

## INDICATING OXYGEN TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 30-Jan-2012

A317L

CHEMWATCH 28-0929

Version No:3

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### Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

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#### PRODUCT NAME

INDICATING OXYGEN TRAP

#### PROPER SHIPPING NAME

SELF-HEATING SOLID, TOXIC, INORGANIC, N.O.S.(contains manganese dioxide and copper(II) oxide)

#### PRODUCT NUMBERS

SGE Part number 103491

#### PRODUCT USE

- Used according to manufacturer's directions.  
Laboratory chemicals.

#### SUPPLIER

Company: SGE Analytical Science Pty Ltd

Address:

7 Argent Place

Ringwood

VIC, 3134

Australia

Telephone: +61 3 9837 4200

Telephone: (800) 945 6154 (US)

Emergency Tel:**1800 800 167 (AUS)**

Emergency Tel:**+800 2790 8999 (within EU)**

Fax: +61 3 9874 5672

Email: techsupport@sge.com

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### Section 2 - HAZARDS IDENTIFICATION

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#### STATEMENT OF HAZARDOUS NATURE

**HAZARDOUS SUBSTANCE. DANGEROUS GOODS. According to the Criteria of NOHSC, and the ADG Code.**

#### RISK

- Contact with combustible material may cause fire.
- Highly flammable.
- Spontaneously flammable in air.
- Harmful by inhalation and if swallowed.
- Irritating to respiratory system and skin.
- Risk of serious damage to eyes.
- Harmful: danger of serious damage to health by prolonged exposure through inhalation.

#### SAFETY

- Keep out of reach of children.
- Keep away from combustible material.
- Do not breathe dust.
- Avoid contact with skin.
- Avoid contact with eyes.
- Wear suitable protective clothing.
- Wear suitable gloves.

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Section 2 - HAZARDS IDENTIFICATION

■ Very toxic to aquatic organisms, may cause long- term adverse effects in the aquatic environment.

- Wear eye/face protection.
- Use only in well ventilated areas.
- Keep container in a well ventilated place.
- Do not empty into drains.
- To clean the floor and all objects contaminated by this material, use water and detergent.
- This material and its container must be disposed of in a safe way.
- Keep away from food, drink and animal feeding stuffs.
- In case of contact with eyes, rinse with plenty of water and contact Doctor or Poisons Information Centre.
- If swallowed, IMMEDIATELY contact Doctor or Poisons Information Centre. (show this container or label).
- Use appropriate container to avoid environmental contamination.
- Avoid release to the environment. Refer to special instructions/Safety data sheets.
- This material and its container must be disposed of as hazardous waste.

## Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

NAME	CAS RN	%
manganese dioxide	1313-13-9	35-55
silica amorphous	7631-86-9	<45
talc	14807-96-6	0-35
copper(II) oxide	1317-38-0	6-25
aluminium oxide	1344-28-1.	0-10
calcium oxide	1305-78-8	0-5
sodium monoxide	1313-59-3	0-5
barium oxide	1304-28-5	<1
silica crystalline - quartz	14808-60-7	<1
zinc oxide	1314-13-2	<1

## Section 4 - FIRST AID MEASURES

### SWALLOWED

- For advice, contact a Poisons Information Centre or a doctor at once.
- Urgent hospital treatment is likely to be needed.
- If swallowed do NOT induce vomiting.
- If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.
- Observe the patient carefully.
- Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.
- Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.
- Transport to hospital or doctor without delay.

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Section 4 - FIRST AID MEASURES

## EYE

■ If this product comes in contact with the eyes:

- Immediately hold eyelids apart and flush the eye continuously with running water.
- Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.
- Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.
- Transport to hospital or doctor without delay.
- Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

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

■ If skin or hair contact occurs:

- Immediately flush body and clothes with large amounts of water, using safety shower if available.
- Quickly remove all contaminated clothing, including footwear.
- Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre.
- Transport to hospital, or doctor.

In case of burns:

- Immediately apply cold water to burn either by immersion or wrapping with saturated clean cloth.
- DO NOT remove or cut away clothing over burnt areas. DO NOT pull away clothing which has adhered to the skin as this can cause further injury.
- DO NOT break blister or remove solidified material.
- Quickly cover wound with dressing or clean cloth to help prevent infection and to ease pain.
- For large burns, sheets, towels or pillow slips are ideal; leave holes for eyes, nose and mouth.
- DO NOT apply ointments, oils, butter, etc. to a burn under any circumstances.
- Water may be given in small quantities if the person is conscious.
- Alcohol is not to be given under any circumstances.
- Reassure.
- Treat for shock by keeping the person warm and in a lying position.
- Seek medical aid and advise medical personnel in advance of the cause and extent of the injury and the estimated time of arrival of the patient.

## 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

■ Treat symptomatically.

for copper intoxication:

- Unless extensive vomiting has occurred empty the stomach by lavage with water, milk, sodium bicarbonate solution or a 0.1% solution of potassium ferrocyanide (the resulting copper ferrocyanide is insoluble).
- Administer egg white and other demulcents.
- Maintain electrolyte and fluid balances.
- Morphine or meperidine (Demerol) may be necessary for control of pain.
- If symptoms persist or intensify (especially circulatory collapse or cerebral disturbances, try BAL intramuscularly or penicillamine in accordance with the supplier's recommendations.
- Treat shock vigorously with blood transfusions and perhaps vasopressor amines.
- If intravascular haemolysis becomes evident protect the kidneys by maintaining a diuresis with mannitol and perhaps by alkalinising the urine with sodium bicarbonate.

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Section 4 - FIRST AID MEASURES

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- It is unlikely that methylene blue would be effective against the occasional methaemoglobinemia and it might exacerbate the subsequent haemolytic episode.
  - Institute measures for impending renal and hepatic failure. [GOSSELIN, SMITH & HODGE: Commercial Toxicology of Commercial Products]
  - A role for activated charcoal or emesis is, as yet, unproven.
  - In severe poisoning CaNa<sub>2</sub>EDTA has been proposed. [ELLENHORN & 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.].

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## Section 5 - FIRE FIGHTING MEASURES

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### EXTINGUISHING MEDIA

#### ■ For SMALL FIRES:

- Dry chemical, CO<sub>2</sub>, water spray or foam.

#### For LARGE FIRES:

- Foam, fog or water spray
- DO NOT use water jets.

### FIRE FIGHTING

- When silica dust is dispersed in air, firefighters should wear inhalation protection as hazardous substances from the fire may be adsorbed on the silica particles.
- When heated to extreme temperatures, (>1700 deg.C) amorphous silica can fuse.
- Alert Fire Brigade and tell them location and nature of hazard.
- Wear full body protective clothing with breathing apparatus.
- Prevent, by any means available, spillage from entering drains or water course.
- Fight fire from a safe distance, with adequate cover.
- If safe, switch off electrical equipment until vapour fire hazard removed.
- Use water delivered as a fine spray to control fire and cool adjacent area.
- Avoid spraying water onto liquid pools.
- 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.

When any large container (including road and rail tankers) is involved in a fire, consider evacuation by 800 metres in all directions.

### FIRE/EXPLOSION HAZARD

■ Under certain conditions the material may become combustible because of the ease of ignition which occurs after the material reaches a high specific area ratio (thin sections, fine particles, or molten states). However, the same material in massive solid form is comparatively difficult to ignite. Nearly all metals will burn in air under certain conditions. Some are oxidised rapidly in the presence of air or moisture, generating sufficient heat to reach their ignition temperatures. Others oxidise so slowly that heat generated during oxidation is dissipated before the metal becomes hot enough to ignite.

Particle size, shape, quantity, and alloy are important factors to be considered when evaluating metal combustibility. Combustibility of metallic alloys may differ and vary widely from the combustibility characteristics of the alloys' constituent elements.

- May ignite on contact with air leading to spontaneous combustion
- May decompose explosively when heated or involved in fire.
- May REIGNITE after fire is extinguished.
- Gases generated in fire may be poisonous, corrosive or irritating.
- Containers may explode on heating.
- Runoff may create multiple fire or explosion hazard.

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Combustion products include: silicon dioxide (SiO<sub>2</sub>), metal oxides.

When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles.

## FIRE INCOMPATIBILITY

- Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result.

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## Section 6 - ACCIDENTAL RELEASE MEASURES

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### MINOR SPILLS

- Eliminate all ignition sources.
- Cover with WET earth, sand or other non-combustible material.
- Use clean, non-sparking tools to collect absorbed material
- Wear gloves and safety glasses as appropriate.

### MAJOR SPILLS

- Clear area of personnel and move upwind.
- Alert Fire Brigade and tell them location and nature of hazard.
- Eliminate all ignition sources (no smoking, flares, sparks or flames)
- Stop leak if safe to do so; prevent entry into waterways, drains or confined spaces.
- May be violently or explosively reactive.
- DO NOT walk through spilled material.
- Wear full protective clothing plus breathing apparatus.
- DO NOT touch damaged containers or spilled material unless wearing appropriate protective clothing.
- Cover with WET earth, sand or other non-combustible material.
- Water spray may be used to knock down vapours or divert vapour clouds.
- Use clean, non-sparking tools to collect absorbed material and place into loosely-covered metal or plastic containers ready for disposal.
- Alternately, the spill may be contained using WET earth, sand or other non-combustible material.
- Recover the liquid using non-sparking appliances and place in labelled, sealable container.
- Wash area with water and dike for later disposal;
- After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.
- If contamination of drains or waterways occurs, advise emergency services.

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

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## Section 7 - HANDLING AND STORAGE

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### PROCEDURE FOR HANDLING

- For large scale or continuous use, spark-free, earthed ventilation system venting directly to the outside and separate from usual ventilation systems
- Provide dust collectors with explosion vents.
- Avoid all personal contact, including inhalation.
- Wear protective clothing when risk of overexposure occurs.
- Use in a well-ventilated area.
- Avoid smoking, naked lights or ignition sources.
- Avoid contact with incompatible materials.
- When handling, DO NOT eat, drink or smoke.
- Keep containers securely sealed when not in use.

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- Avoid physical damage to containers.
- Always wash hands with soap and water after handling.
- Work clothes should be laundered separately and before re-use
- Use good occupational work practice.
- Observe manufacturer's storage and handling recommendations contained within this MSDS.
- Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.

NOTE: The material may remove oxygen from the air thus producing a severe hazard to workers inside enclosed or confined spaces where the material might accumulate. Before entry to such areas, sampling and test procedures for low oxygen levels should be undertaken; control conditions should be established to ensure the availability of adequate oxygen supply.

## SUITABLE CONTAINER

- For low viscosity materials and solids:

Drums and jerricans must be of the non-removable head type.

Where a can is to be used as an inner package, the can must have a screwed enclosure.

For materials with a viscosity of at least 2680 cSt. (23 deg. C):

- Removable head packaging and
- cans with friction closures may be used.

-

Where combination packages are used, there must be sufficient inert absorbent material to absorb completely any leakage that may occur, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.

All combination packages for Packing group I and II must contain cushioning material.

## STORAGE INCOMPATIBILITY

- Derivative of electropositive metal.

For aluminas (aluminium oxide):

- Incompatible with hot chlorinated rubber.
- In the presence of chlorine trifluoride may react violently and ignite.
- May initiate explosive polymerisation of olefin oxides including ethylene oxide.
- Produces exothermic reaction above 200 C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals.
- Produces exothermic reaction with oxygen difluoride.
- May form explosive mixture with oxygen difluoride.
- Forms explosive mixtures with sodium nitrate.
- Reacts vigorously with vinyl acetate.

Silicas:

- react with hydrofluoric acid to produce silicon tetrafluoride gas
- react with xenon hexafluoride to produce explosive xenon trioxide
- reacts exothermically with oxygen difluoride, and explosively with chlorine trifluoride (these halogenated materials are not commonplace industrial materials) and other fluorine-containing compounds
- may react with fluorine, chlorates
- are incompatible with strong oxidisers, manganese trioxide, chlorine trioxide, strong alkalis, metal oxides, concentrated orthophosphoric acid, vinyl acetate
- may react vigorously when heated with alkali carbonates.
- Inorganic oxidising agents can react with reducing agents to generate heat and products that may be gaseous (causing pressurization of closed containers). The products may themselves be capable of further reactions (such as combustion in the air).
- Organic compounds in general have some reducing power and can in principle react with compounds in this class. Actual reactivity varies greatly with the identity of the organic compound.
- Inorganic oxidising agents can react violently with active metals, cyanides, esters, and thiocyanates.
- Inorganic reducing agents react with oxidizing agents to generate heat and products that may be flammable, combustible, or otherwise reactive. Their reactions with oxidizing agents may be violent.
- Incidents involving interaction of active oxidants and reducing agents, either by design or accident, are usually very energetic and examples of so-called redox reactions.
- WARNING: Avoid or control reaction with peroxides. All transition metal peroxides should be considered as potentially explosive. For example transition metal complexes of alkyl hydroperoxides may decompose

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explosively.

- The pi-complexes formed between chromium(0), vanadium(0) and other transition metals (haloarene-metal complexes) and mono-or poly-fluorobenzene show extreme sensitivity to heat and are explosive.
- Avoid reaction with borohydrides or cyanoborohydrides.
- Metals and their oxides or salts may react violently with chlorine trifluoride and bromine trifluoride.
- These trifluorides are hypergolic oxidisers. They ignites on contact (without external source of heat or ignition) with recognised fuels - contact with these materials, following an ambient or slightly elevated temperature, is often violent and may produce ignition.
- The state of subdivision may affect the results.
- Avoid any contamination of this material as it is very reactive and any contamination is potentially hazardous.
- Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.
- Avoid storage with reducing agents.

## STORAGE REQUIREMENTS

- Store under an inert gas, e.g. argon or nitrogen.

### FOR MINOR QUANTITIES:

- Store in an indoor fireproof cabinet or in a room of noncombustible construction.
- Provide adequate portable fire-extinguishers in or near the storage area.

### FOR PACKAGE STORAGE:

- Store in original containers in approved flame-proof area.
- No smoking, naked lights, heat or ignition sources.
- DO NOT store in pits, depressions, basements or areas where vapours may be trapped.
- Keep containers securely sealed.
- Store away from incompatible materials in a cool, dry, well ventilated area.
- Protect containers against physical damage and check regularly for leaks.
- Protect containers from exposure to weather and from direct sunlight unless: (a) the packages are of metal or plastic construction; (b) the packages are securely closed are not opened for any purpose while in the area where they are stored and (c) adequate precautions are taken to ensure that rain water, which might become contaminated by the dangerous goods, is collected and disposed of safely.
- Ensure proper stock-control measures are maintained to prevent prolonged storage of dangerous goods.
- Observe manufacturer's storage and handling recommendations contained within this MSDS.

## Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

### EXPOSURE CONTROLS

Source	Material	TWA mg/m <sup>3</sup>	STEL mg/m <sup>3</sup>	Notes
Australia Exposure Standards	Indicating Oxygen Trap (Emery (dust) (a))	10		(see Chapter 14)
Australia Exposure Standards	Indicating Oxygen Trap (Aluminium (welding fumes) (as Al))	5		
Australia Exposure Standards	Indicating Oxygen Trap (Aluminium (metal dust))	10		
Australia Exposure Standards	Indicating Oxygen Trap (Silica - Amorphous Fumed silica (respirable dust))	2		(see Chapter 14)
Australia Exposure Standards	talca (Talc, (containing no asbestos fibres))	2.5		
Australia Exposure Standards	talca (Soapstone (respirable dust))	3		(see also Soapstone (a))
Australia Exposure Standards	copper(II) oxide (Copper, dusts & mists (as Cu))	1		

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Source	Material	TWA mg/m <sup>3</sup>	STEL mg/m <sup>3</sup>	Notes
Australia Exposure Standards	copper(II) oxide (Copper (fume))	0.2		
Australia Exposure Standards	calcium oxide (Calcium oxide)	2		
Australia Exposure Standards	barium oxide (Barium, soluble compounds (as Ba))	0.5		
Australia Exposure Standards	silica crystalline - quartz (Silica - Crystalline Quartz)	0.1		(see Chapter 14)
Australia Exposure Standards	zinc oxide (Zinc oxide (fume))	5	10	

The following materials had no OELs on our records

- sodium monoxide:

CAS:1313- 59- 3 CAS:12401- 86- 4

### EMERGENCY EXPOSURE LIMITS

Material	Revised	IDLH
manganese dioxide 10492	500	
silica amorphous 10451	3, 000	
talc 10454	3, 000	
talc 10454	1, 000	
calcium oxide 1910	25	
barium oxide 1884	50	
silica crystalline - quartz 21686	50	
zinc oxide 22544	500	

### MATERIAL DATA

INDICATING OXYGEN TRAP:

SILICA AMORPHOUS:

SILICA CRYSTALLINE - QUARTZ:

ZINC OXIDE:

■ 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.

INDICATING OXYGEN TRAP:

MANGANESE DIOXIDE:

SILICA CRYSTALLINE - QUARTZ:

■ Because the margin of safety of the quartz TLV is not known with certainty and given the associated link between silicosis and lung cancer it is recommended that quartz concentrations be maintained as far below the TLV as prudent practices will allow.

ALUMINIUM OXIDE:

INDICATING OXYGEN TRAP:

- For aluminium oxide:

The experimental and clinical data indicate that aluminium oxide acts as an "inert" material when inhaled and seems to have little effect on the lungs nor does it produce significant organic disease or toxic effects when exposures are kept under reasonable control.

[Documentation of the Threshold Limit Values], ACGIH, Sixth Edition.

COPPER(II) OXIDE:

INDICATING OXYGEN TRAP:

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INDICATING OXYGEN TRAP:

SILICA AMORPHOUS:

- 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.

INDICATING OXYGEN TRAP:

MANGANESE DIOXIDE:

■ Ceiling values were recommended for manganese and compounds in earlier publications. As manganese is a chronic toxin a TWA is considered more appropriate. Because workers exposed to fume exhibited manganism at air-borne concentrations below those that affect workers exposed to dust a lower value has been proposed to provide an extra margin of safety. This value is still above that experienced by two workers exposed to manganese fume in the course of one study.

CALCIUM OXIDE:

INDICATING OXYGEN TRAP:

- For calcium oxide:

The TLV-TWA is thought to be protective against undue irritation and is analogous to that recommended for sodium hydroxide.

INDICATING OXYGEN TRAP:

ZINC OXIDE:

- for zinc oxide:

Zinc oxide intoxication (intoxication zincale) is characterised by general depression, shivering, headache, thirst, colic and diarrhoea.

Exposure to the fume may produce metal fume fever characterised by chills, muscular pain, nausea and vomiting. Short-term studies with guinea pigs show pulmonary function changes and morphologic evidence of small airway inflammation. A no-observed-adverse-effect level (NOAEL) in guinea pigs was 2.7 mg/m<sup>3</sup> zinc oxide. Based on present data, the current TLV-TWA may be inadequate to protect exposed workers although known physiological differences in the guinea pig make it more susceptible to functional impairment of the airways than humans.

INDICATING OXYGEN TRAP:

TALC:

- For talc (a form of magnesium silicate):

Most health problems associated with occupational exposure to talcs appear to evolve mostly from the nonplatifrom content of the talc being mined or milled (being the asbestos-like amphiboles, serpentines (asbestiformes) and other minerals in the form of acicular, prismatic and fibrous crystals including, possibly, asbestos).

Because of severe health effects associated with exposures to asbestos, regulatory agencies tend to regard all elongate mineral crystal particles, whether prismatic, acicular, fibrous, as asbestos - the only provision is the particles have an aspect ratio (length to diameter) of 3:1 or greater.

Consideration is also given to their respirability, their width being less than or equal to 3 µm. Only limited data, however, exists on the health effects of elongate mineral particles having prismatic, acicular or fibrous (non-asbestos) forms. Experimental evidence indicates that the carcinogen potential of mineral fibres is related to the size class with diameter of 8 µm with shorter, thicker particles having little biological activity.

Dust of nonfibrous talc, consisting entirely of platifrom talc crystals and containing no asbestos poses a relatively small respiratory hazard.

Difficulties exist, however, in the determination of asbestos as cleavage fragments of prismatic or acicular crystals, nonasbestos fibres and asbestos fibres are very similar.

Subject to an accurate determination of asbestos and crystalline silica, exposure at or below the recommended TLV-TWA, is thought to protect workers from the significant risk of nonmalignant respiratory effects associated with talc dusts.

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INDICATING OXYGEN TRAP:

MANGANESE DIOXIDE:

■ The International Agency for Research on Cancer (IARC) has classified occupational exposures to respirable (<5 µm) crystalline silica as being carcinogenic to humans. This classification is based on what IARC considered sufficient evidence from epidemiological studies of humans for the carcinogenicity of inhaled silica in the forms of quartz and cristobalite. Crystalline silica is also known to cause silicosis, a non-cancerous lung disease.

Intermittent exposure produces; focal fibrosis, (pneumoconiosis), cough, dyspnoea, liver tumours.

\* Millions of particles per cubic foot (based on impinger samples counted by light field techniques).

NOTE : the physical nature of quartz in the product determines whether it is likely to present a chronic health problem. To be a hazard the material must enter the breathing zone as respirable particles.

INDICATING OXYGEN TRAP:

MANGANESE DIOXIDE:

■ **WARNING:** For inhalation exposure ONLY: This substance has been classified by the IARC as Group 1: **CARCINOGENIC TO HUMANS.**

INDICATING OXYGEN TRAP:

■ For aluminium oxide and pyrophoric grades of aluminium:

Twenty seven year experience with aluminium oxide dust (particle size 96% 1,2 µm) without adverse effects either systemically or on the lung, and at a calculated concentration equivalent to 2 mg/m<sup>3</sup> over an 8-hour shift has lead to the current recommendation of the TLV-TWA.

The limit should also apply to aluminium pyro powders whose toxicity is reportedly greater than aluminium dusts and should be protective against lung changes.

MANGANESE DIOXIDE:

■ A number of studies have shown that susceptibility to the effects of manganese at or about 1 - 5 mg/m<sup>3</sup> (TWA) can lead to clinical manifestations of manganism or more commonly to the development of indicators of sub-clinical manganism (e.g. hand tremor, exaggerated reflexes, short-term memory deficits, poor psychomotor performance). Controlling long-term exposure to the recommended ES TWA level or below should provide protection for those individuals susceptible to neurological effects of prolonged exposure.

ALUMINIUM OXIDE:

■ The TLV is based on the exposures to aluminium chloride and the amount of hydrolysed acid and the corresponding acid TLV to provide the same degree of freedom from irritation. Workers chronically exposed to aluminium dusts and fumes have developed severe pulmonary reactions including fibrosis, emphysema and pneumothorax. A much rarer encephalopathy has also been described.

SODIUM MONOXIDE:

■ for sodium hydroxide:

The TLV-C is recommended based on concentrations that produce noticeable but not excessive, ocular and upper respiratory tract irritation.

No exposure limits set by NOHSC or ACGIH.

Exposure limits with "skin" notation indicate that vapour and liquid may be absorbed through intact skin.

Absorption by skin may readily exceed vapour inhalation exposure. Symptoms for skin absorption are the same as for inhalation. Contact with eyes and mucous membranes may also contribute to overall exposure and may also invalidate the exposure standard.

OEL STEL (Russia): 2 mg/m<sup>3</sup> SKIN

BARIUM OXIDE:

■ for barium compounds:

The recommended TLV-TWA is based on satisfactory results achieved while employing an internal limit for barium nitrate at a national laboratory. It is not known what degree of added safety this limit incorporates.

SILICA CRYSTALLINE - QUARTZ:

■ **WARNING:** For inhalation exposure ONLY:

This substance has been classified by the ACGIH as A2 Suspected Human Carcinogen.

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# INDICATING OXYGEN TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

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Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

## ZINC OXIDE:

■ 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.

Sensory irritants are chemicals that produce temporary and undesirable side-effects on the eyes, nose or throat. Historically occupational exposure standards for these irritants have been based on observation of workers' responses to various airborne concentrations. Present day expectations require that nearly every individual should be protected against even minor sensory irritation and exposure standards are established using uncertainty factors or safety factors of 5 to 10 or more. On occasion animal no-observable-effect-levels (NOEL) are used to determine these limits where human results are unavailable. An additional approach, typically used by the TLV committee (USA) in determining respiratory standards for this group of chemicals, has been to assign ceiling values (TLV C) to rapidly acting irritants and to assign short-term exposure limits (TLV STELs) when the weight of evidence from irritation, bioaccumulation and other endpoints combine to warrant such a limit. In contrast the MAK Commission (Germany) uses a five-category system based on intensive odour, local irritation, and elimination half-life. However this system is being replaced to be consistent with the European Union (EU) Scientific Committee for Occupational Exposure Limits (SCOEL); this is more closely allied to that of the USA.

OSHA (USA) concluded that exposure to sensory irritants can:

- cause inflammation
- cause increased susceptibility to other irritants and infectious agents
- lead to permanent injury or dysfunction
- permit greater absorption of hazardous substances and
- acclimate the worker to the irritant warning properties of these substances thus increasing the risk of overexposure.

## PERSONAL PROTECTION

### EYE

- Chemical goggles.
- Full face shield may be required for supplementary but never for primary protection of eyes
- 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].

### HANDS/FEET

#### ■ NOTE:

- The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
  - frequency and duration of contact,
  - chemical resistance of glove material,
  - glove thickness and
  - dexterity

Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national

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equivalent).

- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- Contaminated gloves should be replaced.

Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly.

Application of a non-perfumed moisturiser is recommended.

- Fire resistant/ heat resistant gloves where practical, otherwise
- Heavy-duty chemically resistant gloves capable of providing short-term protection against spontaneous ignition.

### OTHER

- Wear protective clothing appropriate for the work situation.

For large scale or continuous use, when handling dry powder, wear :

- non-sparking safety footwear,
- tight-weave, non-static, noncombustible or flameproof clothing without cuffs, metallic fasteners, pockets, or laps in which powder may collect.

### RESPIRATOR

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

- If inhalation risk above the TLV exists, wear approved dust respirator.

Use respirators with protection factors appropriate for the exposure level.

- Up to 5 X TLV, use valveless mask type; up to 10 X TLV, use 1/2 mask dust respirator
- Up to 50 X TLV, use full face dust respirator or demand type C air supplied respirator
- Up to 500 X TLV, use powered air-purifying dust respirator or a Type C pressure demand supplied-air respirator
- Over 500 X TLV wear full-face self-contained breathing apparatus with positive pressure mode or a combination respirator with a Type C positive pressure supplied-air full-face respirator and an auxiliary self-contained breathing apparatus operated in pressure demand or other positive pressure mode.
- Respirators may be necessary when engineering and administrative controls do not adequately prevent exposures.
- The decision to use respiratory protection should be based on professional judgment that takes into account toxicity information, exposure measurement data, and frequency and likelihood of the worker's exposure - ensure users are not subject to high thermal loads which may result in heat stress or distress due to personal protective equipment (powered, positive flow, full face apparatus may be an option).
- Published occupational exposure limits, where they exist, will assist in determining the adequacy of the selected respiratory . These may be government mandated or vendor recommended.
- Certified respirators will be useful for protecting workers from inhalation of particulates when properly selected and fit tested as part of a complete respiratory protection program.
- Use approved positive flow mask if significant quantities of dust becomes airborne.
- Try to avoid creating dust conditions.

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.

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Employers may need to use multiple types of controls to prevent employee overexposure.

- Local exhaust ventilation is required where solids are handled as powders or crystals; even when particulates are relatively large, a certain proportion will be powdered by mutual friction.
- Exhaust ventilation should be designed to prevent accumulation and recirculation of particulates in the workplace.
- If in spite of local exhaust an adverse concentration of the substance in air could occur, respiratory protection should be considered. Such protection might consist of:
  - (a): particle dust respirators, if necessary, combined with an absorption cartridge;
  - (b): filter respirators with absorption cartridge or canister of the right type;
  - (c): fresh-air hoods or masks
- Build-up of electrostatic charge on the dust particle, may be prevented by bonding and grounding.
- Powder handling equipment such as dust collectors, dryers and mills may require additional protection measures such as explosion venting.

## Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

### APPEARANCE

Coloured solid with no odour; does not mix with water.

### PHYSICAL PROPERTIES

Does not mix with water.

Sinks in water.

State	Divided Solid	Molecular Weight	Not Applicable
Melting Range (°C)	Not Available	Viscosity	Not Applicable
Boiling Range (°C)	Not Available	Solubility in water (g/L)	Immiscible
Flash Point (°C)	Not Applicable	pH (1% solution)	Not Applicable
Decomposition Temp (°C)	Not Available	pH (as supplied)	Not Applicable
Autoignition Temp (°C)	Not Applicable	Vapour Pressure (kPa)	Not Applicable
Upper Explosive Limit (%)	Not Applicable	Specific Gravity (water=1)	1.2
Lower Explosive Limit (%)	Not Applicable	Relative Vapour Density (air=1)	Not Applicable
Volatile Component (%vol)	Not Applicable	Evaporation Rate	Not Applicable

## Section 10 - STABILITY AND REACTIVITY

### CONDITIONS CONTRIBUTING TO INSTABILITY

- May heat spontaneously
- Identify and remove sources of ignition and heating.
- Incompatible material, especially oxidisers, and/or other sources of oxygen may produce unstable product(s).
- Hazardous polymerization will not occur.

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

## Section 11 - TOXICOLOGICAL INFORMATION

### POTENTIAL HEALTH EFFECTS

### ACUTE HEALTH EFFECTS

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## SWALLOWED

■ Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.

Acute toxic responses to aluminium are confined to the more soluble forms.

Not normally a hazard due to the physical form of product. The material is a physical irritant to the gastrointestinal tract.

Numerous cases of a single oral exposure to high levels of copper have been reported. Consumption of copper-contaminated drinking water has been associated with mainly gastrointestinal symptoms including nausea, abdominal pain, vomiting and diarrhoea. A metallic taste, nausea, vomiting and epigastric burning often occur after ingestion of copper and its derivatives. The vomitus is usually green/blue and discolours contaminated skin. Acute poisonings from the ingestion of copper salts are rare due to their prompt removal by vomiting. Vomiting is due mainly to the local and astringent action of copper ion on the stomach and bowel. Emesis usually occurs within 5 to 10 minutes but may be delayed if food is present in the stomach. Should vomiting not occur, or is delayed, gradual absorption from the bowel may result in systemic poisoning with death, possibly, following within several days. Apparent recovery may be followed by lethal relapse. Systemic effects of copper resemble other heavy metal poisonings and produce wide-spread capillary damage, kidney and liver damage and central nervous system excitation followed by depression. Haemolytic anaemia (a result of red-blood cell damage) has been described in acute human poisoning. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products.]

Other symptoms of copper poisoning include lethargy, neurotoxicity, and increased blood pressure and respiratory rates. Coma and death have followed attempted suicides using solutions of copper sulfate. Copper is an essential element and most animal tissues have measurable amounts of copper associated with them. Humans have evolved mechanisms which maintain its availability whilst limiting its toxicity (homeostasis). Copper is initially bound in the body to a blood-borne protein, serum albumin and thereafter is more firmly bound to another protein, alpha-ceruloplasmin. Such binding effectively "inactivates" the copper, thus reducing its potential to produce toxic damage. In healthy individuals, bound copper can reach relatively high levels without producing adverse health effects. Excretion in the bile represents the major pathway by which copper is removed from the body when it reaches potentially toxic levels. Copper may also be stored in the liver and bone marrow where it is bound to another protein, metallothionein. A combination of binding and excretion ensures that the body is able to tolerate relatively high loadings of copper. Poisonings rarely occur after oral administration of manganese salts as they are generally poorly absorbed from the gut (generally less than 4%) and seems to be dependent, in part, on levels of dietary iron and may increase following the consumption of alcohol. A side-effect of oral manganese administration is an increase in losses of calcium in the faeces and a subsequent lowering of calcium blood levels. Absorbed manganese tends to be slowly excreted in the bile. Divalent manganese appears to be 2.5-3 times more toxic than the trivalent form.

## EYE

■ When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.

Copper salts, in contact with the eye, may produce conjunctivitis or even ulceration and turbidity of the cornea.

## SKIN

■ Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus. Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles.

Open cuts, abraded or irritated skin should not be exposed to this material.

Exposure to copper, by skin, has come from its use in pigments, ointments, ornaments, jewellery, dental amalgams and IUDs and as an antifungal agent and an algicide. Although copper algicides are used in the treatment of water in swimming pools and reservoirs, there are no reports of toxicity from these applications. Reports of allergic contact dermatitis following contact with copper and its salts have appeared in the literature, however the exposure concentrations leading to any effect have been poorly characterised. In one study, patch testing of 1190 eczema patients found that only 13 (1.1%) cross-reacted with 2% copper sulfate in petrolatum. The investigators warned, however, that the possibility of contamination with nickel (an established contact allergen) might have been the cause of the reaction. Copper salts often produce an itching eczema in contact with skin. This is, likely, of a non-allergic nature. Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce

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systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.

Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.

## INHALED

■ Inhalation of dusts, generated by the material, during the course of normal handling, may be harmful.

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.

Effects on lungs are significantly enhanced in the presence of respirable particles. Overexposure to respirable dust may produce wheezing, coughing and breathing difficulties leading to or symptomatic of impaired respiratory function.

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.

Copper poisoning following exposure to copper dusts and fume may result in headache, cold sweat and weak pulse. Capillary, kidney, liver and brain damage are the longer term manifestations of such poisoning.

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Acute silicosis occurs under conditions of extremely high silica dust exposure particularly when the particle size of the dust is small. It differs greatly from classical silicosis both clinically and pathologically.

The disease is rapidly progressive with diffuse pulmonary involvement developing only months after the initial exposure and causing deaths within 1 to 2 years. It is often complicated by an associated tuberculosis. The lungs of victims contain no classical silicotic nodules or only a few, microscopic abortive nodules, whereas the air spaces are diffusively filled and distended with silica-containing, lipoprotein paste in which degenerating and necrotic macrophages are sometimes discernible - the condition is sometimes described as alveolar lipoproteinosis. The uptake of silica particles by macrophages and lysosomal incorporation, is followed by rupture of the lysosomal membrane and release of lysosomal enzymes into cytoplasm of the macrophage. This causes the macrophage to be digested by its own enzymes and after lysis the free silica is released to be ingested by other macrophages thus continuing initiate collagen formation in the lung tissue producing the characteristic nodule found in classical (chronic) silicosis.

## CHRONIC HEALTH EFFECTS

■ Harmful: danger of serious damage to health by prolonged exposure through inhalation.

Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or

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contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.

On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals.

Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers.

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 alveoli (sub 5 um) are able to produce pathogenic effects in the lungs.

Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic.

Because aluminium competes with calcium for absorption, increased amounts of dietary aluminium may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies, aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseous tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory. These salts' estrogen-like effects have led to their classification as a metalloestrogen. Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.

After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin.

Aluminium can enter the brain and reach the placenta and foetus. Aluminium may persist for a very long time

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in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans.

At high levels of exposure, some aluminium compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminium compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminium potassium sulphate at high levels in the diet. Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer's disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produce lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, testes, embryotoxicity, and the developing nervous system of 52, 75, 100, and 50 mg aluminium/kg bw/day, respectively. Similarly, the lowest no-observed-adverse-effect levels (NOAELs) for effects on these endpoints were reported at 30, 27, 100, and for effects on the developing nervous system, between 10 and 42 mg aluminium/kg bw per day, respectively.

Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 mg/l aluminium compared with communities where the aluminium level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminium exposure to brain disease. Aluminium concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramid-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations.

Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of "tau" a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminium hyperphosphorylates tau in vitro. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade. Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA).

Aluminium enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminium include baking powder, antacids and aluminium products used for general food preparation and storage (over 12 months, aluminium levels in soft drink packed in aluminium cans rose from 0.05 to 0.9 mg/l). [Walton, J and Bryson-Taylor, D. - Chemistry in Australia, August 1995]. Chronic symptoms produced by crystalline silicas included decreased vital lung capacity and chest infections. Lengthy exposure may cause silicosis a disabling form of pneumoconiosis which may lead to fibrosis, a scarring of the lining of the air sacs in the lung. Symptoms may appear 8 to 18 months after initial exposure. Smoking increases this risk. Classic silicosis is a chronic disease characterised by the formation of scattered, rounded or stellate silica-containing nodules of scar tissue in the lungs ranging from microscopic to 1.0 cm or more. The nodules isolate the inhaled silica particles and protect the surrounding normal and functioning tissue from continuing injury. Simple silicosis (in which the nodules are less than 1.0 cm in diameter) is generally asymptomatic but may be slowly progressive even in the absence of continuing exposure. Simple silicosis can develop in complicated silicoses (in which nodules are greater than 1.0 cm in diameter) and can produce disabilities including an associated tuberculous infection (which 50 years ago accounted for 75% of the deaths among silicotic workers). Crystalline silica deposited in the lungs causes epithelial and macrophage injury and activation. Crystalline silica translocates to the interstitium and the regional lymph nodes and cause the recruitment of inflammatory cells in a dose dependent manner. In humans, a large fraction of crystalline silica persists in the lungs. The question of potential carcinogenicity associated with chronic inhalation of crystalline silica remains equivocal with some studies supporting the proposition and others finding no significant association. The results of recent epidemiological studies suggest that lung cancer risk is elevated only in those patients with overt silicosis. A relatively large

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number of epidemiological studies have been undertaken and in some, increased risk gradients have been observed in relation to dose surrogates - cumulative exposure, duration of exposure, the presence of radiographically defined silicosis, and peak intensity exposure. Chronic inhalation in rats by single or repeated intratracheal instillation produced a significant increase in the incidences of adenocarcinomas and squamous cell carcinomas of the lung. Lifetime inhalation of crystalline silica (87% alpha-quartz) at 1 mg/m<sup>3</sup> (74% respirable) by rats, produced an increase in animals with keratinising cystic squamous cell tumours, adenomas, adenocarcinomas, adenosquamous cell carcinomas, squamous cell carcinoma and nodular bronchiolar alveolar hyperplasia accompanied by extensive subpleural and peribronchiolar fibrosis, increased pulmonary collagen content, focal lipoproteinosis and macrophage infiltration. Thoracic and abdominal malignant lymphomas developed in rats after single intrapleural and intraperitoneal injection of suspensions of several types of quartz.

Some studies show excess numbers of cases of scleroderma, connective tissue disorders, lupus, rheumatoid arthritis chronic kidney diseases, and end-stage kidney disease in workers

NOTE: Some jurisdictions require health surveillance be conducted on workers occupationally exposed to silica, crystalline. Such surveillance should emphasise

- demography, occupational and medical history and health advice
- standardised respiratory function tests such as FEV<sub>1</sub>, FVC and FEV<sub>1</sub>/FVC
- standardised respiratory function tests such as FV<sub>1</sub>, FVC and FEV<sub>1</sub>/FVC
- chest X-ray, full size PA view
- records of personal exposure.

Overexposure to respirable dust may cause coughing, wheezing, difficulty in breathing and impaired lung function. Chronic symptoms may include decreased vital lung capacity, chest infections. Repeated exposures, in an occupational setting, to high levels of fine- divided dusts may produce a condition known as pneumoconiosis which is the lodgement of any inhaled dusts in the lung irrespective of the effect. This is particularly true when a significant number of particles less than 0.5 microns (1/50,000 inch), are present. Lung shadows are seen in the X-ray. Symptoms of pneumoconiosis may include a progressive dry cough, shortness of breath on exertion (exertional dyspnea), increased chest expansion, weakness and weight loss. As the disease progresses the cough produces a stringy mucous, vital capacity decreases further and shortness of breath becomes more severe. Other signs or symptoms include altered breath sounds, diminished lung capacity, diminished oxygen uptake during exercise, emphysema and pneumothorax (air in lung cavity) as a rare complication.

Removing workers from possibility of further exposure to dust generally leads to halting the progress of the lung abnormalities. Where worker-exposure potential is high, periodic examinations with emphasis on lung dysfunctions should be undertaken

Dust inhalation over an extended number of years may produce pneumoconiosis.. Pneumoconiosis is the accumulation of dusts in the lungs and the tissue reaction in its presence. It is further classified as being of noncollagenous or collagenous types. Noncollagenous pneumoconiosis, the benign form, is identified by minimal stromal reaction, consists mainly of reticulin fibres, an intact alveolar architecture and is potentially reversible.

Chronic copper poisoning is rarely recognised in man although in one instance, at least, symptoms more commonly associated with exposures to mercury, namely infantile acrodynia (pink disease), have been described. Tissue damage of mucous membranes may follow chronic dust exposure. A hazardous situation is exposure of a worker with the rare hereditary condition (Wilson's disease or hereditary hepatolenticular degeneration) to copper exposure which may cause liver, kidney, CNS, bone and sight damage and is potentially lethal. Haemolytic anaemia (a result of red-blood cell damage) is common in cows and sheep poisoned by copper derivatives. Overdosing of copper feed supplements has resulted in pigmentary cirrhosis of the liver. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products].

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

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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.

The synthetic, amorphous silicas are believed to represent a very greatly reduced silicosis hazard compared to crystalline silicas and are considered to be nuisance dusts.

When heated to high temperature and a long time, amorphous silica can produce crystalline silica on cooling.

Inhalation of dusts containing crystalline silicas may lead to silicosis, a disabling pulmonary fibrosis that may take years to develop. Discrepancies between various studies showing that fibrosis associated with chronic exposure to amorphous silica and those that do not may be explained by assuming that diatomaceous earth (a non-synthetic silica commonly used in industry) is either weakly fibrogenic or nonfibrogenic and that fibrosis is due to contamination by crystalline silica content.

## TOXICITY AND IRRITATION

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

COPPER(II) OXIDE:

CALCIUM OXIDE:

SODIUM MONOXIDE:

BARIUM OXIDE:

TALC:

■ 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.

BARIUM OXIDE:

SODIUM MONOXIDE:

■ The material may produce respiratory tract irritation. Symptoms of pulmonary irritation may include coughing, wheezing, laryngitis, shortness of breath, headache, nausea, and a burning sensation.

Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence).

The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties.

COPPER(II) OXIDE:

ALUMINIUM OXIDE:

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SODIUM MONOXIDE:

BARIUM OXIDE:

MANGANESE DIOXIDE:

■ No significant acute toxicological data identified in literature search.

TALC:

SILICA AMORPHOUS:

■ 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.

INDICATING OXYGEN TRAP:

■ Not available. Refer to individual constituents.

MANGANESE DIOXIDE:

TOXICITY

Oral (Rat) LD50: >3478 mg/kg

IRRITATION

SILICA AMORPHOUS:

TOXICITY

Oral (rat) LD50: 3160 mg/kg

Dermal (rabbit) LD50: >5000 mg/kg \*

Inhalation (rat) LC50: >0.139 mg/l/14h \* \* [Grace]

■ 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/m<sup>3</sup> to 150 mg/m<sup>3</sup>. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m<sup>3</sup>. When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m<sup>3</sup>. 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

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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.

Reports indicate high/prolonged exposures to amorphous silicas induced lung fibrosis in experimental animals; in some experiments these effects were reversible. [PATTYS]

TALC:  
TOXICITY

IRRITATION  
Skin (human): 0.3 mg/3d- I Mild

■ For talc (a form of magnesium silicate)

The overuse of talc in nursing infants has resulted in pulmonary oedema, pneumonia and death within hours of inhaling talcum powder. The powder dries the mucous membranes of the bronchioles, disrupts pulmonary clearance, clogs smaller airways. Victims display wheezing, rapid or difficult breathing, increased pulse, cyanosis, fever. Mild exposure may cause relatively minor inflammatory lung disease.

Long term exposure may show wheezing, weakness, productive cough, limited chest expansion, scattered rales, cyanosis.

SODIUM MONOXIDE:

■ The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

SILICA CRYSTALLINE - QUARTZ:

TOXICITY

Inhalation (human) LCLo: 0.3 mg/m<sup>3</sup>/10Y  
Inhalation (human) TCLo: 16 mppcf\*/8H/17.9Y  
Inhalation (rat) TCLo: 50 mg/m<sup>3</sup>/6H/71W

IRRITATION  
Nil Reported

■ WARNING: For inhalation exposure ONLY: This substance has been classified by the IARC as Group 1: CARCINOGENIC TO HUMANS.

The International Agency for Research on Cancer (IARC) has classified occupational exposures to respirable (<5 µm) crystalline silica as being carcinogenic to humans. This classification is based on what IARC considered sufficient evidence from epidemiological studies of humans for the carcinogenicity of inhaled silica in the forms of quartz and cristobalite. Crystalline silica is also known to cause silicosis, a non-cancerous lung disease.

Intermittent exposure produces; focal fibrosis, (pneumoconiosis), cough, dyspnoea, liver tumours.

\* Millions of particles per cubic foot (based on impinger samples counted by light field techniques).

NOTE : the physical nature of quartz in the product determines whether it is likely to present a chronic health problem. To be a hazard the material must enter the breathing zone as respirable particles.

ZINC OXIDE:

TOXICITY

Oral (human) LDLo: 500 mg/kg  
Inhalation (human) TCLo: 600 mg/m<sup>3</sup>  
Oral (mouse) LD50: 7950 mg/kg  
Oral (Rat) LD50: >8437 mg/kg

IRRITATION  
Skin (rabbit) : 500 mg/24 h- Mild  
Eye (rabbit) : 500 mg/24 h - Mild

■ The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and

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intracellular oedema of the epidermis.

## CARCINOGEN

Silica, amorphous	International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs	Group	3
Talc containing asbestiform fibres (see Asbestos)	International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs	Group	
Talc not containing asbestos or asbestiform fibres	International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs	Group	3
Talc- based body powder (perineal use of)	International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs	Group	2B
Silica dust, crystalline, in the form of quartz or cristobalite	International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs	Group	1

## Section 12 - ECOLOGICAL INFORMATION

ZINC OXIDE:

COPPER(II) OXIDE:

■ Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters. Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

■ Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

TALC:

COPPER(II) OXIDE:

ALUMINIUM OXIDE:

SODIUM MONOXIDE:

BARIUM OXIDE:

SILICA CRYSTALLINE - QUARTZ:

ZINC OXIDE:

MANGANESE DIOXIDE:

■ Metal-containing inorganic substances generally have negligible vapour pressure and are not expected to partition to air. Once released to surface waters and moist soils their fate depends on solubility and dissociation in water. Environmental processes (such as oxidation and the presence of acids or bases) may transform insoluble metals to more soluble ionic forms. Microbiological processes may also transform insoluble metals to more soluble forms. Such ionic species may bind to dissolved ligands or sorb to solid particles in aquatic or aqueous media. A significant proportion of dissolved/ sorbed metals will end up in sediments through the settling of suspended particles. The remaining metal ions can then be taken up by aquatic organisms.

When released to dry soil most metals will exhibit limited mobility and remain in the upper layer; some will leach locally into ground water and/ or surface water ecosystems when soaked by rain or melt ice.

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Environmental processes may also be important in changing solubilities.

Even though many metals show few toxic effects at physiological pHs, transformation may introduce new or magnified effects.

A metal ion is considered infinitely persistent because it cannot degrade further.

The current state of science does not allow for an unambiguous interpretation of various measures of bioaccumulation.

The counter-ion may also create health and environmental concerns once isolated from the metal. Under normal physiological conditions the counter-ion may be essentially insoluble and may not be bioavailable.

Environmental processes may enhance bioavailability.

SILICA AMORPHOUS:

TALC:

COPPER(II) OXIDE:

ALUMINIUM OXIDE:

CALCIUM OXIDE:

SODIUM MONOXIDE:

BARIUM OXIDE:

SILICA CRYSTALLINE - QUARTZ:

ZINC OXIDE:

MANGANESE DIOXIDE:

■ DO NOT discharge into sewer or waterways.

SILICA AMORPHOUS:

SILICA CRYSTALLINE - QUARTZ:

MANGANESE DIOXIDE:

■ 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  $[\text{Si}(\text{OH})_4]$ . At pH 9.4 the solubility of amorphous silica is about 120 mg  $\text{SiO}_2/\text{l}$ . 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 of opal (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 924 h): >10000 mg/l.

MANGANESE DIOXIDE:

■ 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

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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<sub>3</sub>), and the concentration of manganese is limited by the relatively low solubility (65 mg/L) of MnCO<sub>3</sub>. In relatively oxidized water, the solubility of Mn(II) may be controlled by manganese oxide equilibria, with manganese being converted to the Mn(II) or Mn(IV) oxidation states. In extremely reduced water, the fate of manganese tends to be controlled by formation of a poorly soluble sulfide. Manganese in water may undergo oxidation at high pH or Eh and is also subject to microbial activity. For example, Mn(II) in a lake was oxidized during the summer months, but this was inhibited by a microbial poison, indicating that the oxidation was mediated by bacteria. The microbial metabolism of manganese is presumed to be a function of pH, temperature, and other factors.

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

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

Ecotoxicity:

Manganese ion is toxic to aqueous organisms

Fish LC50 (28 d): orfe 2490 mg/l, trout 2.91 mg/l

Daphnia magna LC50: 50 mg/l

Pseudomonas putida LC50: 10.6 mg/l

Photobacterium phosphoreum LC50: 14.7 mg/l

Turbellarian worms (EC0): Polycelis nigra 660 mg/l (interference threshold); microregma 31 mg/l.

SILICA AMORPHOUS:

mor

COPPER(II) OXIDE:

■ 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. Copper accumulates significantly in the food chain.

Drinking Water Standards:

3000 ug/l (UK max)

2000 ug/l (WHO provisional Guideline)

1000 ug/l (WHO level where individuals complain)

Soil Guidelines: Dutch Criteria

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36 mg/kg (target)

190 mg/kg (intervention)

Air Quality Standards: no data available.

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 (ie.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. Toxicity is also affected by pH and hardness. 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.

Copper exhibits significant toxicity in some aquatic organisms. Some algal species are very sensitive to copper with EC50 (96 hour) values as low as 47 ug/litre dissolved copper whilst for other algal species EC50 values of up to 481 ug/litre have been reported. However many of the reportedly high EC50 values may arise in experiments conducted with a culture media containing copper-complexing agents such as silicate, iron, manganese and EDTA which reduce bioavailability.

Toxic effects arising following exposure by aquatic species to copper are typically:

Algae EC50 (96 h)	Daphnia magna LC50 (48- 96 h)	Amphipods LC50 (48- 96 h)	Gastropods LC50 (48- 96 h)	Crab larvae LC50 (48- 96 h)
47- 481 *	7- 54 *	37- 183 *	58- 112 *	50- 100 *

\* ug/litre

Exposure to concentrations ranging from one to a few hundred micrograms per litre has led to sublethal effects and effects on long-term survival. For high bioavailability waters, effect concentrations for several sensitive species may be below 10 ug Cu/litre.

In fish, the acute lethal concentration of copper ranges from a few ug/litre to several mg/litre, depending both on test species and exposure conditions. Where the value is less than 50 ug Cu/litre, test waters generally have a low dissolved organic carbon (DOC) level, low hardness and neutral to slightly acidic pH. Exposure to concentrations ranging from one to a few hundred micrograms per litre has led to sublethal effects and effects on long-term survival. Lower effect concentrations are generally associated with test waters of high bioavailability.

In summary:

Responses expected for high concentration ranges of copper \*

Total dissolved Cu concentration range (ug/litre)  
1- 10

Effects of high availability in water

Significant effects are expected for diatoms and sensitive invertebrates, notably cladocerans. Effects on fish could be significant in freshwaters with low pH and hardness.

10- 100

Significant effects are expected on various species of microalgae, some species of macroalgae, and a range of invertebrates, including crustaceans, gastropods and sea urchins. Survival of sensitive fish will be affected and a variety of fish show sublethal effects.

100- 1000

Most taxonomic groups of macroalgae and invertebrates will be severely affected. Lethal levels for most fish species will be reached. Lethal concentrations for most tolerant organisms are reached.

>1000

\* Sites chosen have moderate to high bioavailability similar to water used in most toxicity tests.

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In soil, copper levels are raised by application of fertiliser, fungicides, from deposition of highway dusts and from urban, mining and industrial sources. Generally, vegetation rooted in soils reflects the soil copper levels in its foliage. This is dependent upon the bioavailability of copper and the physiological requirements of species concerned.

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

Plants rarely show symptoms of toxicity or of adverse growth effects at normal soil concentrations of copper. Crops are often more sensitive to copper than the native flora, so protection levels for agricultural crops range from 25 mg Cu/kg to several hundred mg/kg, depending on country. Chronic and or acute effects on sensitive species occur at copper levels occurring in some soils as a result of human activities such as copper fertiliser 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. International Programme on Chemical Safety (IPCS): Environmental Health Criteria 200.

## ALUMINIUM OXIDE:

■ For aluminium and its compounds and salts:

Environmental fate:

Aluminium occurs in the environment in the form of silicates, oxides and hydroxides, combined with other elements such as sodium, fluorine and arsenic complexes with organic matter.

Acidification of soils releases aluminium as a transportable solution. Mobilisation of aluminium by acid rain results in aluminium becoming available for plant uptake.

As an element, aluminum cannot be degraded in the environment, but may undergo various precipitation or ligand exchange reactions. Aluminum in compounds has only one oxidation state (+3), and would not undergo oxidation-reduction reactions under environmental conditions. Aluminum can be complexed by various ligands present in the environment (e.g., fulvic and humic acids). The solubility of aluminum in the environment will depend on the ligands present and the pH.

The trivalent aluminum ion is surrounded by six water molecules in solution. The hydrated aluminum ion,  $[\text{Al}(\text{H}_2\text{O})_6]^{3+}$ , undergoes hydrolysis, in which a stepwise deprotonation of the coordinated water ligands forms bound hydroxide ligands (e.g.,  $[\text{Al}(\text{H}_2\text{O})_5(\text{OH})]^{2+}$ ,  $[\text{Al}(\text{H}_2\text{O})_4(\text{OH})_2]^+$ ). The speciation of aluminum in water is pH dependent. The hydrated trivalent aluminum ion is the predominant form at pH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are  $\text{Al}(\text{OH})_2^+$  and  $\text{Al}(\text{OH})_2^+$ , while the solid  $\text{Al}(\text{OH})_3$  is most prevalent between pH 5.2 and 8.8. The soluble species  $\text{Al}(\text{OH})_4^-$  is the predominant species above pH 9, and is the only species present above pH 10. Polymeric aluminum hydroxides appear between pH 4.7 and 10.5, and increase in size until they are transformed into colloidal particles of amorphous  $\text{Al}(\text{OH})_3$ , which crystallise to gibbsite in acid waters. Polymerisation is affected by the presence of dissolved silica; when enough silica is present, aluminum is precipitated as poorly crystallised clay mineral species.

Hydroxyaluminum compounds are considered amphoteric (e.g., they can act as both acids and bases in solution). Because of this property, aluminum hydroxides can act as buffers and resist pH changes within the narrow pH range of 4-5.

Monomeric aluminum compounds, typified by aluminum fluoride, chloride, and sulfate, are considered reactive or labile compounds, whereas polymeric aluminum species react much more slowly in the environment. Aluminum has a stronger attraction for fluoride in an acidic environment compared to other inorganic ligand.

The adsorption of aluminum onto clay surfaces can be a significant factor in controlling aluminum mobility in the environment, and these adsorption reactions, measured in one study at pH 3.0-4.1, have been observed to be very rapid. However, clays may act either as a sink or a source for soluble aluminum depending on the

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degree of aluminum saturation on the clay surface.

Within the pH range of 5-6, aluminum complexes with phosphate and is removed from solution. Because phosphate is a necessary nutrient in ecological systems, this immobilization of both aluminum and phosphate may result in depleted nutrient states in surface water.

Plant species and cultivars of the same species differ considerably in their ability to take up and translocate aluminum to above-ground parts. Tea leaves may contain very high concentrations of aluminum, >5,000 mg/kg in old leaves. Other plants that may contain high levels of aluminum include Lycopodium (Lycopodiaceae), a few ferns, Symplocos (Symplocaceae), and Orites (Proteaceae). Aluminum is often taken up and concentrated in root tissue. In sub-alpine ecosystems, the large root biomass of the Douglas fir, *Abies amabilis*, takes up aluminum and immobilizes it, preventing large accumulation in above-ground tissue. It is unclear to what extent aluminum is taken up into root food crops and leafy vegetables. An uptake factor (concentration of aluminum in the plant/concentration of aluminum in soil) of 0.004 for leafy vegetables and 0.00065 for fruits and tubers has been reported, but the pH and plant species from which these uptake factors were derived are unclear. Based upon these values, however, it is clear that aluminum is not taken up in plants from soil, but is instead biodiluted.

Aluminum concentrations in rainbow trout from an alum-treated lake, an untreated lake, and a hatchery were highest in gill tissue and lowest in muscle. Aluminum residue analyses in brook trout have shown that whole-body aluminum content decreases as the fish advance from larvae to juveniles. These results imply that the aging larvae begin to decrease their rate of aluminum uptake, to eliminate aluminum at a rate that exceeds uptake, or to maintain approximately the same amount of aluminum while the body mass increases. The decline in whole-body aluminum residues in juvenile brook trout may be related to growth and dilution by edible muscle tissue that accumulated less aluminum than did the other tissues.

The greatest fraction of the gill-associated aluminum was not sorbed to the gill tissue, but to the gill mucus. It is thought that mucus appears to retard aluminum transport from solution to the membrane surface, thus delaying the acute biological response of the fish. It has been reported that concentrations of aluminum in whole-body tissue of the Atlantic salmon exposed to high concentrations of aluminum ranging from 3 ug/g (for fish exposed to 33 ug/L) to 96 ug/g (for fish exposed to 264 ug/L) at pH 5.5. After 60 days of exposure, BCFs ranged from 76 to 190 and were directly related to the aluminum exposure concentration. In acidic waters (pH 4.6-5.3) with low concentrations of calcium (0.5-1.5 mg Ca/L), labile aluminum between 25 and 75 ug/L is toxic. Because aluminum is toxic to many aquatic species, it is not bioaccumulated to a significant degree (BCF <300) in most fish and shellfish; therefore, consumption of contaminated fish does not appear to be a significant source of aluminum exposure in humans.

Bioconcentration of aluminum has also been reported for several aquatic invertebrate species. BCF values ranging from 0.13 to 0.5 in the whole-body were reported for the snail. Bioconcentration of aluminum has also been reported for aquatic insects.

Ecotoxicity:

Freshwater species pH >6.5

Fish: Acute LC50 (48-96 h) 5 spp: 0.6 (*Salmo salar*) - 106 mg/L; Chronic NOEC (8-28 d): 7 spp, NOEC, 0.034-7.1 mg/L. The lowest measured chronic figure was an 8-d LC50 of 0.17 mg/L for *Micropterus* sp.

Amphibian: Acute LC50 (4 d): *Bufo americanus*, 0.86-1.66 mg/L; Chronic LC50 (8-d) 2.28 mg/L

Crustaceans LC50 (48 h): 1 sp 2.3-36.9 mg/L; Chronic NOEC (7-28 d) 3 spp, 0.136-1.72 mg/L

Algae EC50 (96 h): population growth, 0.46-0.57 mg/L; 2 spp, chronic NOEC, 0.8-2.0 mg/L

Freshwater species pH <6.5 (all between pH 4.5 and 6.0)

Fish LC50 (24-96 h): 4 spp, 0.015 (*S. trutta*) - 4.2 mg/L; chronic data on *Salmo trutta*, LC50 (21-42 d) 0.015-0.105 mg/L

Amphibians LC50 (4-5 d): 2 spp, 0.540-2.670 mg/L (absolute range 0.40-5.2 mg/L)

Alga: 1 sp NOEC growth 2.0 mg/L

Among freshwater aquatic plants, single-celled plants are generally the most sensitive to aluminum. Fish are generally more sensitive to aluminum than aquatic invertebrates. Aluminum is a gill toxicant to fish, causing both ionoregulatory and respiratory effects.

The bioavailability and toxicity of aluminum is generally greatest in acid solutions. Aluminum in acid habitats has been observed to be toxic to fish and phytoplankton. Aluminum is generally more toxic over the pH range 4.4-5.4, with a maximum toxicity occurring around pH 5.0-5.2. The inorganic single unit aluminum species ( $\text{Al}(\text{OH})_2^+$ ) is thought to be the most toxic. Under very acid conditions, the toxic effects of the high  $\text{H}^+$  concentration appear to be more important than the effects of low concentrations of aluminum; at approximately neutral pH values, the toxicity of aluminum is greatly reduced. The solubility of aluminum is also enhanced under alkaline conditions, due to its amphoteric character, and some researchers found that the acute toxicity of aluminum increased from pH 7 to pH 9. However, the opposite relationship was found in other studies. The uptake and toxicity of aluminum in freshwater organisms generally decreases with

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increasing water hardness under acidic, neutral and alkaline conditions. Complexing agents such as fluoride, citrate and humic substances reduce the availability of aluminium to organisms, resulting in lower toxicity. Silicon can also reduce aluminium toxicity to fish.

Drinking Water Standards:

aluminium: 200 ug/l (UK max.)

200 ug/l (WHO guideline)

chloride: 400 mg/l (UK max.)

250 mg/l (WHO guideline)

fluoride: 1.5 mg/l (UK max.)

1.5 mg/l (WHO guideline)

nitrate: 50 mg/l (UK max.)

50 mg/l (WHO guideline)

sulfate: 250 mg/l (UK max.)

Soil Guideline: none available.

Air Quality Standards: none available.

SODIUM MONOXIDE:

- Prevent, by any means available, spillage from entering drains or water courses.

BARIUM OXIDE:

- For barium and its compounds:

Environmental fate:

Under natural conditions, barium is stable in the +2 valence state and is found primarily in the form of inorganic complexes. Conditions such as pH, Eh (oxidation-reduction potential), cation exchange capacity, and the presence of sulfate, carbonate, and metal oxides (e.g., oxides of aluminum, manganese, silicon, and titanium) will affect the partitioning of barium and its compounds in the environment. The major features of the biogeochemical cycle of barium include wet and dry deposition to land and surface water, leaching from geological formations to groundwater, adsorption to soil and sediment particulates, and biomagnification in terrestrial and aquatic food chains.

Barium is a highly reactive metal that occurs naturally only in a combined state. The element is released to environmental media by both natural processes and anthropogenic sources.

The general population is exposed to barium through consumption of drinking water and foods, usually at low levels. Most barium released to the environment from industrial sources is in forms that do not become widely dispersed. In the atmosphere, barium is likely to be present in particulate form. Although chemical reactions may cause changes in speciation of barium in air, the main mechanisms for the removal of barium compounds from the atmosphere are likely to be wet and dry deposition.

In aquatic media, barium is likely to precipitate out of solution as an insoluble salt (i.e., as BaSO<sub>4</sub> or BaCO<sub>3</sub>). Waterborne barium may also adsorb to suspended particulate matter through the formation of ion pairs with natural anions such as bicarbonate or sulfate in the matter.

Precipitation of barium sulfate salts is accelerated when rivers enter the ocean because of the high sulfate content (905 mg/L) in the ocean. It is estimated that only 0.006% of the total barium input into oceans from freshwater sources remains in solution. Sedimentation of suspended solids removes a large portion of the barium content from surface waters. There is evidence to suggest that the precipitation of barium from the surface of fresh and marine waters occurs, in part, as the result of the barite crystal formation in microorganisms.

Barium in sediments is found largely in the form of barium sulfate (barite). Coarse silt sediment in a turbulent environment will often grind and cleave the barium sulfate from the sediment particles leaving a buildup of dense barites. Estimated soil:water distribution coefficients (K<sub>d</sub>) (i.e., the ratio of the quantity of barium sorbed per gram of sorbent to the concentration of barium remaining in solution at equilibrium) range from 200 to 2,800 for sediments and sandy loam soils. The uptake of barium by fish and marine organisms is also an important removal mechanism. Barium levels in sea water range from 2 to 63 µg/L with a mean concentration of about 13 µg/L. Barium was found to bioconcentrate in marine plants by a factor of 400-4,000 times the level present in the water. Bioconcentration factors in marine animals, plankton, and brown algae of 100, 120, and 260, respectively, have been reported. In freshwater, a bioconcentration factor of 129 was estimated in fish where the barium in water was 0.07 mg/L.

Barium added to soils (e.g., from the land farming of waste drilling muds) may either be taken up by vegetation or transported through soil with precipitation. Relative to the amount of barium found in soils, little is typically bioconcentrated by plants. For example, a bioconcentration factor of 0.4 has been

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estimated for plants in a Virginia floodplain with a barium soil concentration of 104.2 mg/kg . However, there are some plants, such as legumes, forage plants, Brazil nuts, and mushrooms that accumulate barium. Bioconcentration factors from 2 to 20 have been reported for tomatoes and soybeans. Barium is not very mobile in most soil systems, due to the formation of water-insoluble salts and an inability of the barium ion to form soluble complexes with fulvic and humic acids. The rate of transportation of barium in soil is dependent on the characteristics of the soil material. Soil properties that influence the transportation of barium to groundwater are cation exchange capacity, calcium carbonate (CaCO<sub>3</sub>) content and pH. In soil with a high cation exchange capacity (e.g., fine textured mineral soils or soils with high organic matter content), barium mobility will be limited by adsorption. High CaCO<sub>3</sub> content limits mobility by precipitation of the element as BaCO<sub>3</sub>. Barium will also precipitate as barium sulfate in the presence of sulfate ions. Barium is more mobile and is more likely to be leached from soils in the presence of chloride due to the high solubility of barium chloride as compared to other chemical forms of barium. Barium may become more mobile in soils under acid conditions as barium in water-insoluble salts, such as barium sulfate and carbonate, becomes more soluble. Barium complexes with fatty acids (e.g., in acidic landfill leachate) will be much more mobile in the soil due to the lower charge of these complexes and subsequent reduction in adsorption capacity.

## ZINC OXIDE:

■ For zinc and its compounds:

Environmental fate:

Zinc is capable of forming complexes with a variety of organic and inorganic groups (ligands). Biological activity can affect the mobility of zinc in the aquatic environment, although the biota contains relatively little zinc compared to the sediments. Zinc bioconcentrates moderately in aquatic organisms; bioconcentration is higher in crustaceans and bivalve species than in fish. Zinc does not concentrate appreciably in plants, and it does not biomagnify significantly through terrestrial food chains.

However biomagnification may be of concern if concentration of zinc exceeds 1632 ppm in the top 12 inches of soil.

Zinc can persist in water indefinitely and can be toxic to aquatic life. The threshold concentration for fish is 0.1 ppm. Zinc may be concentrated in the aquatic food chain; it is concentrated over 200,000 times in oysters. Copper is synergistic but calcium is antagonistic to zinc toxicity in fish. Zinc can accumulate in freshwater animals at 5 -1,130 times the concentration present in the water. Furthermore, although zinc actively bioaccumulates in aquatic systems, biota appears to represent a relatively minor sink compared to sediments. Steady-state zinc bioconcentration factors (BCFs) for 12 aquatic species range from 4 to 24,000. Crustaceans and fish can accumulate zinc from both water and food. A BCF of 1,000 was reported for both aquatic plants and fish, and a value of 10,000 was reported for aquatic invertebrates. The order of enrichment of zinc in different aquatic organisms was as follows (zinc concentrations in µg/g dry weight appear in parentheses): fish (25), shrimp (50), mussel (60), periphyton (260), zooplankton (330), and oyster (3,300). The high enrichment in oysters may be due to their ingestion of particulate matter containing higher concentrations of zinc than ambient water. Other investigators have also indicated that organisms associated with sediments have higher zinc concentrations than organisms living in the aqueous layer. With respect to bioconcentration from soil by terrestrial plants, invertebrates, and mammals, BCFs of 0.4, 8, and 0.6, respectively, have been reported. The concentration of zinc in plants depends on the plant species, soil pH, and the composition of the soil.

Plant species do not concentrate zinc above the levels present in soil.

In some fish, it has been observed that the level of zinc found in their bodies did not directly relate to the exposure concentrations. Bioaccumulation of zinc in fish is inversely related to the aqueous exposure. This evidence suggests that fish placed in environments with lower zinc concentrations can sequester zinc in their bodies.

The concentration of zinc in drinking water may increase as a result of the distribution system and household plumbing. Common piping materials used in distribution systems often contain zinc, as well as other metals and alloys. Trace metals may enter the water through corrosion products or simply by the dissolution of small amounts of metals with which the water comes in contact. Reactions with materials of the distribution system, particularly in soft low-pH waters, very often have produced concentrations of zinc in tap water much greater than those in the raw or treated waters at the plant of origin. Zinc gives water a metallic taste at low levels. Overexposures to zinc also have been associated with toxic effects. Ingestion of zinc or zinc-containing compounds has resulted in a variety of systemic effects in the gastrointestinal and hematological systems and alterations in the blood lipid profile in humans and animals. In addition, lesions have been observed in the liver, pancreas, and kidneys of animals.

Environmental toxicity of zinc in water is dependent upon the concentration of other minerals and the pH of

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the solution, which affect the ligands that associate with zinc.

Zinc occurs in the environment mainly in the +2 oxidation state. Sorption is the dominant reaction, resulting in the enrichment of zinc in suspended and bed sediments. Zinc in aerobic waters is partitioned into sediments through sorption onto hydrous iron and manganese oxides, clay minerals, and organic material. The efficiency of these materials in removing zinc from solution varies according to their concentrations, pH, redox potential (Eh), salinity, nature and concentrations of complexing ligands, cation exchange capacity, and the concentration of zinc. Precipitation of soluble zinc compounds appears to be significant only under reducing conditions in highly polluted water. Generally, at lower pH values, zinc remains as the free ion. The free ion ( $Zn^{+2}$ ) tends to be adsorbed and transported by suspended solids in unpolluted waters. Zinc is an essential nutrient that is present in all organisms. Although biota appears to be a minor reservoir of zinc relative to soils and sediments, microbial decomposition of biota in water can produce ligands, such as humic acids, that can affect the mobility of zinc in the aquatic environment through zinc precipitation and adsorption.

The relative mobility of zinc in soil is determined by the same factors that affect its transport in aquatic systems (i.e., solubility of the compound, pH, and salinity)

The redox status of the soil may shift zinc partitioning. Reductive dissolution of iron and manganese (hydr)oxides under suboxic conditions release zinc into the aqueous phase; the persistence of suboxic conditions may then lead to a repartitioning of zinc into sulfide and carbonate solids. The mobility of zinc in soil depends on the solubility of the speciated forms of the element and on soil properties such as cation exchange capacity, pH, redox potential, and chemical species present in soil; under anaerobic conditions, zinc sulfide is the controlling species.

Since zinc sulfide is insoluble, the mobility of zinc in anaerobic soil is low. In a study of the effect of pH on zinc solubility: When the pH is  $<7$ , an inverse relationship exists between the pH and the amount of zinc in solution. As negative charges on soil surfaces increase with increasing pH, additional sites for zinc adsorption are activated and the amount of zinc in solution decreases. The active zinc species in the adsorbed state is the singly charged zinc hydroxide species (i.e.,  $Zn[OH]^+$ ). Other investigators have also shown that the mobility of zinc in soil increases at lower soil pH under oxidizing conditions and at a lower cation exchange capacity of soil. On the other hand, the amount of zinc in solution generally increases when the pH is  $>7$  in soils high in organic matter. This is a result of the release of organically complexed zinc, reduced zinc adsorption at higher pH, or an increase in the concentration of chelating agents in soil. For calcareous soils, the relationship between zinc solubility and pH is nonlinear. At a high pH, zinc in solution is precipitated as  $Zn(OH)_2$ , zinc carbonate ( $ZnCO_3$ ), or calcium zincate. Clay and metal oxides are capable of sorbing zinc and tend to retard its mobility in soil. Zinc was more mobile at pH 4 than at pH 6.5 as a consequence of sorption

Zinc concentrations in the air are relatively low, except near industrial sources such as smelters. No estimate for the atmospheric lifetime of zinc is available at this time, but the fact that zinc is transported long distances in air indicates that its lifetime in air is at least on the order of days. There are few data regarding the speciation of zinc released to the atmosphere. Zinc is removed from the air by dry and wet deposition, but zinc particles with small diameters and low densities suspended in the atmosphere travel long distances from emission sources.

Not readily biodegradable

Daphnia magna LC50 (48 h): 0.98 mg/l

Algae EC50: 0.03 mg/l

## Ecotoxicity

Ingredient	Persistence: Water/Soil	Persistence: Air	Bioaccumulation	Mobility
manganese dioxide	No Data Available	No Data Available		
silica amorphous	HIGH	No Data Available	LOW	HIGH
talc	No Data Available	No Data Available		
copper(II) oxide	HIGH	No Data Available	LOW	HIGH
aluminium oxide	No Data Available	No Data Available		

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calcium oxide	No Data Available	No Data Available	LOW
sodium monoxide	No Data Available	No Data Available	
barium oxide	No Data Available	No Data Available	
silica crystalline - quartz	No Data Available	No Data Available	
zinc oxide	No Data Available	No Data Available	LOW

## Section 13 - DISPOSAL CONSIDERATIONS

- Containers may still present a chemical hazard/ danger when empty.
- Return to supplier for reuse/ recycling if possible.

Otherwise:

- If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.

- Where possible retain label warnings and MSDS and observe all notices pertaining to the product.

Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.

A Hierarchy of Controls seems to be common - the user should investigate:

- Reduction
- Reuse
- Recycling
- Disposal (if all else fails)

This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. In most instances the supplier of the material should be consulted.

- DO NOT allow wash water from cleaning or process equipment to enter drains.
- It may be necessary to collect all wash water for treatment before disposal.
- In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
- Where in doubt contact the responsible authority.
- Recycle wherever possible or consult manufacturer for recycling options.
- Consult State Land Waste Authority for disposal.
- Bury or incinerate residue at an approved site.
- Recycle containers if possible, or dispose of in an authorised landfill.

## Section 14 - TRANSPORTATION INFORMATION



Labels Required: SPONTANEOUSLY COMBUSTIBLE, TOXIC

**HAZCHEM:**  
1W (ADG7)

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## ADG7:

Class or Division:	4.2	Subsidiary Risk:	6.1
UN No.:	3191	Packing Group:	II
Special Provision:	274	Limited Quantity:	0
Portable Tanks & Bulk Containers - Instruction:	T3	Portable Tanks & Bulk Containers - Special Provision:	None
Packagings & IBCs - Packing Instruction:	None	Packagings & IBCs - Special Packing Provision:	P410 IBC05

Name and Description: None (contains manganese dioxide and copper(II) oxide)

## Land Transport UNDG:

Class or division:	4.2	Subsidiary risk:	6.1
UN No.:	3191	UN packing group:	II
Shipping Name:	SELF-HEATING SOLID, TOXIC, INORGANIC, N.O.S. (contains manganese dioxide and copper(II) oxide)		

## Air Transport IATA:

ICAO/IATA Class:	4.2	ICAO/IATA Subrisk:	6.1
UN/ID Number:	3191	Packing Group:	II
Special provisions:	A3		
Cargo Only			
Packing Instructions:	470	Maximum Qty/Pack:	50 kg
Passenger and Cargo			
Packing Instructions:	466	Maximum Qty/Pack:	15 kg
Passenger and Cargo			
Limited Quantity			
Packing Instructions:	Forbidden	Maximum Qty/Pack:	Forbidden

Shipping name:SELF-HEATING SOLID, TOXIC, INORGANIC, N.O.S.(contains manganese dioxide and copper(II) oxide)

## Maritime Transport IMDG:

IMDG Class:	4.2	IMDG Subrisk:	6.1
UN Number:	3191	Packing Group:	II
EMS Number:	F- A, S- J	Special provisions:	274
Limited Quantities:	0	Marine Pollutant:	Yes

Shipping name:SELF-HEATING SOLID, TOXIC, INORGANIC, N.O.S.(contains manganese dioxide and copper(II) oxide)

For DOT: Product contains less than 30 g self-heating substance. See 49 CFR Ch. 1 § 173.4 for small quantity exceptions.

For IATA: Product contains less than 30 g self-heating substance. See IATA Section 2.6 for small quantity exceptions.

For both ADR and IMDG: Product contains less than 30 g self-heating substance.

## Section 15 - REGULATORY INFORMATION

POISONS SCHEDULE S5

## REGULATIONS

### Regulations for ingredients

**manganese dioxide (CAS: 1313-13-9) is found on the following regulatory lists;**

"Australia - Tasmania Hazardous Substances Requiring Health Surveillance", "Australia - Western Australia Hazardous Substances Prohibited for Specified Uses or Methods of Handling", "Australia - Western Australia Hazardous Substances Requiring Health Surveillance", "Australia Exposure Standards", "Australia Hazardous

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Substances", "Australia High Volume Industrial Chemical List (HVICL)", "Australia Inventory of Chemical Substances (AICS)", "Australia National Pollutant Inventory", "Australia Therapeutic Goods Administration (TGA) Substances that may be used as active ingredients in Listed medicines", "CODEX General Standard for Food Additives (GSFA) - Additives Permitted for Use in Food in General, Unless Otherwise Specified, in Accordance with GMP", "GESAMP/EHS Composite List - GESAMP Hazard Profiles", "International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs", "International Council of Chemical Associations (ICCA) - High Production Volume List", "International Fragrance Association (IFRA) Survey: Transparency List", "OECD List of High Production Volume (HPV) Chemicals"

**silica amorphous (CAS: 7631-86-9,112945-52-5,67762-90-7,68611-44-9,68909-20-6,112926-00-8,61790-53-2,60676-86-0,91053-39-3,69012-64-2) is found on the following regulatory lists;**

"Australia - Tasmania Hazardous Substances Requiring Health Surveillance", "Australia - Victoria Occupational Health and Safety Regulations - Schedule 5 Hazardous Substances: Substances Prohibited for Specified Uses", "Australia - Western Australia Hazardous Substances Prohibited for Specified Uses or Methods of Handling", "Australia - Western Australia Hazardous Substances Requiring Health Surveillance", "Australia Exposure Standards", "Australia Hazardous Substances", "Australia High Volume Industrial Chemical List (HVICL)", "Australia Inventory of Chemical Substances (AICS)", "Australia National Pollutant Inventory", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix C", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4", "Australia Therapeutic Goods Administration (TGA) Substances that may be used as active ingredients in Listed medicines", "CODEX General Standard for Food Additives (GSFA) - Additives Permitted for Use in Food in General, Unless Otherwise Specified, in Accordance with GMP", "GESAMP/EHS Composite List - GESAMP Hazard Profiles", "International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs", "International Council of Chemical Associations (ICCA) - High Production Volume List", "International Fragrance Association (IFRA) Survey: Transparency List", "OECD List of High Production Volume (HPV) Chemicals", "OSPAR National List of Candidates for Substitution - Norway"

**talca (CAS: 14807-96-6) is found on the following regulatory lists;**

"Australia Exposure Standards", "Australia Hazardous Substances", "Australia High Volume Industrial Chemical List (HVICL)", "Australia Inventory of Chemical Substances (AICS)", "CODEX General Standard for Food Additives (GSFA) - Additives Permitted for Use in Food in General, Unless Otherwise Specified, in Accordance with GMP", "International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs", "OECD List of High Production Volume (HPV) Chemicals", "WHO Food Additives Series - Food Additives considered for specifications only"

**copper(II) oxide (CAS: 1317-38-0,1317-92-6,185461-92-1,1344-70-3) is found on the following regulatory lists;**

"Australia Exposure Standards", "Australia High Volume Industrial Chemical List (HVICL)", "Australia Inventory of Chemical Substances (AICS)", "Australia National Pollutant Inventory", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix A", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6", "Australia Therapeutic Goods Administration (TGA) Substances that may be used as active ingredients in Listed medicines", "International Council of Chemical Associations (ICCA) - High Production Volume List", "International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index", "OECD List of High Production Volume (HPV) Chemicals"

**aluminium oxide (CAS: 1344-28-1) is found on the following regulatory lists;**

"Australia Exposure Standards", "Australia High Volume Industrial Chemical List (HVICL)", "Australia Inventory of Chemical Substances (AICS)", "International Council of Chemical Associations (ICCA) - High Production Volume List", "OECD List of High Production Volume (HPV) Chemicals"

**calcium oxide (CAS: 1305-78-8) is found on the following regulatory lists;**

"Australia Exposure Standards", "Australia Hazardous Substances", "Australia High Volume Industrial Chemical List (HVICL)", "Australia Inventory of Chemical Substances (AICS)", "CODEX General Standard for Food Additives (GSFA) - Additives Permitted for Use in Food in General, Unless Otherwise Specified, in Accordance with GMP", "International Council of Chemical Associations (ICCA) - High Production Volume List", "OECD List of High Production Volume (HPV) Chemicals"

**sodium monoxide (CAS: 1313-59-3,12401-86-4) is found on the following regulatory lists;**

"Australia Inventory of Chemical Substances (AICS)", "OECD List of High Production Volume (HPV) Chemicals"

**barium oxide (CAS: 1304-28-5) is found on the following regulatory lists;**

"Australia Exposure Standards", "Australia Hazardous Substances", "Australia Inventory of Chemical Substances (AICS)", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix I", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6", "OECD List of High Production Volume (HPV) Chemicals"

**silica crystalline - quartz (CAS: 14808-60-7,122304-48-7,122304-49-8,12425-26-2,1317-79-9,70594-95-5,87347-84-0) is found on the following regulatory lists;**

"Australia - New South Wales Hazardous Substances Prohibited for Specific Uses", "Australia - New South Wales Hazardous Substances Requiring Health Surveillance", "Australia - Tasmania Hazardous Substances Requiring Health Surveillance", "Australia - Western Australia Hazardous Substances Prohibited for Specified Uses or Methods of Handling", "Australia - Western Australia Hazardous Substances Requiring Health Surveillance", "Australia Exposure Standards", "Australia Hazardous Substances", "Australia Hazardous Substances Requiring Health Surveillance", "Australia High Volume Industrial Chemical List (HVICL)", "Australia Inventory of Chemical Substances (AICS)", "Australia Occupational Health and Safety (Commonwealth Employment) (National Standards) Regulations 1994 - Hazardous Substances Requiring Health Surveillance", "Australia Therapeutic Goods Administration (TGA) Substances that may be used as active ingredients in Listed medicines", "CODEX General Standard for Food Additives (GSFA) - Additives Permitted for Use in Food in General, Unless Otherwise Specified, in Accordance with GMP", "GESAMP/EHS Composite List - GESAMP Hazard Profiles", "International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs", "International Council of Chemical Associations (ICCA) - High Production Volume List", "International Fragrance Association (IFRA) Survey: Transparency List", "OECD List of High Production Volume (HPV) Chemicals"

**zinc oxide (CAS: 1314-13-2) is found on the following regulatory lists;**

"Australia Exposure Standards", "Australia Hazardous Substances", "Australia High Volume Industrial Chemical List (HVICL)", "Australia Inventory of Chemical Substances (AICS)", "Australia National Pollutant Inventory", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4", "Australia Therapeutic Goods Administration (TGA) Substances that may be used as active ingredients in Listed medicines", "Australia Therapeutic Goods Administration (TGA) Sunscreening agents permitted as active ingredients in listed products", "International Fragrance Association (IFRA) Survey: Transparency List", "OECD List of High Production Volume (HPV) Chemicals"

**No data for Indicating Oxygen Trap (CW: 28-0929)**

continued...

# INDICATING OXYGEN TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 30-Jan-2012

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## Section 16 - OTHER INFORMATION

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### INGREDIENTS WITH MULTIPLE CAS NUMBERS

Ingredient Name	CAS
silica amorphous	7631- 86- 9, 112945- 52- 5, 67762- 90- 7, 68611- 44- 9, 68909- 20- 6, 112926- 00- 8, 61790- 53- 2, 60676- 86- 0, 91053- 39- 3, 69012- 64- 2
copper(II) oxide	1317- 38- 0, 1317- 92- 6, 185461- 92- 1, 1344- 70- 3
sodium monoxide	1313- 59- 3, 12401- 86- 4
silica crystalline - quartz	14808- 60- 7, 122304- 48- 7, 122304- 49- 8, 12425- 26- 2, 1317- 79- 9, 70594- 95- 5, 87347- 84- 0

### MSDS SECTION CHANGES

*The following table displays the version number of and date on which each section was last changed.*

Section Name	Version	Date	Section Name	Version	Date	Section Name	Version	Date
Environmental	2	24- Oct- 2011	Transport	3	30- Jan- 2012			

■ 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](http://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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Issue Date: 30-Jan-2012

Print Date: 27-Feb-2012

*This is the end of the MSDS.*