

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 1 of 21

Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

PRODUCT NAME

MOISTURE TRAP

PRODUCT NUMBERS

SGE Part number 103480, 103487

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. NON-DANGEROUS GOODS. According to the Criteria of NOHSC, and the ADG Code.

RISK

■ Causes burns.
■ Risk of serious damage to eyes.

SAFETY

- Keep locked up.
- 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.
- Use only in well ventilated areas.
- Keep container in a well ventilated place.
- To clean the floor and all objects contaminated by this material, use water and detergent.
- Take off immediately all contaminated clothing.

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 2 of 21

Section 2 - HAZARDS IDENTIFICATION

- In case of contact with eyes, rinse with plenty of water and contact Doctor or Poisons Information Centre.
- In case of accident or if you feel unwell IMMEDIATELY contact Doctor or Poisons Information Centre (show label if possible).

Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

NAME	CAS RN	%
silica amorphous	7631-86-9	0-75
sodium monoxide	1313-59-3	0-30
aluminium oxide	1344-28-1.	0-50
calcium oxide	1305-78-8	0-30
silica crystalline - quartz	14808-60-7	<7

Section 4 - FIRST AID MEASURES

SWALLOWED

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

EYE

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

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.

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.

continued...

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 3 of 21

Section 4 - FIRST AID MEASURES

- If dust is inhaled, remove from contaminated area.
- Encourage patient to blow nose to ensure clear breathing passages.
- Ask patient to rinse mouth with water but to not drink water.
- Seek immediate medical attention.

NOTES TO PHYSICIAN

- Treat symptomatically.
- 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].

For acute or short-term repeated exposures to highly alkaline materials:

- Respiratory stress is uncommon but present occasionally because of soft tissue edema.
- Unless endotracheal intubation can be accomplished under direct vision, cricothyroidotomy or tracheotomy may be necessary.
- Oxygen is given as indicated.
- The presence of shock suggests perforation and mandates an intravenous line and fluid administration.
- Damage due to alkaline corrosives occurs by liquefaction necrosis whereby the saponification of fats and solubilisation of proteins allow deep penetration into the tissue.

Alkalis continue to cause damage after exposure.

INGESTION:

- Milk and water are the preferred diluents
- No more than 2 glasses of water should be given to an adult.
- Neutralising agents should never be given since exothermic heat reaction may compound injury.
 - * Catharsis and emesis are absolutely contra-indicated.
 - * Activated charcoal does not absorb alkali.
 - * Gastric lavage should not be used.

Supportive care involves the following:

- Withhold oral feedings initially.
- If endoscopy confirms transmucosal injury start steroids only within the first 48 hours.
- Carefully evaluate the amount of tissue necrosis before assessing the need for surgical intervention.
- Patients should be instructed to seek medical attention whenever they develop difficulty in swallowing (dysphagia).

SKIN AND EYE:

- Injury should be irrigated for 20-30 minutes.

Eye injuries require saline. [Ellenhorn & Barceloux: Medical Toxicology].

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

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

[Ellenhorn and Barceloux: Medical Toxicology].

continued...

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 4 of 21

Section 5 - FIRE FIGHTING MEASURES

EXTINGUISHING MEDIA

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

FIRE FIGHTING

- When silica dust is dispersed in air, firefighters should wear inhalation protection as hazardous substances from the fire may be adsorbed on the silica particles.
- When heated to extreme temperatures, (>1700 deg.C) amorphous silica can fuse.
- Alert Fire Brigade and tell them location and nature of hazard.
- Wear breathing apparatus plus protective gloves in the event of a fire.
- Prevent, by any means available, spillage from entering drains or water courses.
- Use fire fighting procedures suitable for surrounding area.
- DO NOT approach containers suspected to be hot.
- Cool fire exposed containers with water spray from a protected location.
- If safe to do so, remove containers from path of fire.
- Equipment should be thoroughly decontaminated after use.

FIRE/EXPLOSION HAZARD

- Non combustible.
 - Not considered a significant fire risk, however containers may burn.
- Decomposition may produce toxic fumes of: 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.
May emit poisonous fumes.
May emit corrosive fumes.

FIRE INCOMPATIBILITY

- None known.

HAZCHEM

None

Section 6 - ACCIDENTAL RELEASE MEASURES

MINOR SPILLS

- Remove all ignition sources.
- Clean up all spills immediately.
- Avoid contact with skin and eyes.
- Control personal contact with the substance, by using protective equipment.
- Use dry clean up procedures and avoid generating dust.
- Place in a suitable, labelled container for waste disposal.

MAJOR SPILLS

- Moderate hazard.
- CAUTION: Advise personnel in area.
- Alert Emergency Services and tell them location and nature of hazard.
- Control personal contact by wearing protective clothing.
- Prevent, by any means available, spillage from entering drains or water courses.
- Recover product wherever possible.
- IF DRY: Use dry clean up procedures and avoid generating dust. Collect residues and place in sealed plastic bags or other containers for disposal. IF WET: Vacuum/shovel up and place in labelled containers for disposal.
- ALWAYS: Wash area down with large amounts of water and prevent runoff into drains.
- If contamination of drains or waterways occurs, advise Emergency Services.

continued...

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 5 of 21

Section 6 - ACCIDENTAL RELEASE MEASURES

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

Section 7 - HANDLING AND STORAGE

PROCEDURE FOR HANDLING

- Avoid all personal contact, including inhalation.
- Wear protective clothing when risk of exposure occurs.
- Use in a well-ventilated area.
- Prevent concentration in hollows and sumps.
- DO NOT enter confined spaces until atmosphere has been checked.
- DO NOT allow material to contact humans, exposed food or food utensils.
- Avoid contact with incompatible materials.
- When handling, DO NOT eat, drink or smoke.
- Keep containers securely sealed when not in use.
- Avoid physical damage to containers.
- Always wash hands with soap and water after handling.
- Work clothes should be laundered separately. Launder contaminated clothing before re-use.
- Use good occupational work practice.
- Observe manufacturer's storage and handling recommendations contained within this MSDS.
- Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.

SUITABLE CONTAINER

- Polyethylene or polypropylene container.
- Check all containers are clearly labelled and free from leaks.

STORAGE INCOMPATIBILITY

- For aluminas (aluminium oxide):
 - Incompatible with hot chlorinated rubber.
 - In the presence of chlorine trifluoride may react violently and ignite.
 - May initiate explosive polymerisation of olefin oxides including ethylene oxide.
 - Produces exothermic reaction above 200 C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals.
 - Produces exothermic reaction with oxygen difluoride.
 - May form explosive mixture with oxygen difluoride.
 - Forms explosive mixtures with sodium nitrate.
 - Reacts vigorously with vinyl acetate.
- Calcium oxide:
 - reacts violently with water, evolving high quantities of heat
 - reacts violently, with possible ignition or explosion, with acids, anilinium perchlorate, bromine pentafluoride, chlorine trifluoride, fluorine, hydrogen fluoride, hydrazine, hydrogen sulfide, hydrogen trisulfide, isopropyl isocyanide dichloride, light metals, lithium, magnesium, powdered aluminium, phosphorus, potassium, sulfur trioxide
 - increase the explosive sensitivity of azides, nitroalkanes (e.g. nitroethane, nitromethane, 1-nitropropane etc.)
 - is incompatible with boric acid, boron trifluoride, carbon dioxide, ethanol, halogens (such as fluorine), metal halides, phosphorus pentoxide, selenium oxychloride, sulfur dioxide and many organic materials.
- 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

continued...

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 6 of 21

Section 7 - HANDLING AND STORAGE

- may react vigorously when heated with alkali carbonates.
- Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.
- Avoid contact with copper, aluminium and their alloys.

STORAGE REQUIREMENTS

- Store in original containers.
- Keep containers securely sealed.
- Store in a cool, dry area protected from environmental extremes.
- Store away from incompatible materials and foodstuff containers.
- Protect containers against physical damage and check regularly for leaks.
- Observe manufacturer's storage and handling recommendations contained within this MSDS.

For major quantities:

- Consider storage in bunded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams).
- Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities.

Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

EXPOSURE CONTROLS

Source	Material	TWA mg/m ³	Notes
Australia Exposure Standards	Moisture Trap (Emery (dust) (a))	10	(see Chapter 14)
Australia Exposure Standards	Moisture Trap (Aluminium (welding fumes) (as Al))	5	
Australia Exposure Standards	Moisture Trap (Aluminium (metal dust))	10	
Australia Exposure Standards	Moisture Trap (Silica - Amorphous Fumed silica (respirable dust))	2	(see Chapter 14)
Australia Exposure Standards	calcium oxide (Calcium oxide)	2	
Australia Exposure Standards	silica crystalline - quartz (Silica - Crystalline Quartz)	0.1	(see Chapter 14)

The following materials had no OELs on our records

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

EMERGENCY EXPOSURE LIMITS

Material	Revised	IDLH
silica amorphous 10451	3, 000	
calcium oxide 1910	25	
silica crystalline - quartz 21686	50	

MATERIAL DATA

MOISTURE TRAP:

SILICA AMORPHOUS:

SILICA CRYSTALLINE - QUARTZ:

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

MOISTURE TRAP:

SILICA CRYSTALLINE - QUARTZ:

- Because the margin of safety of the quartz TLV is not known with certainty and given the associated link

continued...

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 7 of 21

Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

between silicosis and lung cancer it is recommended that quartz concentrations be maintained as far below the TLV as prudent practices will allow.

MOISTURE 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.

ALUMINIUM OXIDE:

MOISTURE TRAP:

- For aluminium oxide:

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

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

CALCIUM OXIDE:

MOISTURE TRAP:

- For calcium oxide:

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

MOISTURE TRAP:

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

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

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

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

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

For aluminium oxide and pyrophoric grades of aluminium:

Twenty seven year experience with aluminium oxide dust (particle size 96% 1,2 µm) without adverse effects either systemically or on the lung, and at a calculated concentration equivalent to 2 mg/m³ over an 8-hour shift has led 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.

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

continued...

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 8 of 21

Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

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.

SILICA CRYSTALLINE - QUARTZ:

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

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

PERSONAL PROTECTION

EYE

- Chemical goggles.
- Full face shield may be required for supplementary but never for primary protection of eyes
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lens or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent].

HANDS/FEET

■ 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.

Experience indicates that the following polymers are suitable as glove materials for protection against undissolved, dry solids, where abrasive particles are not present.

- polychloroprene
- nitrile rubber
- butyl rubber
- fluorocacoutchouc
- polyvinyl chloride

Gloves should be examined for wear and/ or degradation constantly.

OTHER

- Overalls.
- P.V.C. apron.
- Barrier cream.
- Skin cleansing cream.
- Eye wash unit.

continued...

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 9 of 21

Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

RESPIRATOR

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

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

Use respirators with protection factors appropriate for the exposure level.

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

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

ENGINEERING CONTROLS

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

The basic types of engineering controls are:

Process controls which involve changing the way a job activity or process is done to reduce the risk.

Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.

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

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.

An approved self contained breathing apparatus (SCBA) may be required in some situations.

Provide adequate ventilation in warehouse or closed storage area.

Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

APPEARANCE

Light grey solid with no odour; does not mix with water.

PHYSICAL PROPERTIES

Does not mix with water.

Floats on water.

Alkaline.

continued...

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 10 of 21

Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

State	Divided Solid	Molecular Weight	Not Applicable
Melting Range (°C)	Not Available	Viscosity	Not Applicable
Boiling Range (°C)	Not Available	Solubility in water (g/L)	Immiscible
Flash Point (°C)	Not Applicable	pH (1% solution)	Not Applicable
Decomposition Temp (°C)	Not Available	pH (as supplied)	Not Applicable
Autoignition Temp (°C)	Not Applicable	Vapour Pressure (kPa)	Not Applicable
Upper Explosive Limit (%)	Not Applicable	Specific Gravity (water=1)	0.8
Lower Explosive Limit (%)	Not Applicable	Relative Vapour Density (air=1)	Not Available
Volatile Component (%vol)	Not Applicable	Evaporation Rate	Not Available

Section 10 - STABILITY AND REACTIVITY

CONDITIONS CONTRIBUTING TO INSTABILITY

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

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

Section 11 - TOXICOLOGICAL INFORMATION

POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

SWALLOWED

■ The material can produce chemical burns within the oral cavity and gastrointestinal tract following ingestion.

Accidental ingestion of the material may be damaging to the health of the individual.

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

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

EYE

■ The material can produce chemical burns to the eye following direct contact. Vapours or mists may be extremely irritating.

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

SKIN

■ The material can produce chemical burns following direct contact with the skin.

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

Irritation caused by calcium oxide is a result of local liberation of heat and dehydration of tissues which occurs on "slaking" of the small size particles and the resulting alkalinity of the slaked product.

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

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.

INHALED

■ Inhalation of dusts, generated by the material during the course of normal handling, may be damaging to the health of the individual.

Inhalation of calcium oxide may produce inflammation of the respiratory passages and ulceration and

continued...

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 11 of 21

Section 11 - TOXICOLOGICAL INFORMATION

perforation of the septum.

Persons with impaired respiratory function, airway diseases and conditions such as emphysema or chronic bronchitis, may incur further disability if excessive concentrations of particulate are inhaled.

If prior damage to the circulatory or nervous systems has occurred or if kidney damage has been sustained, proper screenings should be conducted on individuals who may be exposed to further risk if handling and use of the material result

in excessive exposures.

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

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

■ Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis.

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.

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.

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

When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C.

The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis(aluminosis) in experimental animals, but only when given by the intra-tracheal route.

The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity.

Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction.

There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveoli (sub 5 um) are able

continued...

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 12 of 21

Section 11 - TOXICOLOGICAL INFORMATION

to produce pathogenic effects in the lungs.

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

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

After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus. Aluminium may persist for a very long time 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-

continued...

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 13 of 21

Section 11 - TOXICOLOGICAL INFORMATION

like aggregates form but soon degrade. Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA).

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

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.

continued...

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 14 of 21

Section 11 - TOXICOLOGICAL INFORMATION

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.

TOXICITY AND IRRITATION

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

CALCIUM OXIDE:

SODIUM MONOXIDE:

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

ALUMINIUM OXIDE:

SODIUM MONOXIDE:

■ No significant acute toxicological data identified in literature search.

MOISTURE TRAP:

■ Not available. Refer to individual constituents.

SILICA AMORPHOUS:

TOXICITY

Oral (rat) LD50: 3160 mg/kg

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

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

■ For silica amorphous:

When experimental animals inhale synthetic amorphous silica (SAS) dust, it dissolves in the lung fluid and is rapidly eliminated. If swallowed, the vast majority of SAS is excreted in the faeces and there is little accumulation in the body. Following absorption across the gut, SAS is eliminated via urine without modification in animals and humans. SAS is not expected to be broken down (metabolised) in mammals. After ingestion, there is limited accumulation of SAS in body tissues and rapid elimination occurs. Intestinal absorption has not been calculated, but appears to be insignificant in animals and humans. SASs injected subcutaneously are subjected to rapid dissolution and removal. There is no indication of metabolism of SAS in animals or humans based on chemical structure and available data. In contrast to crystalline silica, SAS is soluble in physiological media and the soluble chemical species that are formed are eliminated via the urinary tract without modification.

Both the mammalian and environmental toxicology of SASs are significantly influenced by the physical and chemical properties, particularly those of solubility and particle size. SAS has no acute intrinsic toxicity by inhalation. Adverse effects, including suffocation, that have been reported were caused by the presence of high numbers of respirable particles generated to meet the required test atmosphere. These results are not representative of exposure to commercial SASs and should not be used for human risk assessment. Though repeated exposure of the skin may cause dryness and cracking, SAS is not a skin or eye irritant, and it is not a sensitiser.

Repeated-dose and chronic toxicity studies confirm the absence of toxicity when SAS is swallowed or upon skin

continued...

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 15 of 21

Section 11 - TOXICOLOGICAL INFORMATION

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:

TOXICITY

Inhalation (human) LCLo: 0.3 mg/m³/10Y

Inhalation (human) TCLo: 16 mppcf*/8H/17.9Y

Inhalation (rat) TCLo: 50 mg/m³/6H/71W

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

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

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

IRRITATION

Nil Reported

continued...

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 16 of 21

Section 11 - TOXICOLOGICAL INFORMATION

* 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
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 $[\text{Si}(\text{OH})_4]$. At pH 9.4 the solubility of amorphous silica is about 120 mg SiO_2/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:

SILICA CRYSTALLINE - QUARTZ:

SODIUM MONOXIDE:

■ 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.

continued...

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 17 of 21

Section 12 - ECOLOGICAL INFORMATION

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.

SODIUM MONOXIDE:

ALUMINIUM OXIDE:

CALCIUM OXIDE:

SILICA CRYSTALLINE - QUARTZ:

SILICA AMORPHOUS:

■ DO NOT discharge into sewer or waterways.

SILICA AMORPHOUS:

mor

SODIUM MONOXIDE:

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

ALUMINIUM OXIDE:

■ For aluminium and its compounds and salts:

Environmental fate:

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

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

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

The trivalent aluminum ion is surrounded by six water molecules in solution. The hydrated aluminum ion, $[Al(H_2O)_6]^{3+}$, undergoes hydrolysis, in which a stepwise deprotonation of the coordinated water ligands forms bound hydroxide ligands (e.g., $[Al(H_2O)_5(OH)]^{2+}$, $[Al(H_2O)_4(OH)_2]^+$). The speciation of aluminum in water is pH dependent. The hydrated trivalent aluminum ion is the predominant form at pH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are $Al(OH)_2^+$ and $Al(OH)_3$, 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 aluminum 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.

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

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

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

continued...

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 18 of 21

Section 12 - ECOLOGICAL INFORMATION

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

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

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

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

Ecotoxicity:

Freshwater species pH >6.5

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

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

Crustaceans LC50 (48 h): 1 sp 2.3-36 9 mg/L; Chronic NOEC (7-28 d) 3 spp, 0.136-1.72 mg/L

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

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

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

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

Alga: 1 sp NOEC growth 2.0 mg/L

Among freshwater aquatic plants, single-celled plants are generally the most sensitive to 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 ($\text{Al}(\text{OH})_2^+$) 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 amphoteric 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.)

continued...

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 19 of 21

Section 12 - ECOLOGICAL INFORMATION

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.

Ecotoxicity

Ingredient	Persistence: Water/Soil	Persistence: Air	Bioaccumulation	Mobility
silica amorphous	HIGH	No Data Available	LOW	HIGH
sodium monoxide	No Data Available	No Data Available		
aluminium oxide	No Data Available	No Data Available		
calcium oxide	No Data Available	No Data Available	LOW	
silica crystalline - quartz	No Data Available	No Data Available		

Section 13 - DISPOSAL CONSIDERATIONS

■ 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 Management Authority for disposal.
- Bury residue in an authorised landfill.
- Recycle containers if possible, or dispose of in an authorised landfill.

Section 14 - TRANSPORTATION INFORMATION

HAZCHEM:

None (ADG7)

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

continued...

MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 20 of 21

Section 14 - TRANSPORTATION INFORMATION

Section 15 - REGULATORY INFORMATION

POISONS SCHEDULE None

REGULATIONS

Regulations for ingredients

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

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

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"

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

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

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

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

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

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

No data for Moisture Trap (CW: 28-0932)

Section 16 - OTHER INFORMATION

INGREDIENTS WITH MULTIPLE CAS NUMBERS

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

MSDS SECTION CHANGES

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

Section Name	Version	Date	Section Name	Version	Date	Section Name	Version	Date
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MOISTURE TRAP

Chemwatch Independent Material Safety Data Sheet (REVIEW)

Issue Date: 27-Feb-2012

A317L

CHEMWATCH 28-0932

Version No:3

CD 2012/1 Page 21 of 21

Section 16 - OTHER INFORMATION

Environmental 2 24- Oct- 2011

■ 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.