Threebond 1217H

Three Bond Chemwatch Hazard Alert Code: 3

Chemwatch: **97-08273**
Version No: **3.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **07/28/2021**
Print Date: **03/10/2023**
L.GHS.AUS.EN

SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	Threebond 1217H
Chemical Name	Not Applicable
Synonyms	Not Available
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	Use according to manufacturer's directions.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**Three Bond**
Address	6184 Schumacher Park Drive West Chester OH 45069 United States
Telephone	+1 513 779 7300
Fax	+1 513 779 7375
Website	https://www.threebond.com/
Email	H2BC@threebond.co.jp

Emergency telephone number

Association / Organisation	**Three Bond**	**CHEMWATCH EMERGENCY RE**
Emergency telephone numbers	+1 800 424 9300	+61 1800 951 288
Other emergency telephone numbers	Not Available	+61 3 9573 3188

Once connected and if the message is not in your preferred language then please dial 01

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable

Classification [1]	Flammable Liquids Category 4, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Seri Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Categ
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (* *VI*

Label elements

Hazard pictogram(s)	
Signal word	**Danger**

Hazard statement(s)

H227	Combustible liquid.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H318	Causes serious eye damage.
H335	May cause respiratory irritation.

Precautionary statement(s) Prevention

P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.

P261	Avoid breathing mist/vapours/spray.
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

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance wi

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
70131-67-8	40-50	dimethylsiloxane, hydroxy-terminated
471-34-1	40-50	calcium carbonate
68611-44-9	1-5	silica amorphous, fumed
2224-33-1	<5	vinyltris(methylethylketoxime)silane
108-88-3	<1	toluene
76735-64-3	<0.5	3-[methylbis[(1-methylethenyl)oxy]silyl]propoxy] ethoxylated
1333-86-4	<0.1	carbon black
96-29-7	NotSpec	methyl ethyl ketoxime

Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (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: • Immediately hold eyelids apart and flush the eye continuously with running water.

	• Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving th lifting the upper and lower lids. • Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at le • Transport to hospital or doctor without delay. • 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 or combustion products are inhaled remove from contaminated area. • Lay patient down. Keep warm and rested. • Prostheses such as false teeth, which may block airway, should be removed, where possible, pr procedures. • Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valv mask as trained. Perform CPR if necessary. • Transport to hospital, or doctor, without delay.
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

Treat symptomatically.

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 c result

Advice for firefighters

Fire Fighting	• When silica dust is dispersed in air, firefighters should wear inhalation protection as hazardous s 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	• High temperature decomposition products include silicon dioxide, small amounts of formaldehyd and traces of silicon polymers. • These gases may ignite and, depending on circumstances, may cause the resin/polymer to ignit • An outer skin of silica may also form. Extinguishing of fire, beneath the skin, may be difficult.

	• When silica dust is dispersed in air, firefighters should wear inhalation protection as hazardous s be adsorbed on the silica particles. • When heated to extreme temperatures, (>1700 deg.C) amorphous silica can fuse. • Combustible. • Slight fire hazard when exposed to heat or flame. • Heating may cause expansion or decomposition leading to violent rupture of containers. • On combustion, may emit toxic fumes of carbon monoxide (CO). • May emit acrid smoke. • Mists containing combustible materials may be explosive. Combustion products include: carbon monoxide (CO) carbon dioxide (CO2) nitrogen oxides (NOx) silicon dioxide (SiO2) metal oxides other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes. Heating calcium carbonate at high temperatures(825 C.) causes decomposition, releases carbon dioxide of alkaline lime
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	Environmental hazard - contain spillage. Slippery when spilt. • Clean up all spills immediately. • 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	Environmental hazard - contain spillage. Slippery when spilt. 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 • 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 wo maintained.
Other information	• Store in original containers. • Keep containers securely sealed. • No smoking, naked lights or ignition sources. • 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	Traces of benzene, a carcinogen, may form when silicones are heated in air above 230 degrees C. Conc cause degradation of polymer. Boiling water may soften and weaken material. Calcium carbonate: • is incompatible with acids, ammonium salts, fluorine, germanium, lead diacetate, magnesium, m silver nitrate, titanium. Contact with acid generates carbon dioxide gas, which may pressurise and then rupture closed containe The substance may be or contains a "metalloid" The following elements are considered to be metalloids; boron,silicon, germanium, arsenic, antimony, tell polonium The electronegativities and ionisation energies of the metalloids are between those of the metals and nor exhibit characteristics of both classes. The reactivity of the metalloids depends on the element with which 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 only salts such as arsenic halides, by the reaction with certain strong acid, but it also forms arsenites by r Most metalloids have a multiplicity of oxidation states or valences. For instance, tellurium has the oxidatio Metalloids react like non-metals when they react with metals and act like metals when they react with nor Silicas:

	• react with hydrofluoric acid to produce silicon tetrafluoride gas • react with xenon hexafluoride to produce explosive xenon trioxide • reacts exothermically with oxygen difluoride, and explosively with chlorine trifluoride (these halo commonplace industrial materials) and other fluorine-containing compounds • may react with fluorine, chlorates • are incompatible with strong oxidisers, manganese trioxide, chlorine trioxide, strong alkalis, met orthophosphoric acid, vinyl acetate • may react vigorously when heated with alkali carbonates. • Avoid strong acids, bases. • Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	calcium carbonate	Calcium carbonate	10 mg/m3	Not Available	Not Available	(a) This value is f no asbestos and
Australia Exposure Standards	toluene	Toluene	50 ppm / 191 mg/m3	574 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	carbon black	Carbon black	3 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
dimethylsiloxane, hydroxy-terminated	190 mg/m3	2,100 mg/m3	13,000 mg/m3
calcium carbonate	45 mg/m3	210 mg/m3	1,300 mg/m3
silica amorphous, fumed	18 mg/m3	100 mg/m3	630 mg/m3
toluene	Not Available	Not Available	Not Available
carbon black	9 mg/m3	99 mg/m3	590 mg/m3
methyl ethyl ketoxime	30 ppm	56 ppm	250 ppm

Ingredient	Original IDLH	Revised IDLH
dimethylsiloxane, hydroxy-terminated	Not Available	Not Available
calcium carbonate	Not Available	Not Available
silica amorphous, fumed	Not Available	Not Available
vinyltris(methylethylketoxime)silane	Not Available	Not Available
toluene	500 ppm	Not Available

Ingredient	TEEL-1	TEEL-2	TEEL-3
3-[methylbis[(1-methylethenyl)oxy]silyl]propoxy] ethoxylated		Not Available	Not Available
carbon black		1,750 mg/m3	Not Available
methyl ethyl ketoxime		Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Ban
vinyltris(methylethylketoxime)silane	D	> 0.1 to ≤ 1 ppm
methyl ethyl ketoxime	D	> 0.1 to ≤ 1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or chemical's potency and the adverse health outcomes associated with exposure. The output of occupational exposure band (OEB), which corresponds to a range of exposure concentrations worker health.*	

MATERIAL DATA

Exposure controls

Appropriate engineering controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. controls can be highly effective in protecting workers and will typically be independent of worker interactio 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 strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air co properly. The design of a ventilation system must match the particular process and chemical or contamin 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. Corre adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is 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 wo "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to eff 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	0.5-1 m/s (100-200 f/min.)

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)	
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 extract
decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed
should be adjusted, accordingly, after reference to distance from the contaminating source. The air veloc
example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2
extraction point. Other mechanical considerations, producing performance deficits within the extraction ap
that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed

Individual protection measures, such as personal protective equipment

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.
  describing the wearing of lenses or restrictions on use, should be created for each workplace or tas
  review of lens absorption and adsorption for the class of chemicals in use and an account of injury e
  aid personnel should be trained in their removal and suitable equipment should be readily available
  exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens sh
  signs of eye redness or irritation - lens should be removed in a clean environment only after worker
  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, wh other protective equipment, to avoid all possible skin contact. • Contaminated leather items, such as shoes, belts and watch-bands should be removed and des
Body protection	See Other protection below
Other protection	• Overalls. • P.V.C apron. • Barrier cream. • Skin cleansing cream. • Eye wash unit.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
***"*Forsberg Clothing Performance Index".**
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
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Material	CPI
PE/EVAL/PE	A
PVA	A
VITON	A
VITON/CHLOROBUTYL	A
TEFLON	B
BUTYL	C
CPE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PVC	C
SARANEX-23	C
SARANEX-23 2-PLY	C
VITON/NEOPRENE	C

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

^ - Full-face

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	Gray paste; does not mix with water.		
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 Applicable	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	275
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	73	**Taste**	Not Available

Evaporation rate	<1	**Explosive properties**	Not Available
Flammability	Combustible.	**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	Immiscible	**pH as a solution (1%)**	Not Applicable
Vapour density (Air = 1)	>1	**VOC g/L**	<2.5

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	• Silicone fluids are stable under normal storage conditions. • Hazardous polymerisation will not occur. • At temperatures > 150 C, silicones can slowly react with the oxygen in air. • When heated > 300 C, silicones can slowly depolymerise to volatile siloxanes whether or not air 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	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory sy number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a ch removing or neutralising the irritant and then repairing the damage. The repair process, which initially evo lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impa primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involvir activation of many cell types, mainly derived from the vascular system. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, redu reflexes, lack of coordination and vertigo. The principal toxic effects of methyl ethyl ketoxime MEKO in animal studies, regardless of the route of ad haemolytic anaemia, increased respiration; and reversible reduction in spontaneous activity, motor coord At high vapour concentration the product has a reversible narcotic action Extremely high concentrations r respiratory failure. Not normally a hazard due to non-volatile nature of product

Ingestion	The material has **NOT** been classified by EC Directives or other classification systems as "harmful by ing the lack of corroborating animal or human evidence. The material may still be damaging to the health of t ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions o are generally based on doses producing mortality rather than those producing morbidity (disease, ill-heal discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignifica to be cause for concern.
Skin Contact	The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the • produces moderate inflammation of the skin in a substantial number of individuals following dire • produces significant, but moderate, inflammation when applied to the healthy intact skin of anim 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 cont The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progr (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular of the skin (spongiosis) and intracellular oedema of the epidermis. Skin contact is not thought to have harmful health effects (as classified under EC Directives); the materia damage following entry through wounds, lesions or abrasions. Application of 0.5 gm methyl ethyl ketoxime (MEKO) to the backs of rabbits for 24 hours under an occlusi irritation (Draize score 1.5 out of 8). MEKO was a strong sensitiser in the maximisation test (8 out of 10 guinea pigs were sensitised. 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 prod harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is Low molecular weight silicone fluids may exhibit solvent action and may produce skin irritation. Excessive use or prolonged contact may lead to defatting, drying and irritation of sensitive skin
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present tw instillation. 0.1 ml of methyl ethyl ketoxime (MEKO) was corrosive to the rabbit eye. When the eyes of human subjects where exposed to silicone fluids, there was evidence of transitory conj few hours; this resolved within 24 hours. When applied to the eyes of rabbits, silicone fluids produced tra no longer than 48 hours. Injection into the various structures of the eye of animals produced corneal scar in the retina, foreign body reaction and cataracts.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathin problems. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation re 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 i airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have bec further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. Thes severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-res 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 people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmage Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper Activities giving rise to short-term peak concentrations should receive particular attention when risk manag Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which ma and there should be appropriate consultation with an occupational health professional over the degree of ris Exposure to the material may cause concerns for human fertility, generally on the basis that results in ani sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evide

occurring at around the same dose levels as other toxic effects, but which are not a secondary non-speci
toxic effects.

Exposure to the material may cause concerns for humans owing to possible developmental toxic effects,
results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of
toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-spec
toxic effects.
Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative he
or biochemical systems.
The synthetic, amorphous silicas are believed to represent a very greatly reduced silicosis hazard compa
are considered to be nuisance dusts.
When heated to high temperature and a long time, amorphous silica can produce crystalline silica on coo
containing crystalline silicas may lead to silicosis, a disabling pulmonary fibrosis that may take years to d
between various studies showing that fibrosis associated with chronic exposure to amorphous silica and
explained by assuming that diatomaceous earth (a non-synthetic silica commonly used in industry) is eith
nonfibrogenic and that fibrosis is due to contamination by crystalline silica content
Pure calcium carbonate does not produce pneumoconiosis probably being eliminated from the lungs slow
As mined, unsterilised particulates can carry bacteria into the air passages and lungs, producing infection
Methyl ethyl ketoxime (MEKO) administered to rats by gavage at 25, 75 and 225 mg/kg/day, 7 days/week
dose-related decreases in red blood cell counts and haemoglobin and haematocrit values accompanied b
reticulocytosis (increased number of young red blood cells).
Other effects included a dose-related pattern of spleen, liver and kidney weights. The spleen and liver sh
compensatory red blood cell production suggesting that, in the rat, MEKO induces haemolytic anaemia w
erythropoiesis. A no-observed-effect-level was not established but effects at 25 mg/kg were described as
When MEKO was administered to rats at dose levels of 0.5, and 1.0 ml/kg/day, daily for 4 weeks, transien
depression immediately followed. At 4 weeks dose-related decreases were seen in red blood cell count a
related increases were evident in spleen weight (from 1.7 to 3.2 fold). It was concluded that 0.1 ml/kg pro
When rats were exposed by inhalation to MEKO vapour for 6 hours/day, 5 days/week for 4 weeks, mild in
corpuscular volume, mean corpuscular haemoglobin, reticulocyte count and red blood cell count were se
Spleen weights were increased and haemosiderosis (deposits of iron) in the spleen were seen at 714 ppm
Haemosiderosis probably resulted from red blood cell haemolysis. Exposures at 60 and 283 ppm produce
An increased incidence of liver tumours was observed microscopically in male mice exposed to 375 ppm
and female mice exposed at 375 ppm showed enlarged livers but tumours did not occur in females.
High blood concentrations of calcium ion may give rise to vasodilation and depress cardiac function leading t
Calcium ions enhance the effects of digitalis on the heart and may precipitate digitalis intoxication. Calcium s
absorption of tetracyclines
In neonates calcification of soft-tissue has been observed following therapeutic administration.
Some studies show that large quantities of calcium intake can cause hypercalcemia, which can in turn lead to
can occur within hours or days or, alternatively, settles gradually, evolving over several years until it reaches
acute renal failure can also develop into chronic forms of the disease.
Hypercalcaemia conditions can be associated with normal or reduced calcium serum levels, as the body tend
metabolism of the mineral, known as the compensation phase. When there is a slight increase in the concentr
calcium excretion markedly increases, while intestinal absorption decreases After kidney damage has set in,
thereby decreasing the serum concentration.
Serum protein levels may decrease as a result of proteinuria in cases of renal complications. Proteinuria is an
and represents an independent risk factor for the progression of such a condition. Increased serum creatinin
important parameter, given that kidney diseases are associated with increased serum creatinine levels. When
progressive loss of glomerular filtration begins, resulting in increased plasma creatinine concentrations. Dur
failure, discrete, but constant, increments in plasma creatinine levels occur.

	Renal disease with albuminuria may also be the cause of hypoalbuminemia in patients with liver disease. In c damage, increased calcium urinary excretion may occur. Therefore, a similar increase may cause the decline current study. 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 nu airborne concentrations ranging from 0.5 mg/m3 to 150 mg/m3. Lowest-observed adverse effect levels (L the range of 1 to 50 mg/m3. When available, the no-observed adverse effect levels (NOAELs) were betw Differences in values may be due to particle size, and therefore the number of particles administered per particle size diminishes so does the NOAEL/ LOAEL. Exposure produced transient increases in lung infla injury and lung collagen content. There was no evidence of interstitial pulmonary fibrosis. On the basis, primarily, of animal experiments, concern has been expressed that the material may produ mutagenic effects; in respect of the available information, however, there presently exists inadequate dat assessment.

Threebond 1217H	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
dimethylsiloxane, hydroxy-terminated	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Not Available
	Oral (Rat) LD50: >5000 mg/kg[2]	
calcium carbonate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit): 0.75 mg/24h - SEVERE
	Inhalation(Rat) LC50: >3 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin (rabbit): 500 mg/24h-moderate
		Skin: no adverse effect observed (not irritating)[1]
silica amorphous, fumed	**TOXICITY**	**IRRITATION**
	Inhalation(Rat) LC50: 0.45 mg/L4h[2]	Not Available
	Oral (Rat) LD50: >5000 mg/kg[2]	
vinyltris(methylethylketoxime)silane	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2009 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
toluene	**TOXICITY**	**IRRITATION**

	Dermal (rabbit) LD50: 12124 mg/kg[2]	Eye (rabbit): 2mg/24h - SEVERE
	Inhalation(Rat) LC50: >13350 ppm4h[2]	Eye (rabbit):0.87 mg - mild
	Oral (Rat) LD50: 636 mg/kg[2]	Eye (rabbit):100 mg/30sec - mild
		Eye: adverse effect observed (irritating)[1]
		Skin (rabbit):20 mg/24h-moderate
		Skin (rabbit):500 mg - moderate
		Skin: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]

3-[methylbis[(1-methylethenyl)oxy]silyl]propoxy] ethoxylated	TOXICITY	IRRITATION
	Not Available	Not Available

carbon black	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

methyl ethyl ketoxime	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >184<1840 mg/kg[1]	Eye (rabbit): 0.1 ml - SEVERE
	Inhalation(Rat) LC50: >4.83 mg/l4h[1]	
	Oral (Rat) LD50: >900 mg/kg[1]	

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

DIMETHYLSILOXANE, HYDROXY-TERMINATED	* [Mobay Chemical Corp] **[GE] For siloxanes: Effects which based on the reviewed literature do not seem to be problematic are a sensitization and genotoxicity. Some studies indicate that some of the siloxanes may have endocrine disrupting pr effects have caused concern about the possible effects of the siloxanes on humans a Only few siloxanes are described in the literature with regard to health effects, and make broad conclusions and comparisons of the toxicity related to short-chained li based on the present evaluation. Data are primarily found on the cyclic siloxanes D4 (octamethylcyclotetrasiloxane) and D5 (decamethylcyclopentasiloxane) and the short-linear HMDS (hexamethyldis

	These three siloxanes have a relatively low order of acute toxicity by oral, dermal a not require classification for this effect. They are not found to be irritating to skin or eyes and are also not found sensitizing respiratory sensitization have not been identified. Subacute and subchronic toxicity studies show that the liver is the main target orga liver cell enzymes. This enzyme induction contributes to the elimination of the subs Primary target organ for D5 exposure by inhalation is the lung. D5 has an enzyme i that of D4. Subacute and subchronic inhalation of HMDS affect in particular the lung None of the investigated siloxanes show any signs of genotoxic effects *in vitro* or *in* indicate that D5 has a potential carcinogenic effect. D4 is considered to impair fertility in rats by inhalation and is classified as a substa category 3 with the risk phrase R62 ('Possible risk of impaired fertility'). The results of a study to screen for oestrogen activity indicate that D4 has very wea antioestrogenic activity and is a partial agonist (enhances the effect of the estrogen compounds that are weakly oestrogenic to also have antioestrogenic properties. Comparison of the oestrogenic ethinyloestradiol (steroid hormone) indicates that D4 is 585,000 times less potent rat stain Sprague- Dawley and 3.7 million times less potent than ethinyloestradiol i Because of the lack of effects on other endpoints designated to assess oestrogenicity of action for the D4 reproductive effects has been questioned. An indirect mode of a LH (luteinising hormone) surge necessary for optimal timing of ovulation has been Based on the reviewed information, the critical effects of the siloxanes are impaired carcinogenic effects (uterine tumours in females). Furthermore there seem to be so following repeated exposures, the liver (D4), kidney (HMDS) and lung (D5 and HMDS) being t A possible oestrogenic effect contributing to the reproductive toxicity of D4 is deba be some indication that this toxicity may be caused by another mechanism than oes Studies are available for linear siloxanes from an analogue group comprising di- to key physicochemical properties, The results of the acute toxicity studies for this ana agreement: there is no evidence from any of the available studies that the substance potential for acute toxicity (in terms of either lethality or adverse clinical effects) by exceeding the maximum dose levels tested according to current OECD guidelines. It across the lack of acute toxicity between the members of the group where there are The metabolism of silanes and siloxanes is influenced by the chemistry of silicon, ar different from that of carbon compounds. These differences are due to the fact that electropositive than carbon; Si-Si bonds are less stable than C-C bonds and Si-O bon latter due to their high bond energy. Functional groups such as -OH, -CO2H, and -CH organic drug metabolites. If such functionalities are formed from siloxane metaboli rearrangement with migration of the Si atom from carbon to oxygen. Consequently, isomerise to silanols and this provides a mechanism by which very polar metabolite hydrophobic alkylsiloxanes in relatively few metabolic steps
CALCIUM CARBONATE	No evidence of carcinogenic properties. No evidence of mutagenic or teratogeni Asthma-like symptoms may continue for months or even years after exposure to th due to a non-allergic condition known as reactive airways dysfunction syndrome (R exposure to high levels of highly irritating compound. Main criteria for diagnosing I previous airways disease in a non-atopic individual, with sudden onset of persisten within minutes to hours of a documented exposure to the irritant. Other criteria for reversible airflow pattern on lung function tests, moderate to severe bronchial hyp challenge testing, and the lack of minimal lymphocytic inflammation, without eosin

	following an irritating inhalation is an infrequent disorder with rates related to the duration of exposure to the irritating substance. On the other hand, industrial bron occurs as a result of exposure due to high concentrations of irritating substance (of completely reversible after exposure ceases. The disorder is characterized by diffic mucus production. The material may produce severe irritation to the eye causing pronounced inflam prolonged exposure to irritants may produce conjunctivitis.
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, s inhalation. Epidemiology studies show little evidence of adverse health effects du exposure (without personal protection) may cause mechanical irritation of the ey skin. When experimental animals inhale synthetic amorphous silica (SAS) dust, it diss rapidly eliminated. If swallowed, the vast majority of SAS is excreted in the faece accumulation in the body. Following absorption across the gut, SAS is eliminated modification in animals and humans. SAS is not expected to be broken down (m After ingestion, there is limited accumulation of SAS in body tissues and rapid el absorption has not been calculated, but appears to be insignificant in animals an subcutaneously are subjected to rapid dissolution and removal. There is no indic in animals or humans based on chemical structure and available data. In contras soluble in physiological media and the soluble chemical species that are formed urinary tract without modification. Both the mammalian and environmental toxicology of SASs are significantly influ chemical properties, particularly those of solubility and particle size. SAS has no inhalation. Adverse effects, including suffocation, that have been reported were high numbers of respirable particles generated to meet the required test atmospl representative of exposure to commercial SASs and should not be used for hum repeated exposure of the skin may cause dryness and cracking, SAS is not a sk a sensitiser. Repeated-dose and chronic toxicity studies confirm the absence of toxicity when skin contact. Long-term inhalation of SAS caused some adverse effects in animals (increases injury and lung collagen content), all of which subsided after exposure. Numerous repeated-dose, subchronic and chronic inhalation toxicity studies hav in a number of species, at airborne concentrations ranging from 0.5 mg/m3 to 15 adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m3. Wh observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m3. The explained by different particle size, and therefore the number of particles adminis general, as particle size decreases so does the NOAEL/LOAEL. Neither inhalation nor oral administration caused neoplasms (tumours). SAS is n genotoxicity was detected in in vivo assays. SAS does not impair development c specifically studied, but the reproductive organs in long-term studies were not af For Synthetic Amorphous Silica (SAS) Repeated dose toxicity Oral (rat), 2 weeks to 6 months, no significant treatment-related adverse effects in the diet. Inhalation (rat), 13 weeks, Lowest Observed Effect Level (LOEL) =1.3 mg/m3 ba

effects in the lungs. Inhalation (rat), 90 days, LOEL = 1 mg/m3 based on reversil
effects in the nasal cavity.
For silane treated synthetic amorphous silica:
Repeated dose toxicity: oral (rat), 28-d, diet, no significant treatment-related adv
tested.
There is no evidence of cancer or other long-term respiratory health effects (for
workers employed in the manufacture of SAS. Respiratory symptoms in SAS wo
correlate with smoking but not with SAS exposure, while serial pulmonary functi
radiographs are not adversely affected by long-term exposure to SAS.
For silane, dichlorodimethyl-, reaction products with silica
Acute oral toxicity is very low for treated silica. Acute inhalation toxicity was only t
and is not relevant for the material used industrially. Changes in respiratory organs
after repeated exposure were reversible in animals that survived the exposure and
valid TLV values, only. If TLV values are maintained no health hazards are expected
sufficiently investigated. Treated silica is not mutagenic. The NOAEL for repro/deve
mg/kg bw.
Acute toxicity: In a limit test giving 10% in the diet (5000 mg/kg bw) to rats the ac
determined to be higher than 5000 mg/kg bw. In another study administering singl
mg/kg bw to rats the LD50 was also concluded to be higher than 5000 mg/kg bw. I
giving still higher single doses in olive oil the LD50 appeared to be above 7900 mg/
were observed in any of these studies.
All inhalation testing has been conducted with a substance that differs significantly
product based on particle size. In these animal tests the experimental design caused
reduced resulting in nearly 100% of the particle fraction being below 10 um and ca
lung (alveolar particle fraction). The alveolar fraction is responsible for the toxicolo
overloading of the lung due to poor dust clearance mechanisms) which were obser
477, 450, 520-1120, and >2280 mg/m3 and corresponding mass median aerodynar
um, 1.24 um, 0.8 – 0.9 um and 0.15 um, respectively. In comparison to the particle s
inhalation animal tests, only minor amounts (less than 1 %) of the commercially av
have been measured as respirable (alveolar fraction < 10 um MMAD) using test me
Using the same method > 99% of the particle fraction is in excess of 90 um and can
(nasal passages and throat) or cannot be inhaled at all. Therefore the tests do not re
behavior of the commercial product and are not considered relevant for inclusion i
definition/hazard assessment of the commercial substance.
Genetic toxicity: The test substance was not mutagenic in the Bacterial Reverse M
with Salmonella *typhimurium* TA98, TA100, TA1535, TA1537 and TA1538 strains a
strain. Also an in vitro chromosomal aberration study in CHO cells gave negative re
Repeat dose toxicity: A 24-month oral feeding study administering a 100 mg/kg d
rats resulted in a NOAEL of 100 mg/kg. No clinical signs or treatment-related chang
observed. There were no carcinogenic effects.A 6-month oral feeding study showed
at the given dose of 500 mg/kg bw to rats (40/sex) resulting in a NOAEL of 500 mg
but reversible -transformation of the adrenal cortex in females was attributed to ch
feeding study (5-8 weeks) exposed rats (5/sex/treatment) to a dose of 500, 1000 o
and increasing these doses gradually to 4000, 8000 and 16000 mg/kg bw, respectiv
and food consumption combined with apathy and decreased grooming activity and
glycogen in hepatocytes may indicate a starving condition of these animals. At the h
animals died. The NOAEL was determined to be 500 mg/kg bw (LOAEL = 1000 mg/
study where a dose of 500 or 1000 mg/kg bw was administered by gavage to 30 rat
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 gran lungs, accumulations of alveolar macrophages, alveolar spaces filled with granular polymorphonuclear leucocytes, alveolar bronchiolisation, interstitial fibrosis and e nodes. In a 2-week study administering 0, 31, 87 or 420 mg/m3 to a total number o females died at the top dose level. The rats at the top dose level showed severe resp A dose-related decrease in body weight was observed at 87 mg/m3 and higher. The as those observed in the 13-week inhalation study. A 3-day study and an 8-12-mon concentration of 50 mg/m3 to rats yielded similar results to the above studies in th particles was determined to be smaller than 7 µm. Changes in respiratory organs (i observed in inhalative repeated dose toxicity testing were reversible in animals tha There was no indication of silicosis. Concentrations of the substances with toxicolo toxicity testing were above the valid TLV values (10mg/m3 USA). If TLV values are hazards are expected. **Reproductive and developmental toxicity:** Two studies are included on repro/d month, 1-generation study in rats combining fertility and prenatal toxicity testing a in the food to 10 females and 2 males. No treatment-related effects were observed i offspring. Therefore the NOAEL for parents and offspring was 500 mg/kg. No effect were observed. In a 2-generation reproduction study 20 male and 20 female rats w oral feed for 24 months (see also repeated dose). No abnormalities were observed i NOAEL of 100 mg/kg bw.
VINYLTRIS(METHYLETHYLKETOXIME)SILANE	alpha,beta-Unsaturated oximes represent two previously unknown class putative metabolites were proposed as sensitising agents. These include alpha,beta-epoxy oximes and a nitro analogue. When tested in the LLNA oximes. Allergic Contact Dermatitis—Formation, Structural Requirements,and R Sensitizers. Ann-Therese Karlberg et al: Chem. Res. Toxicol. 2008, 21, pp 53–69 https://ftp.cdc.gov/pub/Documents/OEL/06.%20Dotson/References/Karl
TOLUENE	For toluene: **Acute Toxicity** Humans exposed to intermediate to high levels of toluene for short periods of tim central nervous system effects ranging from headaches to intoxication, convulsio Similar effects are observed in short-term animal studies. **Humans -** Toluene ingestion or inhalation can result in severe central nervous s large doses, can act as a narcotic. The ingestion of about 60 mL resulted in fatal within 30 minutes in one reported case. Constriction and necrosis of myocardial fibers, markedly swollen liver, congestio lungs and acute tubular necrosis were found on autopsy. Central nervous system effects (headaches, dizziness, intoxication) and eye irrit inhalation exposure to 100 ppm toluene 6 hours/day for 4 days. Exposure to 600 ppm for 8 hours resulted in the same and more serious sympto dilated pupils, convulsions, and nausea . Exposure to 10,000-30,000 ppm has b narcosis and death Toluene can also strip the skin of lipids causing dermatitis **Animals -** The initial effects are instability and incoordination, lachrymation and exposure), followed by narcosis. Animals die of respiratory failure from severe n Cloudy swelling of the kidneys was reported in rats following inhalation exposure hours/day for 3 days **Subchronic/Chronic Effects:**

Repeat doses of toluene cause adverse central nervous system effects and can
respiratory system, the liver, and the kidney. Adverse effects occur as a result fr
inhalation exposures. A reported lowest-observed-effect level in humans for adv
is 88 ppm.

Humans - Chronic occupational exposure and incidences of toluene abuse have
and liver function changes. It has also resulted in nephrotoxicity and, in one case
and fatal cardiotoxin.

Neural and cerebellar dystrophy were reported in several cases of habitual "glue
epidemiological study in France on workers chronically exposed to toluene fume
neutropenia. Exposure levels were not given in the secondary reference; howeve
excretion of hippuric acid, a metabolite of toluene, was given as 4 g/L compared

Animals - The major target organs for the subchronic/chronic toxicity of toluene
liver, and kidney. Depressed immune response has been reported in male mice
mg/kg/day for 28 days. Toluene in corn oil administered to F344 male and femal
days/week for 13 weeks, induced prostration, hypoactivity, ataxia, piloerection, la
salivation, and body tremors at doses 2500 mg/kg. Liver, kidney, and heart weig
this dose and histopathologic lesions were seen in the liver, kidneys, brain and u
observed-adverse effect level (NOAEL) for the study was 312 mg/kg (223 mg/kg
observed-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg

Developmental/Reproductive Toxicity

Exposures to high levels of toluene can result in adverse effects in the developin
studies have indicated that high levels of toluene can also adversely effect the d
laboratory animals.

Humans - Variable growth, microcephaly, CNS dysfunction, attentional deficits,
abnormalities, and developmental delay were seen in three children exposed to
of maternal solvent abuse before and during pregnancy

Animals - Sternebral alterations, extra ribs, and missing tails were reported follo
1500 mg/m3 toluene 24 hours/day during days 9-14 of gestation. Two of the dam
Another group of rats received 1000 mg/m3 8 hours/day during days 1-21 of ges
or toxicity occurred, however, minor skeletal retardation was present in the expo
were exposed to 500 or 1500 mg/m3 toluene continuously during days 6-13 of p
the high dose during the first 24 hours of exposure, however none died at 500 m
weight was reported, but there were no differences in the incidences of skeletal
between the treated and control offspring.

Absorption - Studies in humans and animals have demonstrated that toluene is
lungs and the gastrointestinal tract. Absorption through the skin is estimated at a
by the lungs when exposed to toluene vapor.

Dermal absorption is expected to be higher upon exposure to the liquid; howeve
rapid evaporation of toluene .

Distribution - In studies with mice exposed to radiolabeled toluene by inhalation
were present in body fat, bone marrow, spinal nerves, spinal cord, and brain whi
radioactivity were present in blood, kidney, and liver. Accumulation of toluene ha
adipose tissue, other tissues with high fat content, and in highly vascularised tiss

Metabolism - The metabolites of inhaled or ingested toluene include benzyl alco
hydroxylation of the methyl group. Further oxidation results in the formation of be
acid. The latter is conjugated with glycine to yield hippuric acid or reacted with gl
benzoyl glucuronide. o-cresol and p-cresol formed by ring hydroxylation are cons

Excretion - Toluene is primarily (60-70%) excreted through the urine as hippuric
benzoyl glucuronide accounts for 10-20%, and excretion of unchanged toluene t
accounts for 10-20%. Excretion of hippuric acid is usually complete within 24 hou

3-[METHYLBIS[(1-METHYLETHENYL)OXY]SILYL]PROPOXY] ETHOXYLATED	Polyethers, for example, ethoxylated surfactants and polyethylene glyco towards air oxidation as the ether oxygens will stabilize intermediary rad Investigations of a chemically well-defined alcohol (pentaethylene glycol ethoxylate, showed that polyethers form complex mixtures of oxidation p air. Sensitization studies in guinea pigs revealed that the pure nonoxidized s nonsensitizing but that many of the investigated oxidation products are s hydroperoxides were identified in the oxidation mixture, but only one (16 3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated strong sensitizer in LLNA (local lymph node assay for detection of sensit formation of other hydroperoxides was indicated by the detection of thei aldehydes in the oxidation mixture . On the basis of the lower irritancy, nonionic surfactants are often preferr topical products. However, their susceptibility towards autoxidation also increases the irritation. Bec effect, it is difficult to diagnose ACD to these compounds by patch testing. Allergic Contact Dermatitis—Formation, Structural Requirements,and R Sensitizers. Ann-Therese Karlberg et al; Chem. Res. Toxicol.2008,21,53-69 Polyethylene glycols (PEGs) have a wide variety of PEG-derived mixture linkable terminal primary hydroxyl groups in combination with many poss complexes such as ethers, fatty acids, castor oils, amines, propylene gly derivatives. PEGs and their derivatives are broadly utilized in cosmetic p emulsifiers, cleansing agents, humectants, and skin conditioners. PEGs and PEG derivatives were generally regulated as safe for use in c conditions that impurities and by-products, such as ethylene oxides and known carcinogenic materials, should be removed before they are mixed formulations. Most PEGs are commonly available commercially as mixtures of differer broadly- or narrowly-defined molecular weight (MW) ranges. For instanc designates a mixture of PEG molecules (n = 195 to 265) having an aver is also known as polyethylene oxide (PEO) or polyoxyethylene (POE), w chemical synonyms. However, PEGs mainly refer to oligomers and poly masses below 20,000 g/mol, while PEOs are polymers with molecular m g/mol, and POEs are polymers of any molecular mass. Relatively small are produced by the chemical reaction between ethylene oxide and wate other ethylene glycol oligomers), as catalyzed by acidic or basic catalyst high-molecular weight PEGs, synthesis is performed by suspension poly necessary to hold the growing polymer chain in solution during the cours condensation process. The reaction is catalyzed by magnesium-, alumin organoelement compounds. To prevent coagulation of polymer chains in additives such as dimethylglyoxime are used Safety Evaluation of Polyethyene Glycol (PEG) Compounds for Cosmeti 31:105-136 The Korean Society of Toxicology https://doi.org/10.5487/TR.2015.31.2.105
CARBON BLACK	Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported **WARNING:** This substance has been classified by the IARC as Group 2B: Possi Humans.
METHYL ETHYL KETOXIME	Mammalian lymphocyte mutagen *Huls Canada ** Merck For methyl ethyl ketoxime (MEKO) **Carcinogenicity:** Increased incidences of liver tumours were observed in rat an and there was also an increased incidence of mammary gland tumours in female

	only seen at mid- and/or high concentrations of MEKO. Consideration of the ava genotoxicity indicate that MEKO is not likely to be genotoxic. Accordingly, althou tumours is not fully elucidated, the tumours observed are not considered to have interaction with genetic material. The European Commission (2000) considered that a possible mechanism for the liver tumours in male rats and mice was the metabolism of MEKO to a carcinoge sulfotransferase. The sex and organ specificity of tumour formation correlated w activity of this enzyme in male rodents. **Genotoxicity:** The *in vitro* and *in vivo* genotoxicity results for MEKO were mostly *vivo* study that utilized inhalation exposure and was found to be negative for DN Therefore, based on the available data, MEKO appears to lack mutagenic poten **Repeat dose toxicity:** Non-neoplastic effects were also observed in the nasal c inhalation studies of short-term through to chronic exposure. Also, repeated dos exposure showed effects in the spleen, liver and kidney of rats as well as haema and rabbits. **Reproductive toxicity**: In a one-generation oral rat study, the LOAEL for reproc mg/kg-bw per day, the highest dose, based on a statistically significant decrease (%) , whereas no treatment-related effects on reproductive parameters were obs study in which rats were dosed by gavage at 0-200 mg/kg-bw per day In both th generation rat studies, a parental LOAEL of 10 mg/kg-bw per day, the lowest do based on histopathological effects in the spleen and liver (and in the kidney in th **Developmental toxicity:** Teratogenicity was not observed in pregnant rats and MEKO during gestation. The lowest oral LOAEL for developmental toxicity was 4 highest dose, based on abortions in 3 of 10 adult females in pregnant rabbits do gestation . The lowest oral LOAEL for maternal toxicity was 10 mg/kg-bw per da (increased reticulocytes and methaemoglobin) in rabbits dosed at 0-80 mg/kg-bw developmental study
CALCIUM CARBONATE & VINYLTRIS(METHYLETHYLKETOXIME)SILANE & TOLUENE	The material may cause skin irritation after prolonged or repeated exposure and dermatitis (nonallergic). This form of dermatitis is often characterised by skin red swelling the epidermis. Histologically there may be intercellular oedema of the sp intracellular oedema of the epidermis.
VINYLTRIS(METHYLETHYLKETOXIME)SILANE & METHYL ETHYL KETOXIME	The following information refers to contact allergens as a group and may not be Contact allergies quickly manifest themselves as contact eczema, more rarely as oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymph the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve anti reactions. The significance of the contact allergen is not simply determined by its distribution of the substance and the opportunities for contact with it are equally sensitising substance which is widely distributed can be a more important allerge sensitising potential with which few individuals come into contact. From a clinical are noteworthy if they produce an allergic test reaction in more than 1% of the po
VINYLTRIS(METHYLETHYLKETOXIME)SILANE & 3-[METHYLBIS[(1-METHYLETHENYL)OXY]SILYL]PROPOXY] ETHOXYLATED & CARBON BLACK	No significant acute toxicological data identified in literature search.

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

Threebond 1217H

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

dimethylsiloxane, hydroxy-terminated

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

calcium carbonate

Endpoint	Test Duration (hr)	Species	V
NOEC(ECx)	1h	Fish	4
LC50	96h	Fish	>
EC50	72h	Algae or other aquatic plants	>

silica amorphous, fumed

Endpoint	Test Duration (hr)	Species	V
NOEC(ECx)	24h	Crustacea	>

vinyltris(methylethylketoxime)silane

Endpoint	Test Duration (hr)	Species
NOEC(ECx)	72h	Algae or other aquatic plants
EC50	72h	Algae or other aquatic plants
LC50	96h	Fish
EC50	48h	Crustacea

toluene

Endpoint	Test Duration (hr)	Species	V
LC50	96h	Fish	5
EC50	72h	Algae or other aquatic plants	1
EC50	48h	Crustacea	3
NOEC(ECx)	168h	Crustacea	0
EC50	96h	Algae or other aquatic plants	>

3-[methylbis[(1-methylethenyl)oxy]silyl]propoxy] ethoxylated	**Endpoint**	**Test Duration (hr)**	**Species**
	Not Available	Not Available	Not Available
carbon black	**Endpoint**	**Test Duration (hr)**	**Species**
	LC50	96h	Fish
	EC50	72h	Algae or other aquatic plants
	EC50	48h	Crustacea
	NOEC(ECx)	24h	Crustacea
methyl ethyl ketoxime	**Endpoint**	**Test Duration (hr)**	**Species**
	BCF	1008h	Fish
	NOEC(ECx)	72h	Algae or other aquatic plants
	EC50	72h	Algae or other aquatic plants
	EC50	48h	Crustacea
	LC50	96h	Fish

Legend:	*Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Inform US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*

Toxic to bees.
For siloxanes:
Environmental fate:
It is well accepted that polydimethylsiloxane (PDMS) fluids become permanent residents of sediment but should not exert adverse environmental effects. PDMS in intimate contact with many soils undergo siloxane bond redistribution and hydrolysis Therefore, it is highly likely that substituted polymethylsiloxanes will undergo similar reactions, and this reactivity may prevent suitable adsorption data being obtained.
Silicone fluids are very surface active because the flexible siloxane linkages permit alignment of the hydrophobic methyl substituents towards the non-polar phase, and of the polysiloxane backbone towards the polar phase. The polar medium is generally water, and a polar media to which polydimethylsiloxanes become attached may be textiles, sewage sludge, hair, algae, sediment etc. In aqueous environments, polydimethylsiloxanes are adsorbed onto sedimenting particles. Also, in the presence of nitrate ions, which exist at various concentrations in the environment, short chain siloxanes are photodegraded to the level of silicate within days
The stability of the siloxanes, desirable from a technical point of view, makes the siloxanes very persistent, and once released to the environment the siloxanes remain for many years.
The main source of releases of siloxanes to the air is volatile siloxanes used in cosmetics, wax, polishes, and to a minor extent in several other applications. the volatile siloxanes may account for a significant part of the siloxanes used for cosmetics.
Non-volatile silicone fluids used in cosmetics, wax, polishes, cleaning products and for textile applications (softeners) will to a large extent end up in wastewater and be directed to wastewater treatment plants.
The cyclic siloxanes and small-chain linear siloxanes are bioconcentrated (bioconcentration factors for long-chained siloxanes have not been assessed). The estimated bioconcentration factors (BCF) of the small siloxanes range from 340 for HMDS to 40,000 for a phenylated trisiloxane (phenyl trimethicone). The small phenylated siloxanes seem to have very high BCF, and model estimates indicate that these substances are the most toxic for aquatic organisms.
PBT profiler screening
In order to make a first comparison between the substances as to persistence, bioaccumulation and toxicity, the substances were screened using the PBT profiler developed by U.S. EPA (U.S. EPA 2003). The profiler uses a procedure to predict persistence, bioaccumulation, and toxicity of organic chemicals on the basis of the chemical structure and physical parameters of the substances combined with experimental parameters for substance with a similar structure, using a QSAR approach.

The results for six members of the siloxane family predict the highest bioconcentration factors for the two phenyl siloxanes, one order of magnitudes higher than the values for the cyclic siloxanes and two orders of magnitudes higher than the values for the small linear methyl siloxanes. The predicted toxicity is as well significantly higher (lowest ChV values) for the phenyl siloxanes. The predicted half-life is nearly the same for all substances.
Using U.S. EPA's criteria, the screening indicates that all substances are of high concern as to environmental toxicity, and that the phenyl siloxanes are considered very bioaccumulative.
Ecotoxicity:
The environmental fate and effects of volatile methylsiloxanes (mainly cyclosiloxanes) and polydimethylsiloxane (PDMS) have been reported:
For octamethylcyclosiloxane:
Fish acute LC50 (14 day):: rainbow trout 10 ug/l; sheepshead minnow >6.3 ug/l
Daphnia magna acute EC50 (48 h): >15 ug/l; NOEC 15 ug/l
Mysid shrimp acute LC50 (96 h): >9.1 ug/l; NOEC 9.1 ug/l
For PDMS
Daphnia magna NOEC 572 mg/kg
Physical effects such as surface entrapment have been observed when testing aquatic invertebrates in clean laboratory water, but similar effects are not expected in natural environments where a large variety of other surfaces provide opportunities for deposition
Calcium provides an important link between tectonics, climate and the carbon cycle. In the simplest terms, uplift of mountains exposes Ca-bearing rocks to chemical weathering and releases Ca^{2+} into surface water. This Ca^{2+} eventually is transported to the ocean where it reacts with dissolved CO_2 to form limestone. Some of this limestone settles to the sea floor where it is incorporated into new rocks. Dissolved CO_2, along with carbonate and bicarbonate ions, are referred to as dissolved inorganic carbon (DIC).
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 Amorphous Silica: Amorphous silica is chemically and biologically inert. It is not biodegradable.
Aquatic Fate: Due to its insolubility in water there is a separation at every filtration and sedimentation process. On a global scale, the level of man-made synthetic amorphous silicas (SAS) represents up to 2.4% of the dissolved silica naturally present in the aquatic environment and untreated SAS have a relatively low water solubility and an extremely low vapour pressure. Biodegradability in sewage treatment plants or in surface water is not applicable to inorganic substances like SAS.
Terrestrial Fate: Crystalline and/or amorphous silicas are common on the earth in soils and sediments, and in living organisms (e.g. diatoms), but only the dissolved form is bioavailable. On the basis of these properties it is expected that SAS released into the environment will be distributed mainly into soil/sediment. Surface treated silica will be wetted then adsorbed onto soils and sediments.
Atmospheric Fate: SAS is not expected to be distributed into the air if released.
Ecotoxicity: SAS is not toxic to environmental organisms (apart from physical desiccation in insects). SAS presents a low risk for adverse effects to the environment.
For Silica:
Environmental Fate: Most documentation on the fate of silica in the environment concerns dissolved silica, in the aquatic environment, regardless of origin, (man-made or natural), or structure, (crystalline or amorphous).
Terrestrial Fate: Silicon makes up 25.7% of the Earth's crust, by weight, and is the second most abundant element, being exceeded only by oxygen. Silicon is not found free in nature, but occurs chiefly as the oxide and as silicates. Once released into the environment, no distinction can be made between the initial forms of silica.
Aquatic Fate: At normal environmental pH, dissolved silica exists exclusively as monosilicic acid. At pH 9.4, amorphous silica is highly soluble in water. Crystalline silica, in the form of quartz, has low solubility in water. Silicic acid plays an important role in the biological/geological/chemical cycle of silicon, especially in the ocean. Marine organisms such as diatoms, silicoflagellates and radiolarians use silicic acid in their skeletal structures and their skeletal remains leave silica in sea sediment
Ecotoxicity: Silicon is important to plant and animal life and is practically non-toxic to fish including zebrafish, and Daphnia magna water fleas.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
toluene	LOW (Half-life = 28 days)	LOW (Half-life = 4.33 days)
methyl ethyl ketoxime	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
toluene	LOW (BCF = 90)
methyl ethyl ketoxime	LOW (BCF = 5.8)

Mobility in soil

Ingredient	Mobility
toluene	LOW (KOC = 268)
methyl ethyl ketoxime	LOW (KOC = 130.8)

SECTION 13 Disposal considerations

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 cont 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 • 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
dimethylsiloxane, hydroxy-terminated	Not Available
calcium carbonate	Not Available
silica amorphous, fumed	Not Available
vinyltris(methylethylketoxime)silane	Not Available
toluene	Not Available
3-[methylbis[(1-methylethenyl)oxy]silyl]propoxy] ethoxylated	Not Available
carbon black	Not Available
methyl ethyl ketoxime	Not Available

Transport in bulk in accordance with the IGC Code

Product name	Ship Type
dimethylsiloxane, hydroxy-terminated	Not Available
calcium carbonate	Not Available
silica amorphous, fumed	Not Available
vinyltris(methylethylketoxime)silane	Not Available
toluene	Not Available
3-[methylbis[(1-methylethenyl)oxy]silyl]propoxy] ethoxylated	Not Available
carbon black	Not Available
methyl ethyl ketoxime	Not Available

SECTION 15 Regulatory information

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

dimethylsiloxane, hydroxy-terminated is found on the following regulatory lists

- Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C
- Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
- Australian Inventory of Industrial Chemicals (AIIC)

calcium carbonate is found on the following regulatory lists

- Australian Inventory of Industrial Chemicals (AIIC)
- International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

silica amorphous, fumed is found on the following regulatory lists

- Australian Inventory of Industrial Chemicals (AIIC)
- International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

vinyltris(methylethylketoxime)silane is found on the following regulatory lists

- Australian Inventory of Industrial Chemicals (AIIC)

toluene is found on the following regulatory lists

- Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
- Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
- Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
- Australian Inventory of Industrial Chemicals (AIIC)
- Chemical Footprint Project - Chemicals of High Concern List
- International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

3-[methylbis[(1-methylethenyl)oxy]silyl]propoxy] ethoxylated is found on the following regulatory lists

- Australian Inventory of Industrial Chemicals (AIIC)

carbon black is found on the following regulatory lists

- Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
- Australian Inventory of Industrial Chemicals (AIIC)
- Chemical Footprint Project - Chemicals of High Concern List
- International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
- International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
- International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

methyl ethyl ketoxime is found on the following regulatory lists

- Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
- Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
- Australian Inventory of Industrial Chemicals (AIIC)
- Chemical Footprint Project - Chemicals of High Concern List

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (3-[methylbis[(1-methylethenyl)oxy]silyl]propoxy] ethoxylated)
Canada - NDSL	No (dimethylsiloxane, hydroxy-terminated; silica amorphous, fumed; vinyltris(methylethylketoxime)silane; methyl ethyl ketoxime)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (dimethylsiloxane, hydroxy-terminated; 3-[methylbis[(1-methylethenyl)oxy]silyl]propoxy] ethoxylated)
Japan - ENCS	No (3-[methylbis[(1-methylethenyl)oxy]silyl]propoxy] ethoxylated)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (3-[methylbis[(1-methylethenyl)oxy]silyl]propoxy] ethoxylated)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (vinyltris(methylethylketoxime)silane; 3-[methylbis[(1-methylethenyl)oxy]silyl]propoxy] ethoxylated)
Vietnam - NCI	No (3-[methylbis[(1-methylethenyl)oxy]silyl]propoxy] ethoxylated)
Russia - FBEPH	No (3-[methylbis[(1-methylethenyl)oxy]silyl]propoxy] ethoxylated)

National Inventory	Status
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exem registration.*

SECTION 16 Other information

Revision Date	07/28/2021
Initial Date	06/30/2020

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	11/22/2020	Physical and chemical properties - Appearance, Ecological Information - Environmental, on ingredients - Ingredients, Identification of the substance / mixture and of the company Information, Identification of the substance / mixture and of the company / undertaking -
3.1	07/27/2021	Toxicological information - Acute Health (eye), Toxicological information - Acute Health (information - Acute Health (skin), Toxicological information - Acute Health (swallowed), F properties - Appearance, Toxicological information - Chronic Health, Hazards identificati considerations - Disposal, Exposure controls / personal protection - Engineering Control, Environmental, Firefighting measures - Fire Fighter (fire/explosion hazard), Firefighting m fighting), First Aid measures - First Aid (skin), First Aid measures - First Aid (swallowed), Handling Procedure, Composition / information on ingredients - Ingredients, Stability and Condition, Exposure controls / personal protection - Personal Protection (other), Exposu protection - Personal Protection (hands/feet), Accidental release measures - Spills (majo measures - Spills (minor), Handling and storage - Storage (storage requirement), Handli (suitable container), Identification of the substance / mixture and of the company / under Information, Identification of the substance / mixture and of the company / undertaking - information - Transport, Transport Information, Identification of the substance / mixture a undertaking - Use, Name

Other information

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

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