral oedema and increased intracranial pressure resulting from excessive transudate leakage of fluids through the hypoxic capillaries. Carbon monoxide induced hypoxia in the cochlea and brain stem leads to central hearing loss and vestibular dysfunction (vertigo, nausea, vomiting) with the vestibular symptoms usually more prominent than the hearing loss At low levels carbon monoxide may cause poor concentration, memory and vision problems, vertigo, muscular weakness and loss of muscle coordination, rapid and stretorous breathing, intermittent heart beat, loss of sphincter control and rarely coma and death. At higher levels (200 ppm for 2-3 hours), it may cause headaches, fatigue and nausea. At very high levels (400 ppm) the symptoms intensify and will be life-threatening after three hours. Exposure to levels of 1200 ppm or greater are immediately dangerous to life. When carbon monoxide levels in air exceed 3% (30,000 ppm), death occurs almost at once. Carbon monoxide is not a cumulative poison since COHb is fully dissociable and once exposure has ceased, the hemoglobin will revert to oxyhemoglobin. The biological half life of carbon monoxide in the blood in sedentary adults is 2- 5 hours and the elimination becomes slower as the concentration decreases. 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.
Ingestion	Not normally a hazard due to physical form of product.
Skin Contact	Skin contact does **not** normally present a hazard, though it is always possible that occasionally individuals may be found who react to substances usually regarded as inert. 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.
Eye	Fumes from welding/brazing operations may be irritating to the eyes. Ultraviolet (UV) radiation can also damage the lens of the eye. Many arc welders are aware of the condition known as "arc-eye," a sensation of sand in the eyes. This condition is caused by excessive eye exposure to UV. Exposure to ultraviolet rays may also increase the skin effects of some industrial chemicals (coal tar and cresol compounds, for example). Exposure of the human eye to intense visible light can produce adaptation, pupillary reflex, and shading of the eyes. Such actions are protective mechanisms to prevent excessive light from being focused on the retina. In the arc welding process, eye exposure to intense visible light is prevented for the most part by the welder's helmet. However, some individuals have sustained retinal damage due to careless "viewing" of the arc. At no time should the arc be observed without eye protection.
Chronic	Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminum oxide during abrasives production. Very fine Al_2O_3 powder was not fibrogenic in rats, guinea pigs, or hamsters when inhaled for 6 to 12 months and sacrificed at periods up to 12 months following the last exposure. When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C. The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis(aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity. Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction. There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveolii (sub 5 um) are able to produce pathogenic effects in the lungs. Repeated or prolonged exposure may also damage the liver and may cause a decrease in the heart rate. Systemic poisoning may result from inhalation or chronic ingestion of manganese containing substances. Progressive and permanent disability can occur from chronic manganese poisoning if it is not treated, but it is not fatal. Chronic exposure has been associated with two major effects; bronchitis/pneumonitis following inhalation of manganese dusts and "manganism", a neuropsychiatric disorder that may also arise from inhalation exposures. Chronic exposure to low levels may result in the accumulation of toxic concentrations in critical organs. The brain in particular appears to sustain cellular damage to the ganglion. Symptoms appear before any pathology is evident and may include a mask-like facial expression, spastic gait, tremors, slurred speech, sometimes dystonia (disordered muscle tone), fatigue, anorexia, asthenia (loss of strength and energy), apathy and the inability to concentrate. Insomnia may be an early finding. Chronic poisoning may occur over a 6-24 month period depending on exposure levels. The onset of chronic manganese poisoning is insidious, with apathy, anorexia weakness, headache and spasms. Manganese psychosis follows with certain definitive features: unaccountable laughter, euphoria, impulsive acts, absentmindedness, mental confusion, aggressiveness and hallucinations. The final stage is characterised by speech difficulties, muscular twitching, spastic gait and other nervous system effects. Symptoms resemble those of Parkinson's disease. Rat studies indicate the gradual accumulation of brain manganese to produce lesions mimicking those found in Parkinsonism. If the disease is diagnosed whilst still in the early stages and the patient is removed from exposure, the course may be reversed. Inhalation of manganese fumes may cause 'metal fume fever' characterised by flu-like symptoms: fever, chill, nausea, weakness and body aches. Manganese dust is no longer believed to be a causative factor in pneumonia. If there is any relationship at all, it appears to be as an aggravating factor to a preexisting condition. Prolonged or repeated eye contact may result in conjunctivitis. Manganese is an essential trace element in all living organisms with the level of tissue manganese remaining remarkably constant throughout life. Persons, exposed for long periods to molybdenum oxides, suffer from anaemia. Animals exposed to certain insoluble molybdenum compounds show anorexia, diarrhoea, weight loss, listlessness, and liver and kidney damage. Molybdenum disturbs bone metabolism, giving rise to lameness, bone joint abnormalities, osteoporosis and high serum phosphatase levels.Cattle, rabbits, and chicks on high dietary levels of molybdenum exhibited deformities of joints of the extremities. Low molybdenum intake has been attributed to the high incidence of oesophageal cancer in South Africa among the Bantu of

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Transkei, in China and in Russia.
Chronic exposure of workers in Russian molybdenum-copper plant resulted in a fall in the albumin/globulin ratio owing to a rise in globulins (particularly alpha-globulins) which is interpreted as evidence of liver dysfunction with hyperbilirubinaemia. Hepatotoxic effects are also found in animals given molybdenum salts with a rise in alpha-globulin levels, hypoalbuminaemia and increased serum bilirubin reported. Other reported biochemical effects include an early depletive effect on tissue nicotinamide nucleotides, hyperaminoaciduria, reduction in red blood cell life-span and hyper-thyroidism.
Industrial exposure to some insoluble molybdenum compounds is thought to have resulted in an increased incidence of weakness, fatigue, anorexia, headache and joint and muscular pain. Under the conditions of a 2-year inhalation study* there was equivocal evidence of carcinogenic activity of molybdenum trioxide in male rats, male mice and female mice based on a marginally significant positive trend of alveolar/bronchiolar adenoma or carcinoma. There was no evidence of carcinogenic activity in female rats exposed to 10, 30 and 100 mg/m3. Exposure of male and female rats to molybdenum trioxide by inhalation resulted in increased incidences of chronic alveolar inflammation, hyaline degeneration of the olfactory epithelium (females), hyaline degeneration of the respiratory epithelium and squamous metaplasia of the epiglottis. Exposure of female and male mice to molybdenum trioxide resulted in an increased incidence of metaplasia of the alveolar epithelium, histiocyte cellular inflammation (males), hyaline degeneration of the respiratory epithelium, hyaline degeneration of the olfactory epithelium (females), squamous metaplasia of the epiglottis, and hyperplasia of the larynx.
Guinea pigs exposed to molybdenum trioxide dust for 1 hour daily at 250 mg/m3 showed extreme irritation. Symptoms include loss of appetite, weight loss, diarrhoea, muscular incoordination and loss of hair. Of the 51 animals exposed, 26 died after the tenth exposure. Exposure to freshly generated MoO2 fume under about the same exposure conditions proved unexpectedly less toxic, with only 8.3% mortality compared with 51% mortality with the dust, and no mortality when the exposure level was reduced to about one-third (57 mg Mo/m3). Explanation for this unexpected finding was felt to reside in the more rapid solution and elimination of the large surface area fume particle. [Patty's]
Exposure of male and female rats to molybdenum trioxide resulted in the development of respiratory system lesions. In the lung, the incidence and severity of chronic alveolar inflammation increased with increasing exposure concentration in male and female rats. In some male rats, exposure to the material resulted in alveolar/ bronchiolar adenomas or carcinomas. Lesions in the nose (hyaline degeneration) and larynx (squamous metaplasia) were considered to be a non-specific defensive or adaptive response to chronic inhalation exposure. Inhalation exposure of mice to molybdenum trioxide was associated with the development of lung neoplasms and an increased incidence of alveolar/ bronchiolar carcinoma or adenoma in both sexes. Chronic inflammatory lesions were not present in the lungs. Lesions of the nose and larynx were similar to those observed in rats.
Molybdenum trioxide was not mutagenic in any of five strains of Salmonella typhimurium and did not induce sister chromatid exchanges or chromosomal aberrations in cultured Chinese hamster ovary cells in vitro. All tests were conducted with or without S9 metabolic activation enzymes.
Pneumoconiosis has been described in experimental animals exposed sub-chronically to molybdenum trioxide.
The mechanism of molybdenum trioxide action in lung carcinogenicity is not known; the material is not mutagenic. Non-neoplastic lesions of the nose and larynx of rats and in the nose, larynx and lungs of mice were apparently due to the development of a more durable epithelium in response to chronic exposure.
The US Department of Health and Human Services (1) concluded that there was equivocal evidence of carcinogenic activity in male F344/N rats based on a marginally significant positive trend of alveolar/ bronchiolar adenoma or carcinoma; that there was no evidence of carcinogenic activity in female F344/N rats; that there was some evidence of carcinogenic activity in male B6C3F1 mice and that there was evidence of carcinogenic activity in female B6C3F1 mice
National Toxicology Program: Technical Report Series 462, April 1997

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.
Long-term (chronic) exposure to low levels of carbon monoxide may produce heart disease and damage to the nervous system. Exposure of pregnant animals to carbon monoxide may cause low birthweight, increased foetal mortality and nervous system damage to the offspring.
Carbon monoxide is a common cause of fatal poisoning in industry and homes. Non fatal poisoning may result in permanent nervous system damage. Carbon monoxide reduces the oxygen carrying capacity of the blood. Effects on the body are considered to be reversible as long as brain cell damage or heart failure has not occurred. Avoid prolonged exposure, even to small concentrations. A well-established and probably causal relationship exists between maternal smoking (resulting in carboxyhaemoglobin levels of 2-7% in the foetus) and low birth weight. There also appears to be a dose-related increase in perinatal deaths and a retardation of mental ability in infants born to smoking mothers.
The foetus and newborn infant are considered to be very susceptible to CO exposure for several reasons:

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

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

	TOXICITY	IRRITATION
Hobart Filler Gas Shielded Carbon and Low Alloy Steel	Not Available	Not Available
welding fumes	Not Available	Not Available
aluminium fumes	Oral (rat) LD50: >2000 mg/kg[1]	Not Available

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	TOXICITY	IRRITATION
copper fume	dermal (rat) LD50: >2000 mg/kg[1]	Not Available
	Inhalation (rat) LC50: 0.733 mg/l4 h[1]	
	Oral (rat) LD50: 300-500 mg/kg[1]	
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
molybdenum fume	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg[1]	Not Available
	Oral (rat) LD50: >2000 mg/kg[1]	
silica, fumes	TOXICITY	IRRITATION
	Oral (rat) LD50: 3160 mg/kg[2]	Eye (rabbit): non-irritating *
		Skin (rabbit): non-irritating *

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

Most welding is performed using electric arc processes - manual metal arc, metal inert gas (MIG) and tungsten inert gas welding (TIG) – and most welding is on mild steel.
In 2017, an IARC working group has determined that "sufficient evidence exists that welding fume is a human lung carcinogen (Group 1).
A complicating factor in classifying welding fumes is its complexity. Generally, welding fume is a mixture of metal fumes (i.e., iron, manganese, chromium, nickel, silicon, titanium) and gases (i.e., carbon monoxide, ozone, argon, carbon dioxide). Welding fume can contain varying concentrations of individual components that are classified as human carcinogens, including hexavalent chrome and nickel. However the presence of such metals and the intensity of exposure to each differ significantly according to a number of variables, including the type of welding technique used and the composition of the base metal and consumable. Nonetheless, IARC did not differentiate between these variables in its decision.
There has been considerable evidence over several decades regarding cancer risks in relation to welding activities. Several case-control studies reported excess risks of ocular melanoma in welders. This association may be due to the presence in some welding environments of fumes of thorium-232, which is used in tungsten welding rods
Different welding environments may present different and complex profiles of exposures. In one study to characterise welding fume aerosol nanoparticles in mild steel metal active gas welding showed a mass median diameter (MMMD) of 200-300 nm. A widespread consensus seems to have formed to the effect that some welding environments, notably in stainless steel welding, do carry risks of lung cancer. This widespread consensus is in part based on empirical evidence regarding risks among stainless steel welders and in part on the fact that stainless steel welding entails moderately high exposure to nickel and chromium VI compounds, which are recognised lung carcinogens. The corollary is that welding without the presence of nickel and chromium VI compounds, namely mild-steel welding, should not carry risk. But it appears that this line of reasoning 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
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

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	WARNING: This substance has been classified by the IARC as Group 1: **CARCINOGENIC TO HUMANS**. Not available. Refer to individual constituents.
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.
SILICA, 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 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]

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 available to make classification*
⃠ *– Data Not Available to make classification*

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Hobart Filler Gas Shielded Carbon and Low Alloy Steel

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
Not Available	Not Available	Not Available	Not Available	Not Available

welding fumes

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
Not Available	Not Available	Not Available	Not Available	Not Available

aluminium fumes

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	0.078-0.108mg/L	2
EC50	48	Crustacea	0.7364mg/L	2
EC50	96	Algae or other aquatic plants	0.0054mg/L	2
BCF	360	Algae or other aquatic plants	9mg/L	4
NOEC	72	Algae or other aquatic plants	>=0.004mg/L	2

copper fume

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	0.0028mg/L	2

Continued...

	EC50	48	Crustacea	0.001mg/L	5
	EC50	72	Algae or other aquatic plants	0.013335mg/L	4
	BCF	960	Fish	200mg/L	4
	EC25	6	Algae or other aquatic plants	0.00150495mg/L	4
	NOEC	96	Crustacea	0.0008mg/L	4
iron oxide fume	**ENDPOINT**	**TEST DURATION (HR)**	**SPECIES**	**VALUE**	**SOURCE**
	LC50	96	Fish	0.05mg/L	2
	EC50	72	Algae or other aquatic plants	18mg/L	2
	NOEC	504	Fish	0.52mg/L	2
manganese fume	**ENDPOINT**	**TEST DURATION (HR)**	**SPECIES**	**VALUE**	**SOURCE**
	LC50	96	Fish	>3.6mg/L	2
	EC50	48	Crustacea	>1.6mg/L	2
	EC50	72	Algae or other aquatic plants	2.8mg/L	2
	BCFD	37	Algae or other aquatic plants	2.2mg/L	4
	NOEC	48	Crustacea	1.6mg/L	2
molybdenum fume	**ENDPOINT**	**TEST DURATION (HR)**	**SPECIES**	**VALUE**	**SOURCE**
	LC50	96	Fish	609.1mg/L	2
	EC50	72	Algae or other aquatic plants	289.2mg/L	2
	BCF	336	Algae or other aquatic plants	64mg/L	4
	NOEC	672	Crustacea	0.67mg/L	2
silica, fumes	**ENDPOINT**	**TEST DURATION (HR)**	**SPECIES**	**VALUE**	**SOURCE**
	EC50	72	Algae or other aquatic plants	ca.250mg/L	2

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

DO NOT discharge into sewer or waterways.

Persistence and degradability

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

Bioaccumulative potential

Ingredient	Bioaccumulation
	No Data available for all ingredients

Mobility in soil

Ingredient	Mobility
	No Data available for all ingredients

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	▸ 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.

Ensure that the hazardous substance is disposed in accordance with the Hazardous Substances (Disposal) Notice 2017

Disposal Requirements

Packages that have been in direct contact with the hazardous substance must be only disposed if the hazardous substance was appropriately removed and cleaned out from the package.
The package must be disposed according to the manufacturer's directions taking into account the material it is made of.
Packages which hazardous content have been appropriately treated and removed may be recycled.

The hazardous substance must only be disposed if it has been treated by a method that changed the characteristics or composition of the substance and it is no longer hazardous.
Only dispose to the environment if a tolerable exposure limit has been set for the substance.
Only deposit the hazardous substance into or onto a landfill or sewage facility or incinerator, where the hazardous substance can be handled and treated appropriately.

SECTION 14 TRANSPORT INFORMATION

Continued...

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (UN): 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

This substance is to be managed using the conditions specified in an applicable Group Standard

HSR Number	Group Standard
HSR002531	Cleaning Products (Toxic [6.7]) Group Standard 2017
HSR002596	Laboratory Chemicals and Reagent Kits Group Standard 2017
HSR002607	Lubricants (Toxic [6.7]) Group Standard 2017
HSR002586	Fuel Additives (Toxic [6.7]) Group Standard 2017
HSR002520	Aerosols (Toxic [6.7]) Group Standard 2017
HSR002646	Polymers (Toxic [6.7]) Group Standard 2017
HSR002616	Metal Industry Products (Toxic [6.7]) Group Standard 2017
HSR002512	Additives, Process Chemicals and Raw Materials (Toxic [6.7]) Group Standard 2017
HSR002568	Embalming Chemicals (Toxic [6.7]) Group Standard 2017
HSR002679	Surface Coatings and Colourants (Toxic [6.7]) Group Standard 2017
HSR100425	Pharmaceutical Active Ingredients Group Standard 2017
HSR002601	Leather and Textile Products (Toxic [6.7]) Group Standard 2017
HSR002648	Refining Catalysts Group Standard 2017
HSR002545	Construction Products (Toxic [6.7A]) Group Standard 2017
HSR002551	Corrosion Inhibitors (Toxic [6.7]) Group Standard 2017
HSR100757	Veterinary Medicine (Limited Pack Size, Finished Dose) Standard 2017
HSR100758	Veterinary Medicines (Non-dispersive Closed System Application) Group Standard 2017
HSR100759	Veterinary Medicines (Non-dispersive Open System Application) Group Standard 2017
HSR002655	Solvents (Toxic [6.7]) Group Standard 2017
HSR002625	N.O.S. (Toxic [6.1, 6.7]) Group Standard 2017
HSR002639	Photographic Chemicals (Toxic [6.7]) Group Standard 2017
HSR002560	Dental Products (Toxic [6.7]) Group Standard 2017
HSR002687	Water Treatment Chemicals (Toxic [6.7]) Group Standard 2017

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

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

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

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

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

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

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

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

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

Continued...

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals	New Zealand Workplace Exposure Standards (WES)
New Zealand Inventory of Chemicals (NZIoC)	

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

New Zealand Inventory of Chemicals (NZIoC)	New Zealand Workplace Exposure Standards (WES)

Hazardous Substance Location

Subject to the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Quantity beyond which controls apply for closed containers	Quantity beyond which controls apply when use occurring in open containers
Not Applicable	Not Applicable	Not Applicable

Certified Handler

Subject to Part 4 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Class of substance	Quantities
6.7A	10 kg or more, if solid 10 L or more, if liquid

Refer Group Standards for further information

Tracking Requirements

Not Applicable

National Inventory Status

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

Revision Date	09/05/2018
Initial Date	08/02/2018

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.

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

Definitions and abbreviations

PC－TWA: Permissible Concentration-Time Weighted Average
PC－STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
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