CIGWELD GAS AND TIG WELDING RODS

Chemwatch Independent Material Safety Data Sheet
Issue Date: 24-Oct-2011
A317LP

Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

PRODUCT NAME
CIGWELD GAS AND TIG WELDING RODS

SYNONYMS
"Product Code 321334, 321337, 321339, 322045, 321357, 321360, 321370, 321373, 321411, 321412, 321417, 321418, 321423, 321424 Comweld Mild Steel, High Test, Super Steel, LW1, LW1-6, LW1-3"

PRODUCT USE
Consumable mild steel filler rods used for oxy-acetylene flame welding and/or Gas Tungsten Arc-Welding (GTAW)

SUPPLIER
Company: Cigweld Pty Ltd
Address:
71 Gower Street
Preston
VIC, 3072
Australia
Telephone: +61 3 9474 7400
Telephone: +1 1300 654 674
Emergency Tel:+61 3 9474 7400
Email: cigweldsales@cigweld.com.au

Section 2 - HAZARDS IDENTIFICATION

STATEMENT OF HAZARDOUS NATURE
HAZARDOUS SUBSTANCE. NON-DANGEROUS GOODS. According to the Criteria of NOHSC, and the ADG Code.

RISK
Risk Codes Risk Phrases
R40(3) • Limited evidence of a carcinogenic effect.

SAFETY
Safety Codes Safety Phrases
S24 • Avoid contact with skin.
S36 • Wear suitable protective clothing.
S37 • Wear suitable gloves.
S51 • Use only in well ventilated areas.
S09 • Keep container in a well ventilated place.
S401 • To clean the floor and all objects contaminated by this material, use water and detergent.
S13 • Keep away from food, drink and animal feeding stuffs.
S46 • If swallowed, IMMEDIATELY contact Doctor or Poisons Information Centre. (show this container or label).

continued...
Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

<table>
<thead>
<tr>
<th>NAME</th>
<th>CAS RN</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>steel filler rod which upon use generates</td>
<td>Not avail.</td>
<td>&gt;60</td>
</tr>
<tr>
<td>welding fumes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>including</td>
<td></td>
<td></td>
</tr>
<tr>
<td>iron oxide fume</td>
<td>1309-37-1</td>
<td></td>
</tr>
<tr>
<td>manganese fume</td>
<td>7439-96-5</td>
<td></td>
</tr>
<tr>
<td>silica welding fumes</td>
<td>69012-64-2</td>
<td></td>
</tr>
<tr>
<td>copper fume</td>
<td>7440-50-8</td>
<td></td>
</tr>
<tr>
<td>action of arc on air may produce</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ozone</td>
<td>10028-15-6</td>
<td></td>
</tr>
<tr>
<td>nitrogen oxides</td>
<td>Mixture</td>
<td></td>
</tr>
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</table>

Section 4 - FIRST AID MEASURES

SWALLOWED
- Not normally a hazard due to the physical form of product. The material is a physical irritant to the gastro-intestinal tract.

EYE
- 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.

SKIN
- If skin or hair contact occurs:
  - Flush skin and hair with running water (and soap if available).
  - Seek medical attention in event of irritation.

INHALED
- 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.

NOTES TO PHYSICIAN
- 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].

continued...
Section 5 - FIRE FIGHTING MEASURES

EXTINGUISHING MEDIA
• There is no restriction on the type of extinguisher which may be used.

FIRE FIGHTING
• Alert Fire Brigade and tell them location and nature of hazard.
• Wear breathing apparatus plus protective gloves for fire only.
• 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.

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.

HAZCHEM
None

Section 6 - ACCIDENTAL RELEASE MEASURES

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 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 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 MSDS.

continued...
Section 7 - HANDLING AND STORAGE

PROCEDURE FOR 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 storing and handling recommendations.
- Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.

SUITABLE CONTAINER
- Packaging as recommended by manufacturer.
- Check that containers are clearly labelled.

Multi-wall paper container NOTE: Bags should be stacked, blocked, interlocked, and limited in height so that they are stable and secure against sliding or collapse.

STORAGE INCOMPATIBILITY
- Segregate from strong acids and strong oxidisers.

STORAGE REQUIREMENTS
- Store in original containers.
- Keep containers securely sealed.
- Store in a cool, dry, well-ventilated area.
- Store away from incompatible materials and foodstuff containers.
- Protect containers against physical damage and check regularly for leaks.
- Observe manufacturer's storing and handling recommendations.

Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

EXPOSURE CONTROLS

<table>
<thead>
<tr>
<th>Source</th>
<th>Material</th>
<th>TWA mg/m³</th>
<th>Peak ppm</th>
<th>Peak mg/m³</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australia Exposure Standards</td>
<td>welding fumes (Welding fumes (not otherwise classified))</td>
<td>5</td>
<td></td>
<td></td>
<td>(see Chapter 17)</td>
</tr>
<tr>
<td>Australia Exposure Standards</td>
<td>ozone (Ozone)</td>
<td>0.1</td>
<td>0.2</td>
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</tbody>
</table>

The following materials had no OELs on our records
- silica welding fumes: CAS:69012- 64- 2

continued...
Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

MATERIAL DATA

CIGWELD GAS AND TIG WELDING RODS:
■ None assigned. Refer to individual constituents.

WELDING FUMES:
■ In addition to complying with any individual exposure standards for specific contaminants, where current manual welding processes are used, the fume concentration inside the welder’s helmet should not exceed 5 mg/m$^3$, when collected in accordance with the appropriate standard (AS 3640, for example).

- **ES** TWA: 5 mg/m$^3$
- **TLV** TWA: 5 mg/m$^3$, B2 (a substance of variable composition)
- **OES** TWA: 5 mg/m$^3$

Most welding, even with primitive ventilation, does not produce exposures inside the welding helmet above 5 mg/m$^3$. That which does should be controlled (ACGIH). Inspirable dust concentrations in a workers breathing zone shall be collected and measured in accordance with AS 3640, for example. Metal content can be analytically determined by OSHA Method ID25 (ICP-AES) after total digestion of filters and dissolution of captured metals. Sampling of the Respirable Dust fraction requires cyclone separator devices (elutriators) and procedures to comply with AS 2985 (for example).

IRON OXIDE FUME:
■ For iron oxide (ferric oxide):

Inhalation of iron oxide dust or fume may produce a benign pneumoconiosis (siderosis). The TLV-TWA is recommended to minimise the potential for development of X-ray changes in the lung on long-term exposure. These changes are not considered to be associated with any physical impairment of lung function, although more sophisticated physiological testing, including measurement of the lung’s mechanical properties and expiratory lung flow is required to reach firm and final conclusions.

MANGANESE FUME:
■ It is the goal of the ACGIH (and other Agencies) to recommend TLVs (or their equivalent) for all substances for which there is evidence of health effects at airborne concentrations encountered in the workplace.

At this time no TLV has been established, even though this material may produce adverse health effects (as evidenced in animal experiments or clinical experience). Airborne concentrations must be maintained as low as is practically possible and occupational exposure must be kept to a minimum.

NOTE: The ACGIH occupational exposure standard for Particles Not Otherwise Specified (P.N.O.S) does NOT apply.

SILICA WELDING FUMES:
■ The concentration of dust, for application of respirable dust limits, is to be determined from the fraction that penetrates a separator whose size collection efficiency is described by a cumulative log-normal function with a median aerodynamic diameter of 4.0 µm (+-) 0.3 µm and with a geometric standard deviation of 1.5 µm (+-) 0.1 µm, i.e., generally less than 5 µm.

For amorphous crystalline silica (precipitated silicic acid):

Amorphous crystalline silica shows little potential for producing adverse effects on the lung and exposure standards should reflect a particulate of low intrinsic toxicity. Mixtures of amorphous silicas/diatomaceous earth and crystalline silica should be monitored as if they comprise only the crystalline forms.

The dusts from precipitated silica and silica gel produce little adverse effect on pulmonary functions and are not known to produce significant disease or toxic effect.

IARC has classified silica, amorphous as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.

OZONE:
■ for ozone:
Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

NOTE: Detector tubes for ozone, measuring in excess of 0.05 ppm, are commercially available.
Exposure at 0.2 ppm appears to produce mild acute but not cumulative effects. It is thought that exposures of the order of 0.1 ppm will be tolerated by most workers including asthmatics. Chronic exposure at 0.1 ppm or more can induce significant adverse effects in the lower respiratory tract of both normal and atopic individuals.

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

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

Odour Safety Factor (OSF)
OSF=1.1 (OZONE).

NITROGEN OXIDES:

■ For nitrous oxide:
The human reproductive, haematologic and nervous systems show toxic effects after nitrous oxide exposures. Similarities between epidemiologic and animal studies allow the establishment of a TLV-TWA even in the absence of clearly defined dose-response relationships in humans. The TLV-TWA is thus thought to be protective against embryofoetal toxicity (resulting in an increased risk of spontaneous abortion) and significant loss of human psychomotor and cognitive functions. Evidence of organ system impairment has only been evident when nitrous oxide concentrations reach several hundred to several thousand ppm.

For nitric oxide:

○ Odour Threshold: 0.3 to 1 ppm.

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

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

Odour Safety Factor (OSF)
OSF=7.7 (nitric oxide).

for nitrogen dioxide

Odour Threshold Value: 0.11-0.14 ppm

NOTE: Detector tubes for nitrogen dioxide, measuring in excess of 0.5 ppm, are commercially available.

The TLV-TWA is considered to be sufficiently low to reduce the potential for immediate injury or adverse physiological effects from prolonged daily exposures. Although industrial data may contradict this conclusion, this data is not sufficiently precise to invalidate the TLV.

Short exposures of workmen to nitrogen dioxide concentrations averaging 25 to 38 ppm resulted in observable physiological response, but exposures of 3 to 5 minutes at 80 ppm produced tightness of the chest.

Odour Safety Factor (OSF)
OSF=7.7 (NITROGEN DIOXIDE).

PERSONAL PROTECTION

EYE

■ 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 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].

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 AS 1336...
Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

and AS 1338 which provide the maximum possible facial protection from flying particles and fragments. [WRIA-WTIA Technical Note 7].

HANDS/FEET
- Welding Gloves
- Safety footwear.

OTHER
- 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.

RESPIRATOR
- Type BE Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)

The local concentration of material, quantity and conditions of use determine the type of personal protective equipment required. For further information consult site specific CHEMWATCH data (if available), or your Occupational Health and Safety Advisor.

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.

For brazing or soldering 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 either open or limited spaces, use mechanical (general exhaust or plenum) ventilation. (Open work spaces exceed 300 cubic meters per welder)
For work conducted in 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 from the worker, of 0.5 metre/sec.
If risk of inhalation or overexposure exists, wear SAA approved respirator or work in fume hood.

Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

APPEARANCE
- Comweld Mild Steel - Uncoated rod with no markings.
- Comweld High Test - Copper coated rod with stamped end.
- Comweld Super Steel - Copper coated rod with stamped end.
- Comweld LW1 - Copper coated rod with stamped end.
- Comweld LW1-6 - Copper coated rod with stamped end.
- Comweld LW1-3 - Copper coated rod with stamped end.

continued...
Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

PHYSICAL PROPERTIES

Does not mix with water. Sinks in water.

<table>
<thead>
<tr>
<th>Property</th>
<th>Value</th>
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<tr>
<td>State</td>
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<tr>
<td>Melting Range (°C)</td>
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<tr>
<td>Boiling Range (°C)</td>
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<tr>
<td>Flash Point (°C)</td>
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<td>Decomposition Temp (°C)</td>
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<td>Vapour Pressure (kPa)</td>
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<td>Specific Gravity (water=1)</td>
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<td>Relative Vapour Density (air=1)</td>
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<td>Evaporation Rate</td>
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Section 10 - STABILITY AND REACTIVITY

CONDITIONS CONTRIBUTING TO INSTABILITY

- Presence of incompatible materials.
- Product is considered stable.
- Hazardous polymerisation will not occur.

For incompatible materials - refer to Section 7 - Handling and Storage.

Section 11 - TOXICOLOGICAL INFORMATION

POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

SWALLOWED

- Not normally a hazard due to physical form of product.

EYE

- Fumes from welding/brazing operations may be irritating to the eyes.

SKIN

- 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.

INHALED

- Fumes evolved during welding operations may be irritating to the upper-respiratory tract and may be harmful if 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.

Persons with impaired respiratory function, airway diseases and conditions such as emphysema or chronic bronchitis, may incur further disability if excessive concentrations of particulate are inhaled. If prior damage to the circulatory or nervous systems has occurred or if kidney damage has been sustained,
proper screenings should be conducted on individuals who may be exposed to further risk if handling and use of the material result in excessive exposures. 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.

CHRONIC HEALTH EFFECTS

- 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.

Severe disorders of the nervous system, has been reported in welders working on Mn steels in confined spaces. Ozone is suspected to produce lung cancer in laboratory animals; no reports of this effect have been documented in exposed human populations.

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 AND IRRITATION

- unless otherwise specified data extracted from RTECS - Register of Toxic Effects of Chemical Substances.

NITROGEN OXIDES:

OZONE:

- Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergic 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.

WELDING FUMES:

IRON OXIDE FUME:

COPPER FUME:

CIGWELD GAS AND TIG WELDING RODS:

- Not available. Refer to individual constituents.
■ WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.

MANGANESE FUME:
Skin (rabbit) 500mg/24H Mild
Eye (rabbit) 500mg/24H Mild

SILICA WELDING FUMES:

TOXICITY          IRRITATION
Oral (rat) LD50: 3160 mg/kg    No data [RTECS]

■ 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]

OZONE: 

continued...
■ NOTE: Ozone aggravates chronic obstructive pulmonary diseases. Ozone is suspected also of increasing the risk of acute and chronic respiratory disease, mutagenesis and foetotoxicity. In animals short-term exposure to ambient concentrations of less than 1 ppm results in reduced capacity to kill intrapulmonary organisms and allows purulent bacteria to proliferate [Ellenhorn et al].

NITROGEN OXIDES:

**TOXICITY**

Inhalation (human) LCLo: 200 ppm/1m

Inhalation (man) TCLo: 6200 ppb/10m

Data for nitrogen dioxide:

Substance has been investigated as a mutagen and reproductive effector.

**NOTE:** Interstitial edema, epithelial proliferation and, in high concentrations, fibrosis and emphysema develop after repeated exposure.

**CARCINOGEN**

Ferric oxide

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

**GROUP 3**

**REPROTOXIN**

manganese fume

ILO Chemicals in the electronics industry that have toxic effects on reproduction Reduced fertility or sterility

H si

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**Section 12 - ECOLOGICAL INFORMATION**

MANGANESE FUME:

SILICA WELDING FUMES:

COPPER FUME:

OZONE:

NITROGEN OXIDES:

IRON OXIDE FUME:

■ DO NOT discharge into sewer or waterways.

OZONE:

COPPER FUME:

■ Harmful to aquatic organisms.

MANGANESE FUME:

COPPER FUME:

IRON OXIDE FUME:

■ For Metal:

Atmospheric Fate - Metal-containing inorganic substances generally have negligible vapour pressure and are not expected to partition to air.

Environmental Fate: Environmental processes, such as oxidation, the presence of acids or bases and microbiological processes, may transform insoluble metals to more soluble ionic forms. Environmental processes may enhance bioavailability and may also be important in changing solubilities.

Aquatic/Terrestrial Fate: When released to dry soil, most metals will exhibit limited mobility and remain in the upper layer; some will leach locally into ground water and/or surface water ecosystems when soaked by rain or melt ice. A metal ion is considered infinitely persistent because it cannot degrade further. Once released to surface waters and moist soils their fate depends on solubility and dissociation in water. A significant proportion of dissolved/sorbed metals will end up in sediments through the settling of suspended particles. The remaining metal ions can then be taken up by aquatic organisms. Ionic species may bind to dissolved ligands or sorb to solid particles in water.

continued...
WELDING FUMES:

IRON OXIDE FUME:

MANGANESE FUME:

For manganese and its compounds:

Environmental fate:

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

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

The transport and partitioning of manganese in water is controlled by the solubility of the specific chemical form present, which in turn is determined by pH, Eh (oxidation-reduction potential), and the characteristics of the available anions. The metal may exist in water in any of four oxidation states. Manganese(II) predominates in most waters (pH 4-7) but may become oxidized at a pH >8 or 9. The principal anion associated with Mn(II) in water is usually carbonate (CO$_3$), and the concentration of manganese is limited by the relatively low solubility (65 mg/L) of MnCO$_3$. In relatively oxidized water, the solubility of Mn(II) may be controlled by manganese oxide equilibria, with manganese being converted to the Mn(III) or Mn(IV) oxidation states. In extremely reduced water, the fate of manganese tends to be controlled by formation of a poorly soluble sulfide. Manganese in water may undergo oxidation at high pH or Eh and is also subject to microbial activity. For example, Mn(II) in a lake was oxidized during the summer months, but this was inhibited by a microbial poison, indicating that the oxidation was mediated by bacteria. The microbial metabolism of manganese is presumed to be a function of pH, temperature, and other factors.

Manganese in water may be significantly bioconcentrated at lower trophic levels. A bioconcentration factor (BCF) relates the concentration of a chemical in plant and animal tissues to the concentration of the chemical in the water in which they live. The BCF of manganese was estimated as 2,500 - 6,300 for phytoplankton, 300 -5,500 for marine algae, 80 - 830 for intertidal mussels, and 35 - 930 for coastal fish. Similarly, the BCF of manganese was estimated to be 10,00 -20,000 for marine and freshwater plants, 10,000 -40,000 for invertebrates, and 10 - 600 for fish. In general, these data indicate that lower organisms such as algae have larger BCFS than higher organisms. In order to protect consumers from the risk of manganese bioaccumulation in marine mollusks, the U.S. EPA has set a criterion for manganese at 0.1 mg/L for marine waters.

Elemental manganese and inorganic manganese compounds have negligible vapor pressures but may exist in air as suspended particulate matter derived from industrial emissions or the erosion of soils. Manganese-containing particles are mainly removed from the atmosphere by gravitational settling, with large particles tending to fall out faster than small particles. The half-life of airborne particles is usually on the order of days, depending on the size of the particle and atmospheric conditions. Some removal by washout mechanisms such as rain may also occur, although it is of minor significance in comparison to dry deposition.
CIGWELD GAS AND TIG WELDING RODS
Chemwatch Independent Material Safety Data Sheet
Issue Date: 24-Oct-2011
A317LP

Section 12 - ECOLOGICAL INFORMATION

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

SILICA WELDING FUMES:
For Amorphous Silica: Amorphous silica is chemically and biologically inert. It is not biodegradable.
Aquatic Fate: Due to its insolubility in water there is a separation at every filtration and sedimentation process. On a global scale, the level of man-made synthetic amorphous silicas (SAS) represents up to 2.4% of the dissolved silica naturally present in the aquatic environment and untreated SAS have a relatively low water solubility and an extremely low vapour pressure. Biodegradability in sewage treatment plants or in surface water is not applicable to inorganic substances like SAS.
Terrestrial Fate: Crystalline and/or amorphous silicas are common on the earth in soils and sediments, and in living organisms (e.g. diatoms), but only the dissolved form is bioavailable. On the basis of these properties it is expected that SAS released into the environment will be distributed mainly into soil/sediment. Surface treated silica will be wetted then adsorbed onto soils and sediments.
Atmospheric Fate: SAS is not expected to be distributed into the air if released.
Ecotoxicity: SAS is not toxic to environmental organisms (apart from physical desiccation in insects). SAS presents a low risk for adverse effects to the environment.

For silica:
The literature on the fate of silica in the environment concerns dissolved silica in the aquatic environment, irrespective of its origin (man-made or natural), or structure (crystalline or amorphous). Indeed, once released and dissolved into the environment no distinction can be made between the initial forms of silica. At normal environmental pH, dissolved silica exists exclusively as monosilicic acid [Si(OH)4]. At pH 9.4 the solubility of amorphous silica is about 120 mg SiO2/l. Quartz has a solubility of only 6 mg/l, but its rate of dissolution is so slow at ordinary temperature and pressure that the solubility of amorphous silica represents the upper limit of dissolved silica concentration in natural waters. Moreover, silicic acid is the bioavailable form for aquatic organisms and it plays an important role in the biogeochemical cycle of Si, particularly in the oceans.
In the oceans, the transfer of dissolved silica from the marine hydrosphere to the biosphere initiates the global biological silicon cycle. Marine organisms such as diatoms, silicoflagellates and radiolarians build up their skeletons by taking up silicic acid from seawater. After these organisms die, the biogenic silica accumulated in them partly dissolves. The portion of the biogenic silica that does not dissolve settles and ultimately reaches the sediment. The transformation ofopal (amorphous biogenic silica) deposits in sediments through diagenetic processes allows silica to re-enter the geological cycle. Silica is labile between the water and sediment interface.
Ecotoxicity:
Fish LC50 (96 h): Brachydanio rerio >10000 mg/l; zebra fish >10000 mg/l
Daphnia magna EC50 (24 h): >1000 mg/l; LC50 96 h): >10000 mg/l.
No data

COPPER FUME:
For copper:
Atmospheric Fate - Copper is unlikely to accumulate in the atmosphere due to a short residence time for airborne copper aerosols. Airborne coppers, however, may be transported over large distances. Air Quality Standards: no data available.
Aquatic Fate: Toxicity of copper is affected by pH and hardness of water. Total copper is rarely useful as a predictor of toxicity. In natural sea water, more than 98% of copper is organically bound and in river waters a high percentage is often organically bound, but the actual percentage depends on the river water and its pH.
Ecotoxicity: Copper accumulates significantly in the food chain. The toxic effect of copper in the aquatic biota depends on the bio-availability of copper in water which, in turn, depends on its physico-chemical form (i.e. speciation). Bioavailability is decreased by complexation and adsorption of copper by natural organic matter, iron and manganese hydrated oxides, and chelating agents excreted by algae and other aquatic organisms. Copper exhibits significant toxicity in some aquatic organisms. Some algal species are very sensitive to copper. Silicate, iron, manganese and EDTA may reduce bioavailability.
For copper: Ecotoxicity - Significant effects are expected on various species of microalgae, some species of

continued...
macroalgae, and a range of invertebrates, including crustaceans, gastropods and sea urchins. Copper is moderately toxic to crab and their larvae and is highly toxic to gastropods (mollusks, including oysters, mussels and clams). In fish, the acute lethal concentrations of copper depends both on test species and exposure conditions. Waters with high concentrations of copper can have significant effects on diatoms and sensitive invertebrates, notably cladocerans (water fleas). Most taxonomic groups of macroalgae and invertebrates will be severely affected.

For Copper: Typical foliar levels of copper are: Uncontaminated soils (0.3-250 mg/kg); Contaminated soils (150-450 mg/kg); Mining/smelting soils (6.1-25 mg/kg; 80 mg/kg; 300 mg/kg).

Terrestrial Fate: Plants - Generally, vegetation reflects soil copper levels in its foliage. This is dependent upon the bioavailability of copper and the physiological requirements of species concerned. Crops are often more sensitive to copper than the native flora. Soil: In soil, copper levels are raised by application of fertilizer, fungicides, from deposition of highway dusts and from urban, mining and industrial sources. Chronic and or acute effects on sensitive species occur as a result of human activities such as copper fertilizer addition and addition of sludge. When soil levels exceed 150 mg Cu/kg, native and agricultural species show chronic effects. Soils in the range 500-1000 mg Cu/kg act in a strongly selective fashion allowing the survival of only copper-tolerant species and strains. At 2000 Cu mg/kg, most species cannot survive. By 3500 mg Cu/kg, areas are largely devoid of vegetation cover. The organic content of the soil appears to be a key factor affecting the bioavailability of copper. On normal forest soils, non-rooted plants such as mosses and lichens show higher copper concentrations. The fruiting bodies and mycorrhizal sheaths of soil fungi associated with higher plants in forests often accumulate copper to much higher levels than plants at the same site.

OZONE:
- Ozone is found in the atmosphere in varying proportions as it is produced continuously in the outer layers of the atmosphere by the action of solar UV radiation on oxygen in the air. It is also formed locally in the air from lightning and from electrical sparks. In the upper atmosphere it inhibits penetration of UV radiation and so is beneficial to life. At ground level it is a harmful pollutant because of the damage it can cause to lungs and to a wide range of materials. The material is classified as an ecotoxin* because the Fish LC50 (96 hours) is less than or equal to 0.1 mg/l
* Classification of Substances as Ecotoxic (Dangerous to the Environment) Appendix 8, Table 1


NITROGEN OXIDES:
- Environmental Fate: Oxides of nitrogen are found in soil, water and air. Nitrogen oxides are important in almost all atmospheric reactions. Nitrogen oxides react with water to form nitric acid, a major contributor to 'acid rain'. Oxides of nitrogen are also important in maintaining the level of ozone in the stratosphere. Low concentration of nitrogen dioxide as well as high amount of nitric oxide will reduce the formation of ozone. Further, oxides of nitrogen are also a major contributor to production of photochemical smog.

Environmental Transport: Nitrogen oxides are transported in different environment in the form of gas and as a dissolved gas in water.

Ecotoxicity

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<th>Ingredient</th>
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<th>Persistence: Air</th>
<th>Bioaccumulation</th>
<th>Mobility</th>
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continued...
Section 12 - ECOLOGICAL INFORMATION

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<th>Nitrogen Oxides</th>
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<td>Available</td>
</tr>
<tr>
<td></td>
<td>LOW</td>
<td></td>
</tr>
</tbody>
</table>

Section 13 - DISPOSAL CONSIDERATIONS

- 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 - TRANSPORTATION INFORMATION

HAZCHEM:
None (ADG7)

NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS: ADG7, UN, IATA, IMDG

Section 15 - REGULATORY INFORMATION

POISONS SCHEDULE None

REGULATIONS

Regulations for ingredients

iron oxide fume (CAS: 1309-37-1) is found on the following regulatory lists;
- Australia Exposure Standards
- Australia High Volume Industrial Chemical List (HVICL)
- Australia Inventory of Chemical Substances (AICS)
- International Agency for Research on Cancer (IARC)
- Agents Reviewed by the IARC Monographs
- International Council of Chemical Associations (ICCA)
- High Production Volume List

manganese fume (CAS: 7439-96-5) is found on the following regulatory lists;
- Australia - Australian Capital Territory - Environment Protection Regulation: Ambient environmental standards (Domestic water supply - inorganic chemicals)
- Australia - Australian Capital Territory - Environment Protection Regulation: Ambient environmental standards (IRRIG - inorganic chemicals)
- Australia - Australian Capital Territory - Environment Protection Regulation: Pollutants entering waterways taken to cause environmental harm (Domestic water supply quality)
- Australia - Australian Capital Territory - Environment Protection Regulation: Pollutants entering waterways taken to cause environmental harm (IRRIG)
- Australia Inventory of Chemical Substances (AICS)
- Australia National Pollutant Inventory
- WHO Guidelines for Drinking-water Quality - Guideline values for chemicals that are of health significance in drinking-water

silica welding fumes (CAS: 69012-64-2) is found on the following regulatory lists;
- Australia Inventory of Chemical Substances (AICS)

copper fume (CAS: 7440-50-8) is found on the following regulatory lists;
- Australia - Australian Capital Territory - Environment Protection Regulation: Ambient environmental standards (AQUA/1 to 6 - inorganic chemicals)
- Australia - Australian Capital Territory - Environment Protection Regulation: Ambient environmental standards (Domestic water supply - inorganic chemicals)
- Australia - Australian Capital Territory - Environment Protection Regulation: Ambient environmental standards (IRRIG - inorganic chemicals)
- Australia - Australian Capital Territory - Environment Protection Regulation: Pollutants entering waterways taken to cause environmental harm (Domestic water supply quality)
- Australia - Australian Capital Territory - Environment Protection Regulation: Pollutants entering waterways taken to cause environmental harm (IRRIG)
- Australia - Australian Capital Territory - Environment Protection Regulation: Pollutants entering waterways taken to cause environmental harm (STOCK)
- Australia ADI list - Acceptable daily intakes for agricultural and veterinary chemicals
- Australia Hazardous Substances
- Australia High Volume Industrial Chemical List (HVICL)
- Australia Inventory of Chemical Substances (AICS)
- International Maritime Dangerous Goods Requirements (IMDG Code) - Marine Pollutants
- International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index
- WHO Guidelines for Drinking-water Quality - Guideline values for chemicals that are of health significance in drinking-water

ozone (CAS: 10028-15-6) is found on the following regulatory lists;
- Australia Exposure Standards
- Australia Hazardous Substances
No data for CIGWELD Gas and TIG Welding Rods (CW: 15523)
No data for welding fumes (CAS: , Not avail)
No data for nitrogen oxides (CAS: , Mixture)

Section 16 - 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/references.

- 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.

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This is the end of the MSDS.