Antas Kitchen and Bathroom Silicone Sealant

Antas Sealants and Adhesives

Part Number: **66188**
Version No: **1.2.8.7**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **30/06/2021**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	Antas Kitchen and Bathroom Silicone Sealant
Chemical Name	Not Applicable
Synonyms	AT-KnB
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	waterproof sealing for kitchens, bathrooms and other wet areas.

Details of the supplier of the safety data sheet

Registered company name	**Antas Sealants and Adhesives**
Address	21-23 Pavesi Street Smithfield NSW 2164 Australia
Telephone	+61 2 9157 0368
Fax	Not Available
Website	www.antas.com.au
Email	info@antas.com.au

Emergency telephone number

Association / Organisation	**Not Available**
Emergency telephone numbers	**+61 2 9157 0368**
Other emergency telephone numbers	**National Poison Information Centre 13 11 26**

SECTION 2 Hazards identification

Classification of the substance or mixture

HAZARDOUS CHEMICAL. NON-DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

Poisons Schedule	Not Applicable
Classification [1]	Eye Irritation Category 2A, Acute Aquatic Hazard Category 3, Skin Corrosion/Irritation Category 2, Skin Sensitizer Category 1, Reproductive Toxicity Category 1B, Emit Flammable Gases with Water Category 2, Chronic Aquatic Hazard Category 3
Legend:	*1. Classification by vendor; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

Hazard pictogram(s)	

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Signal word	**Danger**

Hazard statement(s)

H319	Causes serious eye irritation.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H360Fd	May damage fertility. May damage the unborn child.
H261	In contact with water releases flammable gases.
H412	Harmful to aquatic life with long lasting effects.

Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P231+P232	Handle and store contents under inert gas. Protect from moisture.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P223	Do not allow contact with water.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P302+P335+P334	IF ON SKIN: Brush off loose particles from skin. Immerse in cool water [or wrap in wet bandages].
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P370+P378	In case of fire: Use water spray/fog to extinguish.
P302+P352	IF ON SKIN: Wash with plenty of water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

P405	Store locked up.
P402+P404	Store in a dry place. Store in a closed container.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
68611-44-9	10-20	silica amorphous, fumed
1185-55-3	<10	methyltrimethoxysilane
919-30-2	<2	3-aminopropyltriethoxysilane
27858-32-8	<5	titanium(IV) bis(ethyl acetoacetato)diisopropoxide
58-36-6	<1	oxybisphenoxarsine

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Legend:	*1. Classification by vendor; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available*

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Wash out immediately with fresh 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. ▸ 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.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary.
Ingestion	▸ Immediately give a glass of water. ▸ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

Indication of any immediate medical attention and special treatment needed

For acute or short term repeated exposures to arsenic, soluble compounds: Treat as per arsenic poisoning.

- Acute skin lesions such as contact dermatitis usually do not require other treatment than removal from exposure.
- If more severe symptoms of the respiratory system, the skin or the gastro-intestinal tract occur, British Anti-Lewisite (BAL, dimercaprol) may be given. Prompt administration in such cases is vital; to obtain maximum benefit such treatment should be administered within 4 hours of poisoning.
- In addition, general treatment such as prevention of further absorption from the gastro-intestinal tract are mandatory.
- General supportive therapy such as maintenance of respiration and circulation, maintenance of water and electrolyte balance and control of nervous system effects, as well as elimination of absorbed poison through dialysis and exchange transfusion, may be used if feasible.
- Dimercaprol is given by deep intramuscular injection as a 5% solution in peanut oil (or a 10% solution with benzyl-benzoate in vegetable oil). It is usually given in a dose of 3 mg/kg, 4-hourly, for the first two days, or twice daily for up to seven days. [ILO Encyclopedia]
- BAL Therapy is effective for haematological manifestations of chronic arsenic poisoning but not for neurological symptoms. Watch for side effects (e.g. urticaria, burning sensation in the lips, mouth and throat, fever, conjunctivitis etc).
- Some relief results from administration of diphenhydramine (Benadryl) (1.5 mg/kg intramuscularly or by mouth every 6 hour). [Ellenhorn and Barceloux: Medical Toxicology]

BIOLOGICAL EXPOSURE INDEX - BEI (Notice of Intent to Establish)

BEIs represent the levels of determinants which are most likely to be observed in specimens collected from a healthy worker who has been exposed to chemicals to the same extent as a worker with inhalation exposure to the

Exposure Standard (ES or TLV):

Determinant	Index	Sampling Time	Comments
Inorganic arsenic metabolites in urine	35 ug/gm creatinine	End of workweek	B

B: Background levels occur in specimens collected from subjects **NOT** exposed Consult specific documentation.

* Preplacement and periodic medical examinations are essential for workers exposed to arsenic on a regular basis. Preplacement physical examinations should give particular attention to allergic and chronic skin lesions, eye disease, psoriasis, chronic eczematous dermatitis, hyperpigmentation of the skin, keratosis and warts, baseline weight, baseline blood and haemoglobin counts, baseline urinary arsenic determinations.
Annual physical examinations should give attention to general health, weight, skin condition, and any evidence of excessive exposure or absorption of arsenic
For acute and short term repeated exposures to methanol:

- Toxicity results from accumulation of formaldehyde/formic acid.
- Clinical signs are usually limited to CNS, eyes and GI tract Severe metabolic acidosis may produce dyspnea and profound systemic effects which may become intractable. All symptomatic patients should have arterial pH measured. Evaluate airway, breathing and circulation.
- Stabilise obtunded patients by giving naloxone, glucose and thiamine.
- Decontaminate with Ipecac or lavage for patients presenting 2 hours post-ingestion. Charcoal does not absorb well; the usefulness of cathartic is not established.
- Forced diuresis is not effective; haemodialysis is recommended where peak methanol levels exceed 50 mg/dL (this correlates with serum bicarbonate levels below 18 meq/L).
- Ethanol, maintained at levels between 100 and 150 mg/dL, inhibits formation of toxic metabolites and may be indicated when peak methanol levels exceed 20 mg/dL. An intravenous solution of ethanol in D5W is optimal.
- Folate, as leucovorin, may increase the oxidative removal of formic acid. 4-methylpyrazole may be an effective adjunct in the treatment. 8.Phenytoin may be preferable to diazepam for controlling seizure.

[Ellenhorn Barceloux: Medical Toxicology]

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BIOLOGICAL EXPOSURE INDEX - BEI

Determinant	Index	Sampling Time	Comment
1. Methanol in urine	15 mg/l	End of shift	B, NS
2. Formic acid in urine	80 mg/gm creatinine	Before the shift at end of workweek	B, NS

B: Background levels occur in specimens collected from subjects **NOT** exposed.

NS: Non-specific determinant - observed following exposure to other materials.

SECTION 5 Firefighting measures

Extinguishing media

- Foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.
- Water spray or fog - Large fires only.

Special hazards arising from the substrate or mixture

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

Advice for firefighters

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. ‣ Prevent, by any means available, spillage from entering drains or water courses. ‣ Use water delivered as a fine spray to control fire and cool adjacent 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	‣ 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. Combustible. Will burn if ignited. Combustion products include: , carbon monoxide (CO) , carbon dioxide (CO2) , silicon dioxide (SiO2) , arsenic compounds , other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	‣ Clean up all spills immediately.

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	‣ Avoid contact with skin and eyes. ‣ Wear impervious gloves and safety goggles. ‣ Trowel up/scrape up. ‣ Place spilled material in clean, dry, sealed container. ‣ Flush spill area with water.
Major Spills	Minor hazard. ‣ Clear area of personnel. ‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Control personal contact with the substance, by using protective equipment as required. ‣ Prevent spillage from entering drains or water ways. ‣ Contain spill with sand, earth or vermiculite. ‣ Collect recoverable product into labelled containers for recycling. ‣ Absorb remaining product with sand, earth or vermiculite and place in appropriate containers for disposal. ‣ Wash area and prevent runoff into drains or waterways. ‣ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

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

Conditions for safe storage, including any incompatibilities

Suitable container	‣ Metal can or drum ‣ Packaging as recommended by manufacturer. ‣ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	The substance may be or contains a "metalloid" The following elements are considered to be metalloids; boron,silicon, germanium, arsenic, antimony, tellurium and (possibly) polonium The electronegativities and ionisation energies of the metalloids are between those of the metals and nonmetals, so the metalloids exhibit characteristics of both classes. The reactivity of the metalloids depends on the element with which they are reacting. For example, boron acts as a nonmetal when reacting with sodium yet as a metal when reacting with fluorine. Unlike most metals, most metalloids are amphoteric- that is they can act as both an acid and a base. For instance, arsenic forms not only salts such as arsenic halides, by the reaction with certain strong acid, but it also forms arsenites by reactions with strong bases. Most metalloids have a multiplicity of oxidation states or valences. For instance, tellurium has the oxidation states +2, -2, +4, and +6. Metalloids react like non-metals when they react with metals and act like metals when they react with non-metals. ‣ Contact with water liberates highly flammable gases Silicas: ‣ react with hydrofluoric acid to produce silicon tetrafluoride gas

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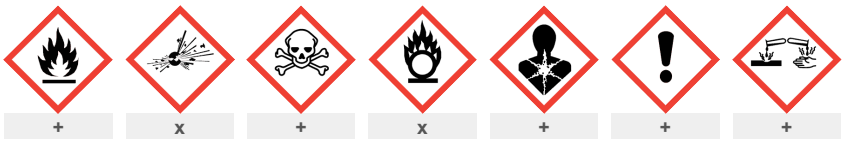
- react with xenon hexafluoride to produce explosive xenon trioxide
- reacts exothermically with oxygen difluoride, and explosively with chlorine trifluoride (these halogenated materials are not commonplace industrial materials) and other fluorine-containing compounds
- may react with fluorine, chlorates
- 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.
- Arsine, an extremely poisonous (lethal) gas with a garlic odour can be generated when the material reacts with acids, alkalis or water in the presence of an active metal (zinc, aluminium, magnesium, sodium, iron etc.).
- Avoid reaction with oxidising agents



+	x	+	x	+	+	+

X — *Must not be stored together*
0 — *May be stored together with specific preventions*
+ — *May be stored together*

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
silica amorphous, fumed	18 mg/m3	100 mg/m3	630 mg/m3
methyltrimethoxysilane	38 mg/m3	410 mg/m3	2,500 mg/m3
3-aminopropyltriethoxysilane	1.9 mg/m3	21 mg/m3	350 mg/m3
titanium(IV) bis(ethyl acetoacetato)diisopropoxide	30 mg/m3	330 mg/m3	2,000 mg/m3
oxybisphenoxarsine	5 mg/m3	14 mg/m3	84 mg/m3

Ingredient	Original IDLH	Revised IDLH
silica amorphous, fumed	Not Available	Not Available
methyltrimethoxysilane	Not Available	Not Available
3-aminopropyltriethoxysilane	Not Available	Not Available
titanium(IV) bis(ethyl acetoacetato)diisopropoxide	Not Available	Not Available
oxybisphenoxarsine	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
methyltrimethoxysilane	E	≤ 0.1 ppm
3-aminopropyltriethoxysilane	E	≤ 0.1 ppm
titanium(IV) bis(ethyl acetoacetato)diisopropoxide	E	≤ 0.1 ppm
oxybisphenoxarsine	E	≤ 0.01 mg/m³
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

Use control measures / protective gear to avoid any personal contact. The ES-TWA is based solely on the prevention of systemic effects due to inhalation and is not protective against the substantial risk of cancer produced by exposure to inorganic arsenic. Some jurisdictions require health surveillance be performed on

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occupationally exposed workers.

Such surveillance should emphasise

- demography, occupational and medical history and health advice
- physical examination with emphasis on the peripheral nervous system and skin
- urinary total arsenic
- records of personal exposure

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 um (+-) 0.3 um and with a geometric standard deviation of 1.5 um (+-) 0.1 um, i.e..generally less than 5 um.
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.

Exposure controls

Appropriate engineering controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.
An approved self contained breathing apparatus (SCBA) may be required in some situations.
Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)

Within each range the appropriate value depends on:

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

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

Personal protection

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Eye and face protection	‣ 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 lenses 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]
Skin protection	See Hand protection below
Hands/feet protection	‣ Wear chemical protective gloves, e.g. PVC. ‣ Wear safety footwear or safety gumboots, e.g. Rubber **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.
Body protection	See Other protection below
Other protection	‣ Protective overalls, closely fitted at neck and wrist. ‣ Eye-wash unit. **IN CONFINED SPACES:** ‣ Non-sparking protective boots ‣ Static-free clothing. ‣ Ensure availability of lifeline. Staff should be trained in all aspects of rescue work. Rescue gear: Two sets of SCBA breathing apparatus Rescue Harness, lines etc.

Respiratory protection

Particulate. (AS/NZS 1716 & 1715, EN 143:2000 & 149:001, ANSI Z88 or national equivalent)

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	P1 Air-line*	- -	PAPR-P1 -
up to 50 x ES	Air-line**	P2	PAPR-P2
up to 100 x ES	-	P3	-
		Air-line*	-
100+ x ES	-	Air-line**	PAPR-P3

* - Negative pressure demand ** - Continuous flow
A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Not Available

Physical state	Non Slump Paste	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature**	Not Available

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Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Not Available	**pH as a solution (%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	Product is considered stable and hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting. Inhalation of arsenic containing materials resemble those described for ingestion but in addition may produce severe nose and respiratory tract irritation. Acute inhalation exposure can cause cough (with foamy sputum an rales), chest pain, shortness of breath, dizziness, headache, pulmonary oedema, extreme general weakness and cyanosis. High exposures can cause poor appetite, nausea, vomiting and muscle cramps. Heart effects with abnormal EKG can also occur with very high exposures Prolonged or repeated exposures may produce necrosis and perforation of the nasal septum.
Ingestion	Symptoms of acute poisoning by arsenic ingestion, which develop within 4 hours include epigastric pain, vomiting and watery diarrhoea, nausea and vomiting. Blood may appear in vomitus and stools. If amount ingested is sufficiently high, shock, rapid pulse and coma may develop, followed by death within 24 hours. Severe gastritis or gastroenteritis may occur as a result of lesions produced by vascular damage from absorbed arsenic (and not local corrosion); symptoms may be delayed for several hours. Eventually a violent haemorrhagic gastroenteritis leads to profound loss of fluid and electrolyte resulting in shock and death. Occasionally alimentary symptoms are mild or absent in which case symptoms are usually referable to the central nervous system, headache, vertigo, muscle spasm or convulsion, delirium and, sometimes, mania. In advanced poisonings by arsenic and its inorganic salts, nervous symptoms are prominent; disorders of the brain (encephalopathies) and peripheral neuritis (more commonly) have been described. A prickling sensation (paresthesia), decreased sensitivity to sensation and pain (hypoesthesia), eventually paralysis and muscular atrophy appear, usually in the legs. "Glove and stocking' distribution of sensory loss may be prominent. The toxic moiety is presumed to be trivalent arsenic in the form of inorganic arsenious acid (arsenite) or an organic arsenoxide. Arsenites are active enzyme inhibitors. Arsenic and its compounds may damage the stem cell which acts as the precursor to components of the blood. Loss of the stem cell may result in pancytopenia (a reduction in the number of red and white blood cells and platelets) with a latency period corresponding to the lifetime of the individual blood cells. Granulocytopenia (a reduction in granular leukocytes) develops within days and thrombocytopenia (a disorder involving platelets), within 1-2 weeks, whilst loss of erythrocytes (red blood cells) need months to become clinically manifest. Aplastic anaemia develops due to complete destruction of the stem cells. The material has **NOT** been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.

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Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions. Arsenic and its compounds may irritate the skin. Certain individuals may develop sensitisation dermatitis characterised by eczema with scaling and hyperpigmentation of the skin and hyperkeratosis of the palms of the hands and soles of the feet. Skin contact may cause erythema (an abnormal redness caused by capillary congestion), with burning, itching, swelling and skin eruptions. Repeated skin contact can cause thickened skin and/or patchy areas of darkening and loss of pigment. Some persons develop white lines on the nails. 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.
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. Repeated 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.
Chronic	On the basis, primarily, of animal experiments, concern has been expressed 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. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: - clear evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects. Symptoms of chronic arsenic poisoning, by inhalation, include weight loss, nausea and diarrhoea alternating with constipation, pigmentation and eruption of the skin, loss of hair, peripheral neuritis, blood disorders (anaemia), striations on fingernails and toenails. Long-term exposure can cause an ulcer or hole in the "bone'"dividing the inner nose. Hoarseness and sore eyes also occur. anaemia, liver and kidney injury, skin and lung cancer. Renal damage may develop after acute or chronic exposures. Prolonged inhalation of arsenical dusts are implicated in lung cancer. Subacute and chronic exposure to arsenic and its organic salts may produce anorexia, mild gastrointestinal disturbances (nausea and vomiting), low grade fever, persistent headache, pallor, weakness, catarrhal inflammation and neuropathies with severe crippling effects.. Stomatitis (oral lesions) and salivation are common. High or repeated exposure can cause nerve damage with "pins and needles'" burning, numbness, and later weakness (incoordination) of arms and legs, anaemia, liver and kidney injury, skin and lung cancer. Repeated exposure can also damage the liver, causing narrowing of the blood vessels, or interfere with bone marrow's ability to make red blood cells. Skin afflictions are many and varied; erythema, eczema, pigmentation (arsenic melanosis), diffuse alopecia, keratosis (especially of the palms and soles), scaling and desquamation, brittle nails, white lines or bands on the nails (Mees lines), loss of hair and nails and localised subcutaneous oedema (especially of the eye-lids). Signs of renal damage may also develop. Liver enlargement (hepatomegaly) with jaundice (and sometimes pruritus) may develop into cirrhosis with accumulation of body fluids in the abdominal cavity (ascites). Nervous system effects involving the extremities (numbness, tingling, burning pain, weakness, incoordination) may also occur. Arsenic exposure is linked with increase in diseases including ischaemia, cerebrovascular disease and carotid atherosclerosis. Several cytokines and growth factors involved in inflammation, have been identified in humans after prolonged exposure; these may heighten the risk of atherosclerosis (1). Individual variability in arsenic metabolism may determine susceptibility to arsenic disease. Genetic polymorphism (variability) for enzymes instrumental in arsenic metabolism (purine nucleoside phosphorylase and glutathione S-transferase omega 1-1) has been identified amongst individuals of European and Mexican Hispanic

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(indigenous) ancestry (2).
Many cases of skin cancer have been reported among people exposed to arsenic through medical treatment with inorganic trivalent arsenic compounds. In some instances skin cancers have occurred in combination with other cancers, such as liver angiosarcoma, intestinal and urinary bladder carcinomas and meningioma. Epidemiological studies of cancer after medical treatment have shown an excess of skin cancers but no clear association with other cancers has been shown.
Prolonged inhalation of arsenical dusts are implicated in lung cancer. Occupational exposure to inorganic arsenic, especially in mining and copper smelting, has consistently been associated with an increased risk of cancer. An almost tenfold increase in the incidence of lung cancer was found in workers most heavily exposed to arsenic and relatively clear dose-response relationships have been obtained with regard to cumulative
exposure. Other smelter worker population's have been shown to have consistent increases in lung cancer incidence, as well as increases of about 20% in the incidence of gastrointestinal cancer and of 30% for renal cancer and haematolymphatic malignancies
An association between environmental exposure to arsenic through drinking water and skin cancer has been observed and confirmed. Epidemiological studies in areas where drinking water contained 0.35-1.14 mg/l arsenic elevated risks for cancers of the bladder, kidney, skin, liver, lung and colon in both men and women. Taiwanese exposed to naturally occurring arsenic in well-water have experienced skin cancer. Inorganic and organic arsenics are established carcinogens in man. Chronic human exposure to non-overtly toxic doses of arsenic is associated with carcinogenicity, although the mechanism is not fully understood. Arsenic does not directly cause DNA damage or mutations. Data indicates that nontoxic doses of arsenite can interact with glucocorticoid receptor (GR) complexes and selectively inhibit GR-mediated transcription which is associated with altered nuclear function (3).
(1) Wu et al; Environmental Health Perspectives, 111, 1429-1438, 2003
(2) Yu et al; Environmental Health Perspectives, 111, 1421-1427, 2003
(3) Kaltreider et al; Environmental Health Perspectives, 109, pp 245-251, 2001
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
Repeated exposure to synthetic amorphous silicas may produce skin dryness and cracking.
Available data confirm the absence of significant toxicity by oral and dermal routes of exposure.
Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted 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. Differences in values may be due to particle size, and therefore the number of particles administered per unit dose. Generally, as particle size diminishes so does the NOAEL/ LOAEL. Exposure produced transient increases in lung inflammation, markers of cell injury and lung collagen content. There was no evidence of interstitial pulmonary fibrosis.

Antas Kitchen and Bathroom Silicone Sealant	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

silica amorphous, fumed	**TOXICITY**	**IRRITATION**
	Inhalation(Rat) LC50; 0.45 mg/L4h[2]	Not Available
	Oral(Rat) LD50; >5000 mg/kg[2]	

methyltrimethoxysilane	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >9500 mg/kg[1]	Eye (rabbit): 500 mg/24h - mild
	Inhalation(Rat) LC50; >26000 ppm4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral(Mouse) LD50; 7000 mg/kg[1]	Skin (rabbit): 500 mg open - mild
		Skin: no adverse effect observed (not irritating)[1]

3-aminopropyltriethoxysilane	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >5700 mg/kg[1]	Eye (rabbit): 0.75 mg/24h-SEVERE
	Inhalation(Rat) LC50; >7.35 mg/l4h[1]	Eye (rabbit): 100 mg - mild
	Oral(Mouse) LD50; 260 mg/kg[2]	Skin (rabbit): 0.1 mg - mild
		Skin (rabbit): 5.0 mg/24h-SEVERE

titanium(IV) bis(ethyl acetoacetato)diisopropoxide	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 12870 mg/kg[1]	Not Available
	Oral(Rat) LD50; 3980 mg/kg[1]	

oxybisphenoxarsine	**TOXICITY**	**IRRITATION**
	Oral(Guinea) LD50; 24 mg/kg[2]	Skin (g.pig): 250 mg/5d SEVERE

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Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*

SILICA AMORPHOUS, FUMED	For silane, dichlorodimethyl-, reaction products with silica Acute oral toxicity is very low for treated silica. Acute inhalation toxicity was only tested for inhalable particles and is not relevant for the material used industrially. Changes in respiratory organs (inflammatory processes) after repeated exposure were reversible in animals that survived the exposure and were observed above the valid TLV values, only. If TLV values are maintained no health hazards are expected. Repeated dose toxicity is sufficiently investigated. Treated silica is not mutagenic. The NOAEL for repro/developmental toxicity is 500 mg/kg bw. **Acute toxicity:** In a limit test giving 10% in the diet (5000 mg/kg bw) to rats the acute oral LD50 was determined to be higher than 5000 mg/kg bw. In another study administering single doses of 2500 and 5000 mg/kg bw to rats the LD50 was also concluded to be higher than 5000 mg/kg bw. In an acute oral toxicity study giving still higher single doses in olive oil the LD50 appeared to be above 7900 mg/kg bw. No signs of toxicity were observed in any of these studies. All inhalation testing has been conducted with a substance that differs significantly from the commercial product based on particle size. In these animal tests the experimental design caused the particle size to be reduced resulting in nearly 100% of the particle fraction being below 10 um and capable of entering the deep lung (alveolar particle fraction). The alveolar fraction is responsible for the toxicological effects (suffocation; overloading of the lung due to poor dust clearance mechanisms) which were observed with LC50 values of > 477, 450, 520-1120, and >2280 mg/m3 and corresponding mass median aerodynamic diameters (MMAD) of 2.9 um, 1.24 um, 0.8 – 0.9 um and 0.15 um, respectively. In comparison to the particle size used in these acute inhalation animal tests, only minor amounts (less than 1 %) of the commercially available commercial substance have been measured as respirable (alveolar fraction < 10 um MMAD) using test method EN/DIN 481 (ref.35). Using the same method > 99% of the particle fraction is in excess of 90 um and can only reach the upper airways (nasal passages and throat) or cannot be inhaled at all. Therefore the tests do not represent the toxicological behavior of the commercial product and are not considered relevant for inclusion in the hazard definition/hazard assessment of the commercial substance. **Genetic toxicity:** The test substance was not mutagenic in the Bacterial Reverse Mutation Assay (Ames test) with Salmonella *typhimurium* TA98, TA100, TA1535, TA1537 and TA1538 strains and with *E. coli* WP2 uvrA strain. Also an in vitro chromosomal aberration study in CHO cells gave negative results. **Repeat dose toxicity:** A 24-month oral feeding study administering a 100 mg/kg dose to 20 male and 20 female rats resulted in a NOAEL of 100 mg/kg. No clinical signs or treatment-related changes (e.g. bodyweight) were observed. There were no carcinogenic effects.A 6-month oral feeding study showed no treatment-related effects at the given dose of 500 mg/kg bw to rats (40/sex) resulting in a NOAEL of 500 mg/kg bw; a slight progressive – but reversible -transformation of the adrenal cortex in females was attributed to chronic stress. Another oral feeding study (5-8 weeks) exposed rats (5/sex/treatment) to a dose of 500, 1000 or 2000 mg/kg bw initially and increasing these doses gradually to 4000, 8000 and 16000 mg/kg bw, respectively. Decrease in body weight and food consumption combined with apathy and decreased grooming activity and decreased cytoplasmic glycogen in hepatocytes may indicate a starving condition of these animals. At the highest dose group four animals died. The NOAEL was determined to be 500 mg/kg bw (LOAEL = 1000 mg/kg bw). In a limited reported study where a dose of 500 or 1000 mg/kg bw was administered by gavage to 30 rats no treatment-related effects could be found, resulting in a NOAEL of 1000 mg/kg bw. A 13-week inhalation study exposing 70 animals/sex to 35 mg/m3 resulted in granuloma-like lesions of the lungs, accumulations of alveolar macrophages, alveolar spaces filled with granular material, debris and polymorphonuclear leucocytes, alveolar bronchiolisation, interstitial fibrosis and enlarged mediastinal lymph nodes. In a 2-week study administering 0, 31, 87 or 420 mg/m3 to a total number of 40 rats/sex 4 males and 2 females died at the top dose level. The rats at the top dose level showed severe respiratory distress and apathy. A dose-related decrease in body weight was observed at 87 mg/m3 and higher. The lungs showed similar effects as those observed in the 13-week inhalation study. A 3-day study and an 8-12-month study both with a concentration of 50 mg/m3 to rats yielded similar results to the above studies in the lungs and the size of the particles was determined to be smaller than 7 µm. Changes in respiratory organs (inflammatory processes) observed in inhalative repeated dose toxicity testing were reversible in animals that survived the exposure. There was no indication of silicosis. Concentrations of the substances with toxicological effects in inhalative toxicity testing were above the valid TLV values (10mg/m3 USA). If TLV values are maintained no health hazards are expected. **Reproductive and developmental toxicity:** Two studies are included on repro/developmental toxicity. A 6-month, 1-generation study in rats combining fertility and prenatal toxicity testing administered 500 mg/kg bw in the food to 10 females and 2 males. No treatment-related effects were observed in the parents or in the offspring. Therefore the NOAEL for parents and offspring was 500 mg/kg. No effects on the female/male gonads were observed. In a 2-generation reproduction study 20 male and 20 female rats were given 100 mg/kg bw via oral feed for 24 months (see also repeated dose). No abnormalities were observed in the offspring resulting in a NOAEL of 100 mg/kg bw.
METHYLTRIMETHOXYSILANE	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
3-AMINOPROPYLTRIETHOXYSILANE	For alkoxysilanes: Low molecular weight alkoxysilanes (including alkyl orthosilicates) are a known concern for lung toxicity, due to inhalation of vapours or aerosols causing irreversible lung damage at low doses. Alkoxysilane groups that rapidly hydrolyse when in contact with water, result in metabolites that may only cause mild skin irritation. Although there appears to be signs of irritation under different test conditions, based on the available information, the alkoxysilanes cannot be readily classified as a skin irritant.

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The trimethoxysilane group of chemicals have previously been associated with occupational eye irritation in exposed workers who experienced severe inflammation of the cornea . Based on the collective information, these substances are likely to be severe irritants to the eyes.
Methoxysilanes are generally reported to possess higher reactivity and toxicity compared to ethoxysilanes; some methoxysilanes appear to be carcinogenic .In the US, alkoxysilanes with alkoxy groups greater than C2 are classified as moderate concern.
Based on available information on methoxysilanes, the possibility that this family causes skin sensitisation cannot be ruled out. Amine-functional methoxysilanes have previously been implicated as a cause of occupational contact dermatitis, often as a result of repeated skin exposure with workers involved in the manufacture or use of the resins containing the chemical during fibreglass production.

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
While it is difficult to generalise about the full range of potential health effects posed by exposure to the many different amine compounds, characterised by those used in the manufacture of polyurethane and polyisocyanurate foams, it is agreed that overexposure to the majority of these materials may cause adverse health effects.

- Many amine-based compounds can induce histamine liberation, which, in turn, can trigger allergic and other physiological effects, including bronchoconstriction or bronchial asthma and rhinitis.
- Systemic symptoms include headache, nausea, faintness, anxiety, a decrease in blood pressure, tachycardia (rapid heartbeat), itching, erythema (reddening of the skin), urticaria (hives), and facial edema (swelling). Systemic effects (those affecting the body) that are related to the pharmacological action of amines are usually transient.

Typically, there are four routes of possible or potential exposure: inhalation, skin contact, eye contact, and ingestion.
Inhalation:
Inhalation of vapors may, depending upon the physical and chemical properties of the specific product and the degree and length of exposure, result in moderate to severe irritation of the tissues of the nose and throat and can irritate the lungs.
Products with higher vapour pressures have a greater potential for higher airborne concentrations. This increases the probability of worker exposure.
Higher concentrations of certain amines can produce severe respiratory irritation, characterised by nasal discharge, coughing, difficulty in breathing, and chest pains.
Chronic exposure via inhalation may cause headache, nausea, vomiting, drowsiness, sore throat, bronchopneumonia, and possible lung damage. Also, repeated and/or prolonged exposure to some amines may result in liver disorders, jaundice, and liver enlargement. Some amines have been shown to cause kidney, blood, and central nervous system disorders in laboratory animal studies.
While most polyurethane amine catalysts are not sensitisers, some certain individuals may also become sensitized to amines and may experience respiratory distress, including asthma-like attacks, whenever they are subsequently exposed to even very small amounts of vapor. Once sensitised, these individuals must avoid any further exposure to amines. Although chronic or repeated inhalation of vapor concentrations below hazardous or recommended exposure limits should not ordinarily affect healthy individuals, chronic overexposure may lead to permanent pulmonary injury, including a reduction in lung function, breathlessness, chronic bronchitis, and immunologic lung disease.
Inhalation hazards are increased when exposure to amine catalysts occurs in situations that produce aerosols, mists, or heated vapors. Such situations include leaks in fitting or transfer lines. Medical conditions generally aggravated by inhalation exposure include asthma, bronchitis, and emphysema.
Skin Contact:
Skin contact with amine catalysts poses a number of concerns. Direct skin contact can cause moderate to severe irritation and injury-i.e., from simple redness and swelling to painful blistering, ulceration, and chemical burns. Repeated or prolonged exposure may also result in severe cumulative dermatitis.
Skin contact with some amines may result in allergic sensitisation. Sensitised persons should avoid all contact with amine catalysts. Systemic effects resulting from the absorption of the amines through skin exposure may include headaches, nausea, faintness, anxiety, decrease in blood pressure, reddening of the skin, hives, and facial swelling. These symptoms may be related to the pharmacological action of the amines, and they are usually transient.
Eye Contact:
Amine catalysts are alkaline in nature and their vapours are irritating to the eyes, even at low concentrations.
Direct contact with the liquid amine may cause severe irritation and tissue injury, and the “burning” may lead to blindness. (Contact with solid products may result in mechanical irritation, pain, and corneal injury.)
Exposed persons may experience excessive tearing, burning, conjunctivitis, and corneal swelling.
The corneal swelling may manifest itself in visual disturbances such as blurred or “foggy” vision with a blue tint (“blue haze”) and sometimes a halo phenomenon around lights. These symptoms are transient and usually disappear when exposure ceases.
Some individuals may experience this effect even when exposed to concentrations below doses that ordinarily cause respiratory irritation.
Ingestion:
The oral toxicity of amine catalysts varies from moderately to very toxic.
Some amines can cause severe irritation, ulceration, or burns of the mouth, throat, esophagus,and gastrointestinal tract.
Material aspirated (due to vomiting) can damage the bronchial tubes and the lungs.
Affected persons also may experience pain in the chest or abdomen, nausea, bleeding of the throat and the gastrointestinal tract, diarrhea, dizziness, drowsiness, thirst, circulatory collapse, coma, and even death.
Polyurethane Amine Catalysts: Guidelines for Safe Handling and Disposal; Technical Bulletin June 2000 Alliance for Polyurethanes Industry
For 3-aminopropyltriethoxsilane (APTES):

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	Acute toxicity: 3-Aminopropyltriethoxysilane (APTES) has been tested for acute toxicity by the oral, dermal, and inhalation routes of exposure. Acute oral LD50s in rats range from 1570 to 3650 mg/kg bw. The dermal LD50 is 4.29 g/kg bw and the 4-hour inhalation LC50 of the hydrolysate is greater than 7.35 mg/L. Six hours of exposure to substantially saturated vapor of APTES did not kill any of the 5 male or female rats (LC50 > 6 hours). The kidney is a target organ for toxicity for oral and dermal exposures. APTES is severely irritating to the skin and eyes. In a Buehler study in guinea pigs, 7/30 animals showed a skin sensitisation response. The hydrolysis products of this material do not elicit a sensitisation response in a guinea pig maximization test. **Repeat dose toxicity:** Repeated inhalation exposure of rats to 147 mg/m3 of APTES hydrolysate respirable aerosol for four weeks produced squamous metaplasia and foci of minimal granulomatous laryngitis. No systemic toxicity was observed in rabbits after 9 repeated dermal doses of 17 or 84 mg/kg bw/day or three repeated dermal doses of 126 mg/kg bw/day of APTES; the site of contact NOAEL is less than 17 mg/kg bw/day. The no-observed-adverse-effect level (NOAEL) of APTES in a 90-day oral (gavage) study with rats was 200 mg/kg bw/day. **Genotoxicity:** APTES has been tested in several bacterial reverse mutation/Ames assays, *in vitro* V79 hamster lung cell and Chinese hamster fibroblast chromosome aberration assays, two Chinese hamster ovary cell HGPRT gene mutation assays, and an *in vivo* mouse micronucleus assay. *In vivo* and *in vitro* screening assays have not revealed any evidence of genotoxic potential. **Reproductive and developmental toxicity:** At the highest dose-level (600 mg/kg/day) in a 90 day oral gavage study in rats, no effects were seen on parameters of oestrus cycle and spermatogenesis or reproductive organs. The NOAEL for developmental effects has been identified for APTES following exposure via oral (gavage) in rats, with a value of 100 mg/kg bw/day, the NOAEL for maternal toxicity based on deaths and ulceration of the GI tract is <0.5 mL/kg.
TITANIUM(IV) BIS(ETHYL ACETOACETATO)DIISOPROPOXIDE	No significant acute toxicological data identified in literature search.
Antas Kitchen and Bathroom Silcone Sealant & 3-AMINOPROPYLTRIETHOXYSILANE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
Antas Kitchen and Bathroom Silicone Sealant & OXYBISPHENOXARSINE	Arsenic is a potent toxicant that may exist in several oxidation states and in a number of inorganic and organic forms. Most cases of arsenic-induced toxicity in humans are due to exposure to inorganic arsenic, and there is an extensive database on the human health effects of the common arsenic oxides and oxyacids. Although there may be some differences in the potency of different chemical forms (e.g., arsenites tend to be somewhat more toxic than arsenates), these differences are usually minor. Humans may be exposed to organic arsenicals (mainly methyl and phenyl derivatives of arsenic acid) that are used in agriculture and to organic arsenicals found in fish and shellfish (arsenobetaine and arsenocholine). Although the toxicity of organic arsenicals has not been as extensively investigated as inorganic arsenicals, there are sufficient animal data to evaluate the toxicity of methyl arsenates (e.g., monomethylarsonic acid [MMA] and dimethylarsinic acid [DMA]) and roxarsone. The so-called “fish arsenic” compounds (e.g., arsenobetaine) are not thought to be toxic. The methylation of inorganic arsenic may yield metabolites that alter the cellular oxidation status by potently inhibiting the reduction of glutathione disulfide. The alteration of the oxidation status of the cell by these arsenicals may lead to more serious cytotoxic effects. It is generally accepted that the arsenic-carbon bond is quite strong and most mammalian species do not have the capacity to break this bond; thus, inorganic arsenic is not formed during the metabolism of organic arsenicals. In most species, including humans, ingested (or exogenous) MMA(V) and DMA(V) undergo limited metabolism, do not readily enter the cell, and are primarily excreted unchanged in the urine. This is in contrast to inorganic arsenic, which undergoes sequential reduction and methylation reactions leading to the formation of MMA and DMA. Inorganic As(V) is readily reduced to inorganic As(III), which is taken up by the cell. Within the cell (primarily in the liver), As(III) is methylated to form MMA(V), which is reduced to MMA(III); MMA(III) subsequently undergoes oxidative methylations to form DMA(V). DMA(V) is the primary excretion product in humans. Because inorganic and organic arsenicals exhibit distinct toxicokinetic characteristics, the health effects and MRLs are considered separately. There are limited data on the toxicity of organic arsenicals following inhalation exposure in humans and animals and these data do not allow for identification of critical effects. Keratosis was observed in workers exposed to 0.065 mg/m3 arsanilic acid (i.e., 4-aminophenyl arsenic acid); no alterations in gastrointestinal symptoms or haematological alterations were observed. In animals, very high concentrations (>3,000 mg/m3) of DMA results in respiratory distress, diarrhea, and erythematous lesions on the feet and ears. No adverse effects were observed in rats exposed to DMA concentrations as high as 100 mg DMA/m3 for 95 days. Similarly, the available dermal toxicity data do not allow for identification of critical effects. Contact dermatitis was observed in workers applying DMA (and its sodium salt) and mild dermal irritation was observed in a Draize test in rabbits (adverse effect level not reported). Intermediate duration (21 days) exposure studies in rabbits did not result in systemic toxicity or skin irritation following 5 day/week exposure to 1,000 mg/kg/day MMA or DMA. The preponderance of toxicity data for organic arsenicals involves oral exposure. Human data are limited to three case reports of individuals intentionally ingesting pesticides containing organic arsenicals. Gastrointestinal irritation (vomiting, nausea, and diarrhea) were consistently reported in these cases. Animal data has primarily focused on the toxicity of MMA, DMA, and roxarsone; these data suggest that the targets of toxicity may differ between the compounds.Common signs of toxicity of the organic arsenicals in mice include depression of motility and respiration, irritability, ataxia and convulsions. Death appears to be due to respiratory

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	depression For DMA and TMAO, a period of increased spontaneous motility preceded the death of the mice. More than half of the mice that received a lethal dose of MMA, DMA or TMAO had diarrhoea, which may have contributed to their deaths The LD50s for the organic arsenicals are lower after parenteral administration than after oral administration. Inorganic arsenic but not organic arsenic induces keratinocyte hyperproliferation and disrupts the process of terminal epidermal differentiation in the epidermis. Since methylation serves to expedite the excretion of inorganic arsenic, which is more toxic than organoarsenicals, issues such as whether demethylation occurs and if methylation is saturable, inducible, or inhibitable under expected environmental exposure conditions are critical. Genotoxic effects induced by the organic arsenicals include excess tetraploids (DMA, TMAO), and mitotic arrest (MMA, DMA and TMAO) in Chinese hamster lung (V79) cells . Arsenocholine and arsenobetaine are not genotoxic in V79 cells . In mouse lymphoma cells (L5178Y/TK+/-), incubation of organic arsenicals for 4 h induced cytotoxicity and clastogenicity. MMA was more potent than DMA, but less so than the inorganic arsenicals In vitro studies have proven DMA to be a potent clastogenic agent, capable of inducing DNA damage including double strand breaks and cross-link formation. Reports of successful cancer induction in animals by inorganic arsenic (arsenite and arsenate) have been rare, and most carcinogenetic studies have used organic arsenicals such as DMA combined with other tumor initiators. In rats, the methylated arsenicals, dimethylarsinic acid promotes diethylnitrosamine-initiated liver tumors, whereas trimethylarsine oxide induces liver adenomas. There are a variety of potential mechanisms for arsenical-induced hepatocarcinogenesis, such as oxidative DNA damage, impaired DNA damage repair, acquired apoptotic tolerance, hyperproliferation, altered DNA methylation, and aberrant estrogen signaling. Some of these mechanisms may be liver specific/selective. Overall, accumulating evidence clearly indicates that the liver could be an important target of arsenic carcinogenesis. Arsenic compounds are classified by the European Union as toxic by inhalation and ingestion and toxic to aquatic life and long lasting in the environment. IARC classify arsenic in drinking water as a confirmed human carcinogen (IARC 1). The main inorganic forms of arsenic relevant for human exposures are pentavalent arsenic (also called arsenate, As(V), or As+5) and trivalent arsenic (also called arsenite, As(III), or As+3). These inorganic species undergoes a series of reduction and oxidative/methylation steps in human liver and other tissues to form tri- and pentavalent methylated metabolites of methylarsonite [MA(III)], methylarsonate [MA(V)], dimethylarsinite [DMA(III)], and dimethylarsinate [DMA(V)]. Some mammalian species also produce trimethylated metabolites, trimethylarsine oxide The distinction between inorganic and organic forms is important because it is generally accepted that the organic species are excreted more quickly from the body and generally considered less toxic, with a relative rank order of As(III) > As(V) >> MA(V), DMA(V) >> arsenobetaine. However, the methylated trivalent metabolites, MA(III) and DMA(III), are significantly more toxic than their pentavalent counterpart and either As(III) or As(V) . In many cases, biomonitoring or environmental occurrence data are reported as total arsenic and do not distinguish between the different species. In those situations, understanding the relevant sources of arsenic is essential to evaluate potential arsenic related health effects, especially those related to inorganic arsenic exposure.
Antas Kitchen and Bathroom Silicone Sealant & SILICA AMORPHOUS, FUMED	For silica amorphous: Derived No Adverse Effects Level (NOAEL) in the range of 1000 mg/kg/d. In humans, synthetic amorphous silica (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. 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,

Continued...

	but the reproductive organs in long-term studies were not affected. For Synthetic Amorphous Silica (SAS) Repeated dose toxicity Oral (rat), 2 weeks to 6 months, no significant treatment-related adverse effects at doses of up to 8% silica in the diet. Inhalation (rat), 13 weeks, Lowest Observed Effect Level (LOEL) =1.3 mg/m3 based on mild reversible effects in the lungs. Inhalation (rat), 90 days, LOEL = 1 mg/m3 based on reversible effects in the lungs and effects in the nasal cavity. For silane treated synthetic amorphous silica: Repeated dose toxicity: oral (rat), 28-d, diet, no significant treatment-related adverse effects at the doses tested. 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.
3-AMINOPROPYLTRIETHOXYSILANE & TITANIUM(IV) BIS(ETHYL ACETOACETATO)DIISOPROPOXIDE	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.
3-AMINOPROPYLTRIETHOXYSILANE & OXYBISPHENOXARSINE	The material may produce severe 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) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.

Acute Toxicity	✗	**Carcinogenicity**	✗
Skin Irritation/Corrosion	✓	**Reproductivity**	✓
Serious Eye Damage/Irritation	✓	**STOT - Single Exposure**	✗
Respiratory or Skin sensitisation	✓	**STOT - Repeated Exposure**	✗
Mutagenicity	✗	**Aspiration Hazard**	✗

Legend: ✗ *– Data either not available or does not fill the criteria for classification*
✓ *– Data available to make classification*

SECTION 12 Ecological information

Toxicity

Antas Kitchen and Bathroom Silicone Sealant

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

silica amorphous, fumed

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	24h	Crustacea	>=10000mg/l	1

methyltrimethoxysilane

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>3.6mg/l	2
EC50	48h	Crustacea	>122mg/l	2
LC50	96h	Fish	>110mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	>=3.6mg/l	2

3-aminopropyltriethoxysilane

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	672h	Fish	<0.53	7
NOEC(ECx)	72h	Algae or other aquatic plants	1.3mg/l	2
EC50	72h	Algae or other aquatic plants	603mg/l	2

Continued...

	LC50	96h	Fish	>934mg/l	2
	EC50	48h	Crustacea	331mg/l	2

titanium(IV) bis(ethyl acetoacetato)diisopropoxide					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	>11mg/l	2
	LC50	96h	Fish	4200mg/l	2
	EC50	48h	Crustacea	>35mg/l	2
	NOEC(ECx)	48h	Crustacea	9.8mg/l	2

oxybisphenoxarsine					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50(ECx)	48h	Crustacea	0.005mg/L	4
	LC50	96h	Fish	0.004mg/L	4
	EC50	48h	Crustacea	0.005mg/L	4

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

Harmful 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.
Speciation of arsenic is an important consideration in the fate, movement, and action of this substance. Chemical and biochemical transformations of arsenic include oxidation, reduction and methylation which affects its volatilisation, adsorption, dissolution and biological disposition. The transport of arsenic in the environment is largely controlled by absorption/desorption processes in soils and sediments. Sediment movement is responsible for transport of arsenic soil residues to their ultimate sinks in deep ocean sediments. The clay fraction, plus ferrous and aluminium oxides which coat clay particles, adsorb arsenicals which then undergo transformation as discussed earlier. Conversions of arsenic to volatile alkylarsines leads to air transport losses from soil. Inorganic arsenic occurs in water in different oxidation states depending on the pH and Eh of the water. Arsenate is apparently reduced by bacteria to arsenite in marine environments because the ratio of total arsenate to total arsenic is much lower than that predicted thermodynamically. Methylation of arsenic occurs in both freshwater and marine systems where arsenate occurs as arsenate, arsenite, methanearsonic acid and dimethylarsinic acid. Arsenate predominates because this is the most stable form.
Bioaccumulation occurs in some aquatic species such as seaweeds, freshwater algae and crustaceans. Some arsenic in water flea (Daphnia Magna) and algae occurs as arseno-analogues of phospholipids leading to the mistaken impression that accumulation and utilisation of arsenic takes place at the expense of phosphate. Crabs, lobsters and other marine organisms accumulate organo-arsenicals in the food chain. Although human activity may alter the local picture of environmental arsenic there is little evidence that this affects the global scale arsenic cycle.
Airborne concentrations of arsenic in urban areas may range from a few nanograms to a few tenths of a microgram per cubic meter; airborne arsenic is generally inorganic. In oxygenated soils inorganic arsenic is present in pentavalent form; under reducing conditions it occurs as the trivalent form.
Drinking Water Standards:
arsenic: 50 ug/1 (UK max.) 0.01mg/L (WHO provisional guideline)
chloride:400 mg/l (UK max.)
250 mg/l (WHO guideline)
Soil Guideline:
Dutch Criteria: 29 mg/kg (target)
55 mg/kg (intervention)
Air Quality Standard:
No safe levels recommended due to carcinogenic properties (WHO guideline)
Microbial methylation plays important roles in the biogeochemical cycling of the metalloids and possibly in their detoxification. Many microorganisms (bacteria, fungi, and yeasts) and animals are now known to biomethylate arsenic, forming both volatile (e.g., methylarsines) and nonvolatile (e.g., methylarsonic acid and dimethylarsinic acid) compounds. Antimony and bismuth, also undergo biomethylation to some extent. Trimethylstibine formation by microorganisms is now well established, but this process apparently does not occur in animals. Formation of trimethylbismuth by microorganisms has been reported in a few cases.
For silica amorphous:
Amorphous silica is chemically and biologically inert. It is not biodegradable. Due to its insolubility in water there is a separation at every filtration and sedimentation process.]
Crystalline and/or amorphous silicas are ubiquitous on the earth in soils and sediments, and in living organisms (e.g. diatoms), but only the dissolved form is bioavailable. On a global scale, the level of man-made synthetic amorphous silicas (SAS) represents up to 2.4% of the dissolved silica naturally present in the aquatic environment. The rate of SAS released into the environment during the product life cycle is negligible in comparison with the natural flux of silica in the environment
Untreated SASs have a relatively low water solubility of 1.91 to 2.51 mmol/l (114 - 151 mg/l) and an extremely low vapour pressure (e.g. < 10–3 Pa at 20° C for Aerosil R972). On the basis of these properties it is expected that SAS released into the environment will be distributed mainly into soil/sediment, slightly into water, and probably not at all into air.
With surface-treated SASs, the addition of organosilicon compounds increases the hydrophobicity. Consequently, the water solubility is lower than that of untreated silica. The vapour pressure remains extremely low. Due to the presence of organic substances such as surfactants, salts, acids and alkalis in the environment, it is expected that surface-treated silica will be wetted and then adsorbed onto soils or sediments.
SAS is regarded as an inert substance and is not expected to undergo any transformation in the atmospheric or terrestrial compartment, apart from dissolution by water.

Continued...

Biodegradability in sewage treatment plant or in surface water is not applicable to inorganic substances like SAS. Therefore the biodegradation endpoint has limited relevance for SAS. In surface modified SASs, the most common treating agents are organosilicon compounds and these generally represent less than 5% of the material. Biodegradation in sewage treatment plant or in surface water is not expected. Some biodegradation in soil may occur by analogy with the behaviour of linear polydimethylsiloxane in this compartment

Ecotoxicity:

Based on available data, SAS is not toxic to environmental organisms (apart from physical desiccation in insects). SAS presents a low risk for adverse effects to the environment.

When hydrophilic SASs (Aerosil 200 and Ultrasil VN3; purity 100% and 98%, respectively), were tested for their acute toxicity to fish and crustaceans, the LC50 and EC50 values were higher than 10,000 mg/l and 1,000 mg/l, respectively.

The zebra fish (*Brachydanio rerio)* test was performed with SAS in suspension, due to the insolubility of the SAS. No mortality was observed for the fish after 96 hours of exposure at 1,000 mg/l and 10,000 mg/l. The test media remained turbid throughout the test, indicating that the limit of solubility of the product was exceeded.

With the water flea (*Daphnia magna*), SAS suspensions exceeding the limit of solubility were tested.; some immobilisation was observed. However, no significant immobilisation was observed when a solution filtered through microfibre glass filter was tested. The observed effects were likely caused by physical hampering of the *Daphnia* due to the presence of undissolved particles.

A surface-treated SAS (Aerosil R974; 99.9% pure) was tested on fish and crustaceans. The LC50 to fish and EC50 to *Daphnia* were found to be higher than 10,000 mg/l and 1,000 mg/l, respectively

The EC50 to algae was found to be higher than 10,000 mg/l filtered suspension The actual dissolved concentrations were not determined. There was no inhibition of the biomass or of the growth rate with the 10,000 mg/l filtered suspension.

The antibacterial effect of pressed and unpressed high purity SAS (Aerosil, unspecified) (0.2 g silica + 0.15 ml bacteria strain suspension) kept at 22 C has been investigated (SAS is sometimes pressed to remove air before transportation). The following micro-organisms were studied: *Escherichia coli*, *Proteus* sp., *Pseudomonas aeruginosa*, *Aerobacter aerogenes*, *Micrococcus pyrogenes aureus*, *Streptococcus faecalis*, *Streptococcus pyrogenes humans*, *Corynebacterium diphtheria*, *Candida albicans* and *Bacillus subtilis*. The SAS was contaminated either by hand contact, by saliva droplets or by contact with the atmosphere. Rodshaped gram-negative organisms (*Escherichia coli*, *Bacterium proteus*, *Pseudomonas aeruginosa* and *Aerobacter aerogenes*) died between 6 hours and 3 days in contact with unpressed SAS. Gram-positive micro-organisms were somewhat more resistant. In addition, the tests demonstrated that survival of bacteria was shorter in unpressed than in pressed SAS.

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

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
methyltrimethoxysilane	HIGH	HIGH
3-aminopropyltriethoxysilane	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
methyltrimethoxysilane	LOW (LogKOW = -0.6716)
3-aminopropyltriethoxysilane	LOW (BCF = 5.4)

Mobility in soil

Ingredient	Mobility
methyltrimethoxysilane	LOW (KOC = 381.3)
3-aminopropyltriethoxysilane	LOW (KOC = 12150)

SECTION 13 Disposal considerations

Continued...

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. ▸ **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 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Transport in bulk according to Annex II of MARPOL and the IBC code

Not Applicable

Transport in bulk in accordance with MARPOL Annex V and the IMSBC Code

Product name	Group
silica amorphous, fumed	Not Available
methyltrimethoxysilane	Not Available
3-aminopropyltriethoxysilane	Not Available
titanium(IV) bis(ethyl acetoacetato)diisopropoxide	Not Available
oxybisphenoxarsine	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
silica amorphous, fumed	Not Available
methyltrimethoxysilane	Not Available
3-aminopropyltriethoxysilane	Not Available
titanium(IV) bis(ethyl acetoacetato)diisopropoxide	Not Available
oxybisphenoxarsine	Not Available

SECTION 15 Regulatory information

Safety, health and environmental regulations / legislation specific for the substance or mixture

silica amorphous, fumed is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

methyltrimethoxysilane is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

3-aminopropyltriethoxysilane is found on the following regulatory lists

Continued...

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

titanium(IV) bis(ethyl acetoacetato)diisopropoxide is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

oxybisphenoxarsine is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

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

Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (silica amorphous, fumed; methyltrimethoxysilane; 3-aminopropyltriethoxysilane; titanium(IV) bis(ethyl acetoacetato)diisopropoxide; oxybisphenoxarsine)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (silica amorphous, fumed)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	No (oxybisphenoxarsine)
Taiwan - TCSI	Yes
Mexico - INSQ	No (titanium(IV) bis(ethyl acetoacetato)diisopropoxide; oxybisphenoxarsine)
Vietnam - NCI	Yes
Russia - FBEPH	No (titanium(IV) bis(ethyl acetoacetato)diisopropoxide; oxybisphenoxarsine)
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 Other information

Revision Date	30/06/2021
Initial Date	30/06/2021

SDS Version Summary

Version	Date of Update	Sections Updated
0.0.2.1	26/04/2021	Regulation Change
0.0.3.1	03/05/2021	Regulation Change
0.0.4.1	06/05/2021	Regulation Change
0.0.5.1	10/05/2021	Regulation Change
0.0.5.2	30/05/2021	Template Change
0.0.5.3	04/06/2021	Template Change
0.0.5.4	05/06/2021	Template Change
0.0.6.4	07/06/2021	Regulation Change
0.0.6.5	09/06/2021	Template Change
0.0.6.6	11/06/2021	Template Change
0.0.6.7	15/06/2021	Template Change
0.0.7.7	17/06/2021	Regulation Change
0.0.8.7	21/06/2021	Regulation Change

Continued...

Other information

Ingredients with multiple cas numbers

Name	CAS No
silica amorphous, fumed	68611-44-9, 112945-52-5, 60842-32-2

Classification of the preparation and its individual components has drawn on official and authoritative sources using available literature references.

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

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
ES: Exposure Standard
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index
AIIC: Australian Inventory of Industrial Chemicals
DSL: Domestic Substances List
NDSL: Non-Domestic Substances List
IECSC: Inventory of Existing Chemical Substance in China
EINECS: European INventory of Existing Commercial chemical Substances
ELINCS: European List of Notified Chemical Substances
NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory
KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals
PICCS: Philippine Inventory of Chemicals and Chemical Substances
TSCA: Toxic Substances Control Act
TCSI: Taiwan Chemical Substance Inventory
INSQ: Inventario Nacional de Sustancias Químicas
NCI: National Chemical Inventory
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances