OXYGEN TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)
Issue Date: 27-Feb-2012
A317L

Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

PRODUCT NAME
OXYGEN TRAP

PROPER SHIPPING NAME
SELF-HEATING SOLID, INORGANIC, N.O.S.(contains copper(II) oxide)

PRODUCT NUMBERS
SGE Part number 103482, 103486

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

Section 2 - HAZARDS IDENTIFICATION

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

RISK
■ May cause fire.
■ Highly flammable.
■ Spontaneously flammable in air.
■ Harmful by inhalation and if swallowed.
■ Irritating to eyes, respiratory system and skin.
■ Harmful: danger of serious damage to health by prolonged exposure through inhalation.
■ Very toxic to aquatic organisms, may cause long- term adverse effects in the aquatic environment.

SAFETY
■ Keep out of reach of children.
■ Do not breathe dust.
■ Avoid contact with skin.
■ Avoid contact with eyes.
■ Wear suitable protective clothing.
■ Wear suitable gloves.
■ Wear eye/face protection.

continued...
**Section 2 - HAIRDTS IDENTIFICATION**

- 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**

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<thead>
<tr>
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<th>CAS RN</th>
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**Section 4 - FIRST AID MEASURES**

**SWALLOWED**

- IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY.
- For advice, contact a Poisons Information Centre or a doctor.
- Urgent hospital treatment is likely to be needed.
- In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition.
- If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the MSDS should be provided. Further action will be the responsibility of the medical specialist.
- If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the MSDS.

Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:
- INDUCE vomiting with fingers down the back of the throat, ONLY IF CONSCIOUS. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.

NOTE: Wear a protective glove when inducing vomiting by mechanical means.

continued...
EYE
• If in eyes, hold eyelids apart and flush the eye continuously with running water.
• Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.
• Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.
• Seek medical attention without delay; if pain persists or recurs seek medical attention.
• Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

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, without delay.

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.
• 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 for charcoals or emesis is, as yet, unproven.
• In severe poisoning CaNa2EDTA has been proposed. [ELLENHORN & BARCELOUX: Medical Toxicology].
• Absorption of zinc compounds occurs in the small intestine.
• The metal is heavily protein bound.
• Elimination results primarily from faecal excretion.
• The usual measures for decontamination (Ipecac Syrup, lavage, charcoal or cathartics) may be administered, although patients usually have sufficient vomiting not to require them.
• CaNa2EDTA has been used successfully to normalise zinc levels and is the agent of choice. [Ellenhorn and Barceloux: Medical Toxicology].
• Manifestation of aluminium toxicity include hypercalcaemia, anaemia, Vitamin D refractory osteodystrophy and a progressive encephalopathy (mixed dysarthria-apraxia of speech, asterixis, tremulousness, myoclonus, dementia, focal seizures). Bone pain, pathological fractures and proximal myopathy can occur.
• Symptoms usually develop insidiously over months to years (in chronic renal failure patients) unless dietary aluminium loads are excessive.
• Serum aluminium levels above 60 ug/ml indicate increased absorption. Potential toxicity occurs above 100 ug/ml and clinical symptoms are present when levels exceed 200 ug/ml.
• Deferoxamine has been used to treat dialysis encephalopathy and osteomalacia. CaNa2EDTA is less effective in chelating aluminium. [Ellenhorn and Barceloux: Medical Toxicology].

Section 5 - FIRE FIGHTING MEASURES

EXTINGUISHING MEDIA
For SMALL FIRES:
• Dry chemical, CO2, water spray or foam.
For LARGE FIRES:
• Foam, fog or water spray
• DO NOT use water jets.

FIRE FIGHTING
• Wear SCBA and fully-encapsulating, gas-tight suits when handling these substances.
• Always wear thermal protective clothing when handling molten substances.
• Structural fire fighter's uniform will only provide limited protection.
• 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.
• Consider evacuation (or protect in place).
• 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.
• Equipment should be thoroughly decontaminated after use.
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

continued...
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.

Combustion products include: silicon dioxide (SiO2), 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. A fire in bulk finely divided carbon may not be obviously visible unless the material is disturbed and sparks appear. A straw broom may be useful to produce the disturbance.

Explosion and Ignition Behaviour of Carbon Black with Air

Lower Limit for Explosion: 50 g/m³ (carbon black in air)
Maximum Explosion Pressure: 10 bar
Maximum Rate of Pressure Rise: 30-100 bar/sec
Minimum Ignition Temperature: 315 deg. C.
Ignition Energy: >1 kJ
Glow Temperature: 500 deg. C. (approx.)

Notes on Test Methods:
Tests 1, 2 and 3 were conducted by Bergwerkschaftliche Versuchstrecke, Dortmunde-Derne, using a 1 m³ vessel with two chemical igniters having an intensity of 5000 W.S. Tests 1 and 2 results are confirmed by information in the Handbook of Powder Technology, Vol. 4 (P. Field) In Test 4, a modified Godbert-Greenwald furnace was used. See U.S. Bureau of Mines, Report 5624, 1960, p.5, “Lab Equipment and Test Procedures”. Test 5 used a 1 m³ vessel with chemical igniters of variable intensity. Test 6 was conducted in a laboratory oven. Active glowing appeared after 3 minutes exposure. (European Committee for Biological Effects of Carbon Black) (2/84).

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

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.
Section 6 - ACCIDENTAL RELEASE MEASURES

- May be violently or explosively reactive.
- DO NOT walk through spilled material.
- DO NOT touch damaged containers or spilled material unless wearing appropriate protective clothing.
- Cover with WET earth, sand or other non-combustible material.
- 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 vermiculite and then covered with a high boiling point mineral oil.
- Recover the liquid using non-sparking appliances and place in labelled, sealable container.
- Water spray may be used to knock down vapours or divert vapour clouds.
- 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.

Section 7 - HANDLING AND STORAGE

PROCEDURE FOR HANDLING

- NOTE:
  - Wet, activated carbon removes oxygen from the air thus producing a severe hazard to workers inside carbon vessels and in enclosed or confined spaces where activated carbons 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.
  - For large scale or continuous use, spark-free, earthen 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.
  - 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

For aluminas (aluminium oxide):
- Incompatible with hot chlorinated rubber.
- 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.
- Forms explosive mixtures with sodium nitrate.
- Reacts vigorously with vinyl acetate.

Zinc oxide:
- slowly absorbs carbon dioxide from the air.
- may react, explosively with magnesium and chlorinated rubber when heated
- is incompatible with linseed oil (may cause ignition).

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 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 ignite 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.

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.
- Avoid strong acids, bases.

For carbon powders:
- Avoid oxidising agents, reducing agents.
- Reaction with finely divided metals, bromates, chlorates, chloramine monoxide, dichlorine oxide, iodates, metal nitrates, oxygen difluoride, peroxyformic acid, peroxyfuroic acid and trioxegen difluoride may result in an exotherm with ignition or explosion. Less active forms of carbon will ignite or explode on suitably intimate contact with oxygen, oxides, peroxides, oxosalts, halogens, interhalogens and other oxidising species.
- Explosive reaction with ammonium nitrate, ammonium perchlorate, calcium hypochlorite and iodine pentoxide may occur following heating. Carbon may react violently with nitric acid and may be explosively reactive with nitrogen trifluoride at reduced temperatures. In the presence of nitrogen oxide, incandescence and ignition may occur. Finely divided or highly porous forms of carbon, exhibiting a high surface area to mass (up to 2000 m2/g) may function as unusually active fuels possessing both adsorptive and catalytic properties which accelerate the release of energy in the presence of oxidising substances. Dry metal-impregnated charcoal catalysts may generate sufficient static, during handling, to cause ignition.
- Graphite in contact with liquid potassium, rubidium or caesium at 300 deg. C. produces intercalation compounds (C8M) which ignite in air and may react explosively with water. The fusion of powdered diamond and potassium hydroxide may produce explosive decomposition.
- Activated carbon, when exposed to air, represents a potential fire hazard due to a high surface area and...
adsorptive capacity. Freshly prepared material may ignite spontaneously in the presence of air especially at high humidity. Spontaneous combustion in air may occur at 90-100 deg. C. The presence of moisture in air facilitates the ignition. Drying oils and oxidising oils promote spontaneous heating and ignition; contamination with these must be avoided. Unsaturated drying oils (linseed oil etc.) may ignite following adsorption owing to an enormous increase in the surface area of oil exposed to air; the rate of oxidation may also be catalysed by metallic impurities in the carbon. A similar, but slower effect occurs on fibrous materials such as cotton waste. Spontaneous heating of activated carbon is related to the composition and method of preparation of the activated carbon. Free radicals, present in charcoal, are responsible for autoignition. Self-heating and autoignition may also result from adsorption of various vapours and gases (especially oxygen). For example, activated carbon auto-ignites in flowing air at 452-518 deg. C.; when the base, triethylenediamine, is adsorbed on the carbon (5%) the autoignition temperature is reduced to 230-260 deg. C.. An exotherm is produced at 230-260 deg. C., at high flow rates of air, although ignition did not occur until 500 deg. C.. Mixtures of sodium borohydride with activated carbons, in air, promote the oxidation of sodium borohydride, producing a self-heating reaction that may result in the ignition of charcoal and in the production of hydrogen through thermal decomposition of the borohydride.

STORAGE REQUIREMENTS

Carbon and charcoal may be stabilised for storage and transport, without moistening, by treatment with hot air at 50 deg. C.. Use of oxygen-impermeable bags to limit oxygen and moisture uptake has been proposed. Surface contamination with oxygenated volatiles may generate a heat of reaction (spontaneous heating). Should stored product reach 110 deg. C., stacked bags should be pulled apart with each bag separated by an air space to permit cooling away from other combustible materials.

• 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 and 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.

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<th>Source</th>
<th>Material</th>
<th>TWA mg/m³</th>
<th>STEL mg/m³</th>
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continued...
Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

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The following materials had no OELs on our records
- sodium monoxide:
  CAS:1313-59-3 CAS:12401-86-4

EMERGENCY EXPOSURE LIMITS

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MATERIAL DATA

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.

CALCIUM OXIDE:

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.

ALUMINIUM OXIDE:

OXYGEN TRAP:

continued...
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.

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.

Graphite pneumonia resembles coal workers' pneumoconiosis. Data indicate that the higher the crystalline silica content of graphite the more likely the disease will increase in severity. The presence of anthracite coal in the production of some synthetic grades of graphite appears to make arbitrary the use of the term, "synthetic", "artificial" or "natural".

The TLV-TWA for carbon black is recommended to minimise complaints of excessive dirtiness and applies only to commercially produced carbon blacks or to soots derived from combustion sources containing absorbed polycyclic aromatic hydrocarbons (PAHs). When PAHs are present in carbon black (measured as the cyclohexane-extractable fraction) NIOSH has established a REL-TWA of 0.1 mg/m$^3$ and considers the material to be an occupational carcinogen.

The NIOSH REL-TWA was "selected on the basis of professional judgement rather than on data delineating safe from unsafe concentrations of PAHs". This limit was justified on the basis of feasibility of measurement and not on a demonstration of its safety.

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$^3$ 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.

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

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

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

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

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

For aluminium oxide and pyrophoric grades of aluminium:
Twenty seven year experience with aluminium oxide dust (particle size 96% 1,2 um) without adverse effects
either systemically or on the lung, and at a calculated concentration equivalent to 2 mg/m3 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.

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.

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/m3 SKIN

SILICA CRYSSTALLINE - QUARTZ:
- WARNING: For inhalation exposure ONLY:

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

EYE

• Safety glasses with side shields.
• Chemical goggles.
• 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 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 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.
Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

- 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

- Exhaust ventilation should be designed to prevent accumulation and recirculation in the workplace and safely remove carbon black from the air.

Note: Wet, activated carbon removes oxygen from the air and thus presents a severe hazard to workers inside carbon vessels and enclosed or confined spaces. Before entering such areas sampling and test procedures for low oxygen levels should be undertaken and control conditions set up to ensure ample oxygen availability.[Linde].

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.

The basic types of engineering controls are:
- Process controls which involve changing the way a job activity or process is done to reduce the risk.
- Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.

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

- Local exhaust ventilation 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 granules with no odour; does not mix with water.

PHYSICAL PROPERTIES
Does not mix with water.

continued...
Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

Floats on water.

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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

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.

Ingestion of finely divided carbon may produce gagging and constipation. Aspiration does not appear to be a concern as the material is generally regarded as inert and is often used as a food additive. Ingestion may produce a black stool.

Soluble zinc salts produces irritation and corrosion of the alimentary tract (in a manner similar to copper salts) with pain, vomiting, etc. Delayed deaths have been ascribed to inanition (weakness and extreme weight loss resulting from prolonged and severe food insufficiency) following severe strictures of the oesophagus, and pylorus. Vomiting, abdominal cramps, and diarrhea, in several cases with blood, have been observed after ingestion of zinc sulfate.

Several cases of gastrointestinal disturbances have been reported after ingestion of zinc sulfate. A significant reduction in erythrocyte superoxide dismutase activity (47% decrease), hematocrit, and serum ferritin, compared to pretreatment levels, occurred in female subjects who received supplements (as capsules) of 50 mg zinc/day as zinc gluconate for 10 weeks. A 15% decrease in erythrocyte superoxide dismutase activity was reported in male volunteers receiving 50 mg zinc/day as zinc gluconate for 6 weeks. Another study reported increases in bone specific alkaline phosphatase levels (~25%) and extracellular superoxide dismutase (~15%), while significant decreases were seen in mononuclear white cell 5'-nucleotidase (~30%) and plasma 5'-nucleotidase activity (~36%) following exposure of postmenopausal women to a combined (dietary+supplemental) 53 mg zinc/day as zinc glycine chelate. Healthy men given 200 mg zinc/day as elemental zinc for 6 weeks showed a reduction in lymphocyte stimulation response to phytohemagglutinin as well as chemotaxis and phagocytosis of bacteria by polymorphonuclear leukocytes.; however, no changes in lymphocyte cell number or in the proportion of lymphocyte populations were noted. Exposure of male volunteers to 0.48 mg zinc/kg/day, as zinc glycine chelate, had no effect on markers of coagulation relative to unexposed subjects. While the changes in hematological end points following long-term zinc exposure in humans are noteworthy, they were subclinical in continued...
nature, and therefore, are generally considered to be non-adverse. In animals, following oral administration of zinc compounds, decreased hemoglobin, hematocrit, erythrocyte, and/or leukocyte levels were observed in rats, mice, rabbits, dogs, ferrets, and preruminant calves. A number of intermediate-duration studies have demonstrated renal effects in animals exposed to zinc oxide, zinc sulfate, and zinc acetate. Zinc sulfate caused an increase in the absolute and relative kidney weights and regressive kidney lesions (not specified) in female mice that consumed 1,110 mg zinc/kg/day in the diet for 13 weeks, but no effects occurred in rats that consumed 565 mg zinc/kg/day or in mice that consumed 104 mg zinc/kg/day under similar conditions. Severe diffuse nephrosis was observed in ferrets exposed to 195 mg zinc/kg/day as zinc oxide in the diet. In rats exposed to 191 mg zinc/kg/day as zinc acetate for 3 months, epithelial cell damage in the glomerulus and proximal convoluted tubules and increased plasma creatinine and urea levels were observed. Zinc plays a role in the normal development and maintenance of the immune system, such as in the lymphocyte response to mitogens and as a cofactor for the thymic hormone thymulin. Oral exposure to zinc at levels much higher than the recommended daily dose has impaired immune and inflammatory responses. This was observed in in vivo investigations of the immune competence of blood components taken from 11 healthy adult men after ingestion of 4.3 mg zinc/kg/day as zinc sulfate for 6 weeks. The mitogenic response elicited from peripheral blood lymphocytes and the chemotactic and phagocytic responses of polymorphonuclear leukocytes were impaired after zinc ingestion. No effects were seen on total numbers of lymphocytes or relative numbers of T cells, T cell subsets, or B cells. The relationship between these observations and decreased levels of immune competence that might lead to increased susceptibility to disease is unknown. A later study reported no effects of supplementation of male volunteers with 30 mg zinc/day (0.43 mg zinc/kg/day assuming a reference male body weight of 70 kg) as zinc glycine chelate for 14 weeks on levels of peripheral blood leukocytes or on the frequency of lymphocyte subsets.

Zinc appears to be necessary for normal brain function, but excess zinc is toxic. A 16-year-old boy who ingested .86 mg zinc/kg/day of metallic zinc over a 2-day period in an attempt to promote wound healing, developed signs and symptoms of lethargy, light-headedness, staggering, and difficulty in writing clearly. Lethargy was also observed in a 2-year-old child who ingested a zinc chloride solution (.1,000 mg zinc/kg). It is not known whether these observations represent direct effects on the nervous system. Very limited data were located regarding neurological effects in animals. Minor neuron degeneration and proliferation of oligodendroglia occurred in rats dosed with 487 mg zinc/kg/day as zinc oxide for 10 days. Rats receiving 472 mg zinc/kg/day for 10 days had increased levels of secretory material in the neurosecretory nuclei of the hypothalamus. Mice exposed postnataally to 0.5 mg zinc/kg/day as zinc acetate for 28 days showed no changes in memory formation, but showed a gradual decrease in learning extinction throughout the study. 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.]
OXYGEN TRAP
Chemwatch Independent Material Safety Data Sheet (REVIEW)
Issue Date: 27-Feb-2012
A317L

Section 11 - TOXICOLOGICAL INFORMATION

EYE
■ Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals.
Reversible or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Symptoms of exposure by the eye to carbon particulates include irritation and a burning sensation. Following an industrial explosion, fine particles become embedded in the cornea and conjunctiva resulting in an inflammation which persisted for 2-3 weeks. Some particles remained permanently producing a punctate purplish-black discolouration.
Copper salts, in contact with the eye, may produce conjunctivitis or even ulceration and turbidity of the cornea.

SKIN
■ The material produces mild skin irritation; evidence exists, or practical experience predicts, that the material either
  • produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or
  • produces significant, but mild, 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.
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 systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Repeated or excessive handling, coupled with poor personal hygiene, may result in acne-like eruptions known as “zinc oxide pox”.

INHALED
■ Inhalation of dusts, generated by the material, during the course of normal handling, may be harmful.
Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.
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

continued...
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.

Although carbon itself has no toxic action, associated impurities may be toxic. Iodine is often found as an impurity and air-borne carbon dusts, as a result, may produce irritation of the mucous membranes, the eyes, and skin. Symptoms of exposure may include coughing, irritation of the nose and throat and burning of the eyes.

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

Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure.

CHRONIC HEALTH EFFECTS

Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.

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 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 alveolii (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 osseus 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 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 continued...
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]. Prolonged or repeated inhalation of dust may result in pneumoconiosis (lung disease caused by inhalation dust).

Graphite workers have reported symptoms of headaches, coughing, depression, low appetite, dyspnoea (difficult breathing) and black sputum. A number of studies indicate that graphitosis is a progressive and disabling disease and that the presence of crystalline silica and some silicates as graphite impurities have a pronounced synergistic effect. Workers suffering from graphite pneumoconiosis have generally worked in the industry for long periods, i.e. 10 years or more, although some cases have been reported after as little as four years. Data indicate the higher the crystalline silica content of graphite the greater is the severity of the pneumoconiosis.

Pre-employment and periodic examinations should be directed towards detecting significant respiratory disease through chest X-rays and pulmonary function tests. 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 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/m3 (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 FEV1, FVC and FEV1/FVC
- standardised respiratory function tests such as FV1, FVC and FEV1/FVC
- chest X-ray, full size PA view
- records of personal exposure.

continued...
Epidemiological studies of workers in the carbon black producing industries of North America and Western Europe show no significant health effect due to occupational exposure to carbon black. Early studies in the former USSR and Eastern Europe report respiratory diseases amongst workers exposed to carbon black, including bronchitis, pneumonia, emphysema and rhinitis. These studies are of questionable validity due to inadequate study design and methodology, lack of appropriate controls for cigarette smoking and other confounding factors such as concurrent exposure to carbon dioxide, coal oil and petroleum vapours. Moreover, review of these studies indicates that the concentrations of carbon black were greater than current occupational standards.

Carbon black may cause adverse pulmonary changes following prolonged or repeated inhalation of the dust; these include oral mucosal lesions, bronchitis and pneumoconiosis which may lead to lung tumours. In a study on the mortality and morbidity experience from all forms of cancer (over a period of 17.5 years) in employees of a large producer of carbon black, it was concluded that the incidence of cancer and death rate from cancer was low, compared to a cohort. In IARC Monograph 65, (April 1996) it was concluded that "although one cohort study on the carbon black production industry showed slight excesses of cancer, the totality of the epidemiological studies, both in the carbon black production industry and in some user industries, suggest that there is inadequate evidence for the carcinogenicity in humans of carbon black."

In studies employing channel and furnace black, hamsters, mice, guinea pigs, rabbits and monkeys exposed to dusts for 7 hours/day, 5 days/week, at concentrations of 87.4 mg/m3 for channel black and 56.5 mg/m3 for furnace black, no malignancies were observed in any of the animals. Channel black had little if any absorbed polyaromatic hydrocarbons (PAHs) (as benzene extractables) whilst furnace black had 0.28%.

Monkeys exposed to channel black for 1000-1500 hours showed evidence of electrocardiac changes indicative of right atrial and right ventricular strain. These changes increased progressively until after 10,000 hours of exposure, when the changes were marked. The authors of this study concluded that there was no significant effect due to prolonged exposure other than those expected from the accumulation of non-toxic dusts in the pulmonary system. Exposure to furnace black produced a similar picture although electrocardiographic change was first observed in monkeys after 2500 hours' exposure and marked atrial and right ventricular strain after 10,000 hours' exposure. The authors concluded that there was no significant effect due to prolonged exposure other than those expected from the accumulation of nontoxic dusts in the pulmonary system. Exposure to furnace black produced a similar picture although electrocardiographic change was first observed in monkeys after 2500 hours exposure and marked atrial and right ventricular strain after 10,000 hours exposure.

Chromatographic fractions of oily material extracted from carbon black have been shown to be carcinogenic whilst the unfractonated extracts are not. The activity of some carcinogens appear to be inhibited by carbon black itself.

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 carbon 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.

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Zinc is necessary for normal fetal growth and development. Fetal damage may result from zinc deficiency. Only one report in the literature suggested adverse developmental effects in humans due to exposure to excessive levels of zinc. Four women were given zinc supplements of 0.6 mg zinc/kg/day as zinc sulfate during the third trimester of pregnancy. Three of the women had premature deliveries, and one delivered a stillborn infant. However, the significance of these results cannot be determined because very few details were given regarding the study protocol, reproductive histories, and the nutritional status of the women. Other human studies have found no developmental effects in the newborns of mothers consuming 0.3 mg zinc/kg/day as zinc sulfate or zinc citrate or 0.06 mg zinc/kg/day as zinc aspartate during the last two trimesters. There has been a suggestion that increased serum zinc levels in pregnant women may be associated with an increase in neural tube defects, but others have failed to confirm this association. The developmental toxicity of zinc in experimental animals has been evaluated in a number of investigations. Exposure to high levels of zinc in the diet prior to and/or during gestation has been associated with increased fetal resorptions, reduced fetal weights, altered tissue concentrations of fetal iron and copper, and reduced growth in the offspring. Animal studies suggest that exposure to very high levels of dietary zinc is associated with reduced fetal weight, alopecia, decreased hematocrit, and copper deficiency in offspring. For example, second generation mice exposed to zinc carbonate during gestation and lactation (260 mg/kg/day in the maternal diet), and then continued on that diet for 8 weeks, had reduced body weight, alopecia, and signs of copper deficiency (e.g., lowered hematocrit and occasional achromotrichia [loss of hair colour]). Similarly, mink kits from dams that ingested a time-weighted-average dose of 20.8 mg zinc/kg/day as zinc sulfate also had alopecia and achromotrichia. It is likely that the alopecia resulted from zinc-induced copper deficiency, which is known to cause alopecia in monkeys. However, no adverse effects were observed in parental mice or mink. No effects on reproduction were reported in rats exposed to 50 mg zinc/kg/day as zinc carbonate; however, increased stillbirths were observed in rats exposed to 250 mg zinc/kg/day. Welding or flame cutting of metals with zinc or zinc dust coatings may result in inhalation of zinc oxide fume; high concentrations of zinc oxide fume may result in "metal fume fever"; also known as "brass chills", an industrial disease of short duration. [I.L.O] Symptoms include malaise, fever, weakness, nausea and may appear quickly if operations occur in enclosed or poorly ventilated areas.

Genotoxicity studies conducted in a variety of test systems have failed to provide evidence for mutagenicity of zinc. However, there are indications of weak clastogenic effects following zinc exposure. 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.

**CALCIUM OXIDE:**

**SODIUM MONOXIDE:**

**GRAPHITE:**

- Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
GRAPHITE:

SODIUM MONOXIDE:

ALUMINIUM OXIDE:

- No significant acute toxicological data identified in literature search.

OXYGEN TRAP:

- Not available. Refer to individual constituents.

ZINC OXIDE:

TOXICITY

IRRITATION

Oral (human) LDLo: 500 mg/kg
Skin (rabbit): 500 mg/24 h - Mild

Inhalation (human) TCLo: 600 mg/m³
Eye (rabbit): 500 mg/24 h - Mild

Oral (mouse) LD50: 7950 mg/kg

Oral (Rat) LD50: >8437 mg/kg

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

SILICA AMORPHOUS:

TOXICITY

IRRITATION

Oral (rat) LD50: 3160 mg/kg
Skin (rabbit): non-irritating*

Dermal (rabbit) LD50: >5000 mg/kg *
Eye (rabbit): non-irritating *

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/m³ to 150 mg/m³. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m³. When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m³. The difference in values may be explained by different particle size, and therefore the number of particles administered per unit dose. In general, as particle size decreases so does the NOAEL/LOAEL.

Neither inhalation nor oral administration caused neoplasms (tumours). SAS is not mutagenic in vitro. No genotoxicity was detected in in vivo assays. SAS does not impair development of the foetus. Fertility was not specifically studied, but the reproductive organs in long-term studies were not affected.

In humans, SAS is essentially non-toxic by mouth, skin or eyes, and by inhalation. Epidemiology studies show little evidence of adverse health effects due to SAS. Repeated exposure (without personal protection) may
cause mechanical irritation of the eye and drying/cracking of the skin. There is no evidence of cancer or other long-term respiratory health effects (for example, silicosis) in workers employed in the manufacture of SAS. Respiratory symptoms in SAS workers have been shown to correlate with smoking but not with SAS exposure, while serial pulmonary function values and chest radiographs are not adversely affected by long-term exposure to SAS. The substance is classified by IARC as Group 3:
NOT classifiable as to its carcinogenicity to humans.
Evidence of carcinogenicity may be inadequate or limited in animal testing.
Reports indicate high/prolonged exposures to amorphous silicas induced lung fibrosis in experimental animals; in some experiments these effects were reversible. [PATTYS]

SODIUM MONOXIDE:
■ The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
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.
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:
■ WARNING: For inhalation exposure ONLY: This substance has been classified by the IARC as Group 1: CARCINOGENIC TO HUMANS.

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

* Millions of particles per cubic foot (based on impinger samples counted by light field techniques). 
NOTE: the physical nature of quartz in the product determines whether it is likely to present a chronic health problem. To be a hazard the material must enter the breathing zone as respirable particles.

Carcinogen
Silica, amorphous
International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs
Group 3

continued...
Section 11 - TOXICOLOGICAL INFORMATION

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

SILICA CRYSTALLINE - QUARTZ:
SILICA AMORPHOUS:

For silica:
The literature on the fate of silica in the environment concerns dissolved silica in the aquatic environment, irrespective of its origin (man-made or natural), or structure (crystalline or amorphous). Indeed, once released and dissolved into the environment no distinction can be made between the initial forms of silica. At normal environmental pH, dissolved silica exists exclusively as monosilicic acid [Si(OH)₄]. At pH 9.4 the solubility of amorphous silica is about 120 mg SiO₂/l. Quartz has a solubility of only 6 mg/l, but its rate of dissolution is so slow at ordinary temperature and pressure that the solubility of amorphous silica represents the upper limit of dissolved silica concentration in natural waters. Moreover, silicic acid is the bioavailable form for aquatic organisms and it plays an important role in the biogeochemical cycle of Si, particularly in the oceans.

In the oceans, the transfer of dissolved silica from the marine hydrosphere to the biosphere initiates the global biological silicon cycle. Marine organisms such as diatoms, silicoflagellates and radiolarians build up their skeletons by taking up silicic acid from seawater. After these organisms die, the biogenic silica accumulated in them partly dissolves. The portion of the biogenic silica that does not dissolve settles and ultimately reaches the sediment. The transformation 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.

ALUMINIUM OXIDE:
SODIUM MONOXIDE:
SILICA CRYSTALLINE - QUARTZ:

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. 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.

ALUMINIUM OXIDE:
SILICA AMORPHOUS:

continued...
ZINC OXIDE:

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

  continued...
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

**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, fluoride 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, aluminium cannot be degraded in the environment, but may undergo various precipitation or ligand exchange reactions. Aluminium in compounds has only one oxidation state (+3), and would not undergo oxidation-reduction reactions under environmental conditions. Aluminium can be complexed by various ligands present in the environment (e.g., fulvic and humic acids). The solubility of aluminium in the environment will depend on the ligands present and the pH.
    - The trivalent aluminium ion is surrounded by six water molecules in solution. The hydrated aluminium ion, [Al(H_{2}O)_{6}]^{3+}, undergoes hydrolysis, in which a stepwise deprotonation of the coordinated water ligands forms bound hydroxide ligands (e.g., [Al(H_{2}O)_{5}(OH)]^{2+}, [Al(H_{2}O)_{4}(OH)_{2}]^{+}). The speciation of aluminium in water is pH dependent. The hydrated trivalent aluminium ion is the predominant form at pH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are Al(OH)_{2}^{+} and Al(OH)_{2}^{2+}, while the solid Al(OH)_{3} is most prevalent between pH 5.2 and 8.8. The soluble species Al(OH)_{4}^{-} is the predominant species above pH 9, and is the only species present above pH 10. Polymeric aluminium hydroxides appear between pH 4.7 and 10.5, and increase in size until they are transformed into colloidal particles of amorphous Al(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. Hydroxyluminum 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 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 age of 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 spp, 0.28 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 aluminium. Fish are generally more sensitive to aluminium than aquatic invertebrates. Aluminium is a gill toxicant to fish,
causing both ionoregulatory and respiratory effects. The bioavailability and toxicity of aluminium is generally greatest in acid solutions. Aluminium in acid habitats has been observed to be toxic to fish and phytoplankton. Aluminium 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 aluminium species (Al(OH)2 +) is thought to be the most toxic. Under very acid conditions, the toxic effects of the high H+ concentration appear to be more important than the effects of low concentrations of aluminium; at approximately neutral pH values, the toxicity of aluminium is greatly reduced. The solubility of aluminium is also enhanced under alkaline conditions, due to its amphoterically character, and some researchers found that the acute toxicity of aluminium increased from pH 7 to pH 9. However, the opposite relationship was found in other studies. The uptake and toxicity of aluminium in freshwater organisms generally decreases with 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.

SILICA AMORPHOUS:
- mor

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

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Section 13 - DISPOSAL CONSIDERATIONS

- Containers may still present a chemical hazard/ danger when empty.
- Return to supplier for reuse/ recycling if possible.
- Otherwise: continued...
Section 13 - DISPOSAL CONSIDERATIONS

• 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

HAZCHEM:
1Y (ADG7)

ADG7:
Class or Division: 4.2
UN No.: 3190
Special Provision: 274
Portable Tanks & Bulk Containers -
Instruction: None
Packagings & IBCs -
Packing Instruction: None
Name and Description: None (contains copper(II) oxide)

Land Transport UNDG:
Class or division: 4.2
UN No.: 3190
Shipping Name: SELF-HEATING SOLID, INORGANIC, N.O.S. (contains copper(II) oxide)

holding...
Section 14 - TRANSPORTATION INFORMATION

Air Transport IATA:
- ICAO/IATA Class: 4.2
- UN/ID Number: 3190
- Special provisions: Cargo Only
- Packing Instructions: 470 Maximum Qty/Pack: 50 kg
- Passenger and Cargo Packing Instructions: 467 Maximum Qty/Pack: 15 kg
- Limited Quantity Packing Instructions: Forbidden

Maritime Transport IMDG:
- IMDG Class: 4.2
- UN Number: 3190
- EMS Number: F- A, S- J
- Limited Quantities: 0
- Packing Group: II
- Special provisions: 274
- Marine Pollutant: Yes

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

Section 15 - REGULATORY INFORMATION

POISONS SCHEDULE None

REGULATIONS

Regulations for ingredients

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

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

silica amorphous (CAS: 7631-86-9,112945-52-5,67762-90-7,68611-44-9,68909-20-6,112926-00-8, 61790-52-2,60676-86-0,91053-39-3,69012-64-2) 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
- 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
- OECD List of High Production Volume (HPV) Chemicals
- OSPAR National List of Candidates for Substitution – Norway

graphite (CAS: 7782-42-5) is found on the following regulatory lists:
- Australia Dangerous Goods Code (ADG Code) - Goods Too Dangerous To Be Transported
- Australia Exposure Standards
- Australia Hazardous Substances
- Australia High Volume Industrial Chemical List (HVICL)
- Australia Inventory of Chemical Substances (AICS)
- Australia National Pollutant Inventory
- International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs
- 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 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
OXYGEN TRAP

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Section 15 - REGULATORY INFORMATION

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”*

sila 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 Specfic Uses”*;
*“Australia - New South Wales Hazardous Substances Requiring Health Surveillance”*;
*“Australia - South Australia Hazardous Substances Requiring Health Surveillance”*;
*“Australia - Tasmania Hazardous Substances Prohibited for Specified Uses”*;
*“Australia - Western Australia Hazardous Substances Prohibited for Specified Uses or Methods of Handling”*;
*“Australia Hazardous Substances Requiring Health Surveillance”*;
*“Australia High Volume Industrial Chemical List (HVICL)”*;
*“Australia Inventory of Chemical Substances (AICS)”*;
*“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”*

No data for Oxygen Trap (CW: 28-0934)

Section 16 - OTHER INFORMATION

INGREDIENTS WITH MULTIPLE CAS NUMBERS

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MSDS SECTION CHANGES

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Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

A list of reference resources used to assist the committee may be found at: www.chemwatch.net/references.

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

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This is the end of the MSDS.