Dunlop Bond Coat

Ardex (Ardex Australia)

Chemwatch Hazard Alert Code: **4**

Chemwatch: **5464-24**
Version No: **3.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **10/03/2023**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	Dunlop Bond Coat
Chemical Name	Not Applicable
Synonyms	single part water based primer
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	As water based primer to various substrates for improving adhesion of adhesives and levellers.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**Ardex (Ardex Australia)**
Address	20 Powers Road Seven Hills NSW 2147 Australia
Telephone	1800 224 070
Fax	1300 780 102
Website	www.ardexaustralia.com
Email	technicalservices@ardexaustralia.com

Emergency telephone number

Association / Organisation	**Ardex (Ardex Australia)**
Emergency telephone numbers	1800 224 070 (Mon-Fri, 9am-5pm)
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Chemwatch Hazard Ratings

		Min	Max
Flammability	1		
Toxicity	2		
Body Contact	2		
Reactivity	0		
Chronic	4		

0 = Minimum
1 = Low
2 = Moderate
3 = High
4 = Extreme

Poisons Schedule	Not Applicable
Classification [1]	Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2A, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Carcinogenicity Category 1A, Reproductive Toxicity Category 1B, Specific Target Organ Toxicity - Repeated Exposure Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

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Label elements

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

Hazard statement(s)

H317	May cause an allergic skin reaction.
H319	Causes serious eye irritation.
H335	May cause respiratory irritation.
H350	May cause cancer.
H360D	May damage the unborn child.
H373	May cause damage to organs through prolonged or repeated exposure.
H412	Harmful to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P273	Avoid release to the environment.
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

P308+P313	IF exposed or concerned: Get medical advice/ attention.
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.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
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.
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 with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
14807-96-6	10-30	talc
7727-43-7	10-30	barium sulfate
1332-58-7	1-10	kaolin
7631-86-9	<5	silica amorphous
2530-83-8	<1	gamma-glycidoxypropyltrimethoxysilane
13463-67-7	<0.1	titanium dioxide
14808-60-7	<0.1	silica crystalline - quartz
330-54-1	<0.1	diuron
2634-33-5	<0.1	1,2-benzisothiazoline-3-one
2682-20-4	<0.1	2-methyl-4-isothiazolin-3-one
26530-20-1	<0.1	2-octyl-4-isothiazolin-3-one

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CAS No	%[weight]	Name
Not Available	30-60	Ingredients determined not to be hazardous
Legend:	*1. Classified by Chemwatch; 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 or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.
for diuron:
- Symptomatic and supportive action is indicated.
- Methaemoglobinaemia is possible
- if compound is hydrolysed in vivo to aniline.
- Methaemoglobinaemia causes cyanosis. Reversion of methaemoglobin to haemoglobin is spontaneous after removal from exposure, so moderate degrees of cyanosis need be treated only by supportive measures such as bed rest and oxygen inhalation.
- Thorough cleansing of the entire contaminated area of the body, including the scalp and nails is of the utmost importance.

- After ingestion of barium acid salts, severe gastro-intestinal irritation followed by muscle twitching, progressive flaccid paralysis and severe hypokalaemia and hypertension, occurs.
- Respiratory failure, renal failure and occasional cardiac dysrhythmias may result from an acute ingestion.
- Use sodium sulfate as a cathartic. Add 5-10 gm of sodium sulfate to lavage solution or as fluid supplement to Ipecac syrup (the sulfate salt is not absorbed)
- Monitor cardiac rhythm and serum potassium closely to establish the trend over the first 24 hours. Large doses of potassium may be needed to correct the hypokalaemia.
- Administer generous amounts of fluid replacement but monitor the urine and serum for evidence of renal failure. [Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 Firefighting measures

Extinguishing media

The product contains a substantial proportion of water, therefore there are no restrictions on the type of extinguishing media which may be used. Choice of extinguishing media should take into account surrounding areas.
Though the material is non-combustible, evaporation of water from the mixture, caused by the heat of nearby fire, may produce floating layers of combustible substances.
In such an event consider:
- foam.
- dry chemical powder.
- carbon dioxide.

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.

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 full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ Avoid spraying water onto liquid pools. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	▸ 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.

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	▸ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO2) nitrogen oxides (NOx) sulfur oxides (SOx) silicon dioxide (SiO2) metal oxides other pyrolysis products typical of burning organic material. Decomposes at high temperatures to produce barium oxide. Barium oxide is strongly alkaline and, upon contact with water, is exothermic. When barium oxide reacts with oxygen to give a peroxide, there is a fire and explosion risk. 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	▸ Remove all ignition sources. ▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Control personal contact with the substance, by using protective equipment. ▸ Contain and absorb spill with sand, earth, inert material or vermiculite. ▸ Wipe up. ▸ Place in a suitable, labelled container for waste disposal.
Major Spills	Moderate hazard. ▸ Clear area of personnel and move upwind. ▸ 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 course. ▸ No smoking, naked lights or ignition sources. ▸ Increase ventilation. ▸ Stop leak if safe to do so. ▸ Contain spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Absorb remaining product with sand, earth or vermiculite. ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ 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	▸ **DO NOT** allow clothing wet with material to stay in contact with skin ▸ 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. ▸ 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	▸ Avoid strong acids, bases.

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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	talc	Talc, (containing no asbestos fibres)	2.5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	barium sulfate	Barium sulphate	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	kaolin	Kaolin	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Diatomaceous earth (uncalcined)	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Silica gel	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Precipitated silica	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Fume (thermally generated)(respirable dust)	2 mg/m3	Not Available	Not Available	(e) Containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica, fused	0.05 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Fumed silica (respirable dust)	2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	titanium dioxide	Titanium dioxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica crystalline - quartz	Silica - Crystalline: Quartz (respirable dust)	0.05 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	diuron	Diuron	10 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
barium sulfate	15 mg/m3	170 mg/m3	990 mg/m3
silica amorphous	18 mg/m3	200 mg/m3	1,200 mg/m3
silica amorphous	18 mg/m3	100 mg/m3	630 mg/m3
silica amorphous	120 mg/m3	1,300 mg/m3	7,900 mg/m3
silica amorphous	45 mg/m3	500 mg/m3	3,000 mg/m3
silica amorphous	18 mg/m3	740 mg/m3	4,500 mg/m3
gamma-glycidoxypropyltrimethoxysilane	9.3 mg/m3	100 mg/m3	230 mg/m3
titanium dioxide	30 mg/m3	330 mg/m3	2,000 mg/m3
silica crystalline - quartz	0.075 mg/m3	33 mg/m3	200 mg/m3

Ingredient	Original IDLH	Revised IDLH
talc	1,000 mg/m3	Not Available
barium sulfate	Not Available	Not Available
kaolin	Not Available	Not Available
silica amorphous	3,000 mg/m3	Not Available
gamma-glycidoxypropyltrimethoxysilane	Not Available	Not Available
titanium dioxide	5,000 mg/m3	Not Available
silica crystalline - quartz	25 mg/m3 / 50 mg/m3	Not Available
diuron	Not Available	Not Available
1,2-benzisothiazoline-3-one	Not Available	Not Available
2-methyl-4-isothiazolin-3-one	Not Available	Not Available
2-octyl-4-isothiazolin-3-one	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
gamma-	E	≤ 0.1 ppm

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Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
glycidoxypropyltrimethoxysilane		
1,2-benzisothiazoline-3-one	E	≤ 0.01 mg/m³
2-methyl-4-isothiazolin-3-one	D	> 0.01 to ≤ 0.1 mg/m³
2-octyl-4-isothiazolin-3-one	E	≤ 0.1 ppm
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

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.

Individual protection measures, such as personal protective equipment

Eye and face protection

- Safety glasses with side shields.
- Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]
- 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].

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.

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
· frequency and duration of contact,

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	· chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
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:
Dunlop Bond Coat

Material	CPI
BUTYL	A
NEOPRENE	A
VITON	A
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
PE	C
PE/EVAL/PE	C
PVA	C
PVC	C
TEFLON	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 BKAX-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	BKAX-AUS P2	-	BKAX-PAPR-AUS / Class 1 P2
up to 50 x ES	-	BKAX-AUS / Class 1 P2	-
up to 100 x ES	-	BKAX-2 P2	BKAX-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	Grey liquid; mixes with water.

Physical state	Liquid	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available

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Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Applicable
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available
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 Applicable	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Applicable	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Applicable	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ 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 system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Fully cured, vulcanised styrene/ butadiene rubber (SBR) products have a low toxicity. Dusts may cause temporary mild irritation and coughing. Uncured (unvulcanised) SBR may contain styrene, butadiene residues, solvents and processing agents, which may have harmful effects. These chemicals may be inhaled in the form of vapours emitted from the solid material or as components of dust particles. SBR may emit irritating even toxic decomposition products if overheated or burned. Inhalation of dusts, generated by the material during the course of normal handling, may be damaging to the health of the individual. Barium fumes are respiratory irritants. Over-exposure to barium dusts and fume may result in rhinitis, frontal headache, wheezing, laryngeal spasm, salivation and anorexia. Long term effects include nervous disorders and adverse effects on the heart, circulatory system and musculature. Heavy exposures may result in a benign pneumoconiosis.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	Limited 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. Irritation and skin reactions are possible with sensitive skin 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. Repeated or prolonged eye contact may cause inflammation (similar to windburn) characterised by a temporary redness of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	On the basis of epidemiological data, it has been concluded that prolonged inhalation of the material, in an occupational setting, may produce cancer in humans. Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. 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

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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.

Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.
Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.
There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of:
- clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.
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
The health hazards associated with bentonite, kaolin, and common clay, which are commercially important clay products, as well as the related phyllosilicate minerals montmorillonite, kaolinite, and illite, have an extensive literature. Fibrous clay minerals, such as sepiolite, attapulgite, and zeolites, have a separate literature.
The biological effects of clay minerals are influenced by their mineral composition and particle size. The decreasing rank order of the potencies of quartz, kaolinite, and montmorillonite to produce lung damage is consistent with their known relative active surface areas and surface chemistry.
Clays are chemically all described as aluminosilicates; these are further classified as bentonite, kaolin and common clays.
Bentonite is a rock formed of highly colloidal and plastic clays composed mainly of montmorillonite, a clay mineral of the smectite group.
Kaolin or china clay is a mixture of different minerals. Its main component is kaolinite; in addition, it frequently contains quartz, mica, feldspar, illite, and montmorillonite.
The main components of common clay and shale are illite and chlorite. Illite is also a component of ball clays. Illite closely resembles micas,
From the limited data available from studies on bentonite-exposed persons, retained montmorillonite appears to effect only mild nonspecific tissue changes, which are similar to those that have been described in the spectrum of changes of the “small airways mineral dust disease” (nodular peribronchiolar dust accumulations containing refractile material [montmorillonite] in association with limited interstitial fibrosis). In some of the studies, radiological abnormalities have also been reported
Long-term occupational exposures to bentonite dust may cause structural and functional damage to the lungs. However, available data are inadequate to conclusively establish a dose-response relationship or even a cause-and-effect relationship due to limited information on period and intensity of exposure and to confounding factors, such as exposure to silica and tobacco smoke.
Long-term exposure to kaolin may lead to a relatively benign pneumoconiosis, in an exposure-related fashion. known as kaolinosis. Deterioration of lung function has been observed only in cases with prominent radiological alterations. Based on data from china clay workers in the United Kingdom, it can be very roughly estimated that kaolin is at least an order of magnitude less potent than quartz.. Clearcut deterioration of respiratory function and related symptoms have been reported only in cases with prominent radiological findings.
The composition of the clay - i.e., quantity and quality of minerals other than kaolinite — is an important determinant of the effects. Bentonite, kaolin, and other clays often contain quartz, and exposure to quartz is causally related to silicosis and lung cancer. Statistically significant increases in the incidence of or mortality from chronic bronchitis and pulmonary emphysema have been reported after exposure to quartz.
The removal of clay particles from the lungs takes place by solubilisation in situ and by physical clearance.
In humans, there was a rapid initial clearance of 8% and 40% of aluminosilicate particles that were, respectively, 1.9 and 6.1 um in aerodynamic diameter from the lung region over 6 days. Thereafter, 4% and 11% of the two particle sizes were removed following a halftime of 20 days, and the rest with half-times of 330 and 420 days.
Ultrafine particles (<100 nm) have a high deposition in the nasal area; they can penetrate the alveolar/capillary barrier. Epidemiological studies have indicated an increase in morbidity and mortality associated with an increase in airborne particulate matter, particularly in the ultrafine size range
An important determinant of the toxicity of clays is the content of quartz. The presence of quartz in the clays studied hampers reliable independent estimation of the fibrogenicity of other components of clays.
Single intratracheal injection into rodents of bentonite and montmorillonite with low content of quartz produced dose- and particle size-dependent cytotoxic effects, as well as transient local inflammation, the signs of which included oedema and, consequently, increased lung weight. After high doses of intratracheal kaolin (containing 8-65% quartz), fibrosis has been described in some studies, whereas at lower kaolin doses, no fibrosis has been observed in the few available studies.
There are limited data on the effects of multiple exposures of experimental animals to montmorillonite or bentonite. Mice maintained on diets containing 10% or 25% bentonite but otherwise adequate to support normal growth displayed slightly reduced growth rates, whereas mice maintained on a similar diet with 50% bentonite showed minimal growth and developed fatty livers and eventually fibrosis of the liver and benign hepatomas.
In vitro studies of the effects of bentonite on a variety of mammalian cell types usually indicated a high degree of cytotoxicity. Concentrations below 1.0 mg/ml of bentonite and montmorillonite particles less than 5 um in diameter caused membrane damage and even cell lysis, as well as functional changes in several types of cells.
No adequate studies are available on the carcinogenicity of bentonite. In an inhalation study and in a study using intrapleural injection, kaolin did not induce tumours in rats. No studies are available on the genotoxicity of clays.
Single, very limited studies did not demonstrate developmental toxicity in rats after oral exposure to bentonite or kaolin.
Chronic dust inhalation of kaolin, as experienced in mineral extraction, has caused kaolinosis with heavy lung marking, emphysema, and nodular pneumoconiosis.
Evidence of kaolinosis (pneumoconiosis) was found in 9% of 553 Cornish china clay workers who had been exposed to kaolin dust for periods exceeding 5 years, whereas no kaolinosis was observed in workers exposed for less than 5 years. Workers in more heavily exposed jobs of milling, bagging and loading showed a prevalence of kaolinosis rising from 6% in those within between 5 and 15 years exposure to 23% in those exposed for more than 15 years. Workers intermittently and less heavily exposed in the older, outdated drying plants required 25 years of massive exposure before reaching the highest prevalence of 17%. Massive fibrosis was seen in four workers, and six workers needed antituberculosis chemotherapy. Preventative measures instituted include preemployment chest examination and approaches to the problem of dust control.
Sheer, G.; Brit. Jnl. Ind. Med. 21, pp 218-225, 1964
Prolonged inhalation of high concentrations of magnesite (magnesium carbonate) dust caused pulmonary deposition and retention. Roasted magnesite (magnesium oxide) produced a greater degree of fibrosis than did crude magnesite. No cases of human systemic poisoning due to exposure to magnesite have been recorded. Pneumoconiosis was found in about 2% of workers exposed to high concentrations of dust from crude or roasted magnesite that also contained 1-3% silicon dioxide. Exposure periods ranged from 6-20 years. This condition occurred mainly in workers exposed to roasted (calcined) magnesite. The pneumoconiosis appeared to be "benign" and was often associated with chronic bronchitis and lung emphysema.
In other reports the severity of the pneumoconiosis was associated with the crystalline silica content of the dust or in a case of magnesium carbonate used in insulating materials, the severity of the disease depended on the asbestos content.
Complaints of coughing are rare amongst magnesite workers, and more frequent among dianase and grog (crushed refractory materials)

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workers.
Airborne dust concentrations were lowest in dianase facilities but crystalline silica was high. Chronic bronchitis then, appears to increase where concentrations of crystalline silica are highest
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.
Chronic effects of exposure to diuron may initially include skin irritation, or blurring of vision, liver enlargement; spleen and thyroid effects; red blood cell destruction; or reduction of the blood's oxygen carrying capacity with cyanosis (bluish discolourisation), weakness or shortness of breath by formation of methemoglobin. Significant skin permeation after contact appears unlikely. There are no reports of human sensitisation to diuron.
At 2500 ppm diuron in the diet for 2 years, rats and dogs showed growth retardation, slight anaemia, presence of abnormal pigmentation, increased erythropoiesis and splenic haemosiderosis.
Workers exposed to barium compounds have been reported to show an increased incidence of hypertension, irritation of the respiratory system, and damage to the spleen, liver and bone marrow. Long term exposure to some barium compounds (especially inorganic species) may produce a condition known as baritosis, a form of benign pneumoconiosis. X-ray may show this when no other abnormal signs are present.
Symptoms of pneumoconiosis may include a progressive dry cough, shortness of breath on exertion, increased chest expansion, weakness and weight loss. As the disease progresses the cough produces a stringy mucous, vital capacity decreases further and shortness of breath becomes more severe. Pneumoconiosis is the accumulation of dusts in the lungs and the tissue reaction in its presence. Barium sulfate produces noncollagenous pneumoconiosis identified by minimal stromal reaction, consisting mainly of reticulin fibres, an intact alveolar architecture and is potentially reversible. Miners of ores containing barium sulfate do not show symptoms, abnormal physical signs, an incapacity to work, diminished lung function, an increased likelihood of developing pulmonary or other bronchial infections or other thoracic disease despite the fact that particulate matter may have been retained in the lungs for many years.
No changes in mortality were observed in rats chronically exposed to doses as high as 60 mg barium/kg/day as barium chloride in the drinking water. An increase in mortality, attributable to nephropathy, was observed in mice chronically exposed to 160 mg barium/kg/day as barium chloride in drinking water; the number of deaths was similar to controls in mice exposed to 75 mg barium/kg/day. In male mice exposed to 0.95 mg barium/kg/day as barium acetate in drinking water, a significant decrease in longevity (defined as average lifespan of the last five surviving animals) was observed; however, no significant differences in mean lifespan were observed. Similarly, lifespan was not significantly altered in female mice exposed to 0.95 mg barium/kg/day or male or female rats exposed to 0.7 mg barium/kg/day as barium acetate in drinking water.
The potential for barium to induce reproductive and developmental effects has not been well investigated. Decreases in the number of sperm and sperm quality and a shortened estrous cycle and morphological alterations in the ovaries were observed in rats exposed to 2.2 mg barium/m3 and higher in air for an intermediate duration. Interpretation of these data is limited by the poor reporting of the study design and results, in particular, whether the incidence was significantly different from controls. In general, oral exposure studies have not found morphological alterations in reproductive tissues of rats or mice exposed to 180 or 450 mg barium/kg/day, respectively, as barium chloride in drinking water for an intermediate duration. Additionally, no significant alterations in reproductive performance was observed in rats or mice exposed to 200 mg barium/kg/day as barium chloride in drinking water. Decreased pup birth weight and a nonsignificant decrease in litter size have been observed in the offspring of rats exposed to 180/200 mg barium/kg/day as barium chloride in drinking water prior to mating.
Several studies have examined the carcinogenic potential of barium following oral exposure and did not find significant increases in the tumour incidence.
Overexposure to the breathable dust may cause coughing, wheezing, difficulty in breathing and impaired lung function. Chronic symptoms may include decreased vital lung capacity and chest infections. Repeated exposures in the workplace to high levels of fine-divided dusts may produce a condition known as pneumoconiosis, which is the lodgement of any inhaled dusts in the lung, irrespective of the effect. This is particularly true when a significant number of particles less than 0.5 microns (1/50000 inch) are present. Lung shadows are seen in the X-ray. Symptoms of pneumoconiosis may include a progressive dry cough, shortness of breath on exertion, increased chest expansion, weakness and weight loss. As the disease progresses, the cough produces stringy phlegm, vital capacity decreases further, and shortness of breath becomes more severe. Other signs or symptoms include changed breath sounds, reduced oxygen uptake during exercise, emphysema and rarely, pneumothorax (air in the lung cavity).
Removing workers from the possibility of further exposure to dust generally stops the progress of lung abnormalities. When there is high potential for worker exposure, examinations at regular period with emphasis on lung function should be performed.
Inhaling dust over an extended number of years may cause pneumoconiosis, which is the accumulation of dusts in the lungs and the subsequent tissue reaction. This may or may not be reversible.

Dunlop Bond Coat	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

talc	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50: >2.1 mg/l4h[1]	Skin (human): 0.3 mg/3d-I mild
	Oral (Rat) LD50: >5000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

barium sulfate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Not Available
	Oral (Mouse) LD50; >3000 mg/kg[2]	

kaolin	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

silica amorphous	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit): non-irritating ** [Grace]
	Inhalation(Rat) LC50: >0.09<0.84 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >1000 mg/kg[1]	Skin (rabbit): non-irritating *
		Skin: no adverse effect observed (not irritating)[1]

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gamma-glycidoxypropyltrimethoxysilane	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 4247.9 mg/kg[2]	Not Available
	Inhalation(Rat) LC50: >5.3 mg/l4h[1]	
	Oral (Rat) LD50: 7010 mg/kg[2]	
titanium dioxide	**TOXICITY**	**IRRITATION**
	dermal (hamster) LD50: >=10000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50: >2.28 mg/l4h[1]	Skin (human): 0.3 mg /3D (int)-mild *
	Oral (Rat) LD50: >=2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
silica crystalline - quartz	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 500 mg/kg[2]	Not Available
diuron	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50: >5.05 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: 1017 mg/kg[2]	
1,2-benzisothiazoline-3-one	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: adverse effect observed (irreversible damage)[1]
	Oral (Rat) LD50: 454 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
2-methyl-4-isothiazolin-3-one	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: 242 mg/kg[1]	Eye: adverse effect observed (irreversible damage)[1]
	Inhalation(Rat) LC50: 0.1 mg/l4h[1]	Skin: adverse effect observed (corrosive)[1]
	Oral (Rat) LD50: 120 mg/kg[1]	
2-octyl-4-isothiazolin-3-one	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 311 mg/kg[2]	Eye (rabbit): 0.5% non irritant
	Oral (Rat) LD50: 248 mg/kg[2]	Eye (rabbit): 45% conc CORROSIVE
		Eye (rabbit): 5% conc moderate
		Eye(rabbit):100 mg SEVERE
		Eye: adverse effect observed (irreversible damage)[1]
		Skin (rabbit): 45% conc SEVERE
		Skin (rabbit): 500 mg/24 hours
		Skin: adverse effect observed (corrosive)[1]
		Skin: adverse effect observed (irritating)[1]
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*	

TALC	For talc (a form of magnesium silicate) The overuse of talc in nursing infants has resulted in pulmonary oedema, pneumonia and death within hours of inhaling talcum powder.The powder dries the mucous membranes of the bronchioles, disrupts pulmonary clearance, clogs smaller airways. Victims display wheezing, rapid or difficult breathing, increased pulse, cyanosis, fever. Mild exposure may cause relatively minor inflammatory lung disease. Long term exposure may show wheezing, weakness, productive cough, limited chest expansion, scattered rales, cyanosis.
KAOLIN	for bentonite clays: Bentonite (CAS No. 1302-78-9) consists of a group of clays formed by crystallisation of vitreous volcanic ashes that were deposited in water. The expected acute oral toxicity of bentonite in humans is very low (LD50>15 g/kg). However, severe anterior segment inflammation, uveitis and retrocorneal abscess from eye exposure were reported when bentonite had been used as a prophypaste. In a 33 day dietary (2 and 6%) and a 90 day dietary (1, 3 and 5%) studies in chickens, no changes in behaviour, overall state, clinical and biochemical parameters and electrolytic composition of the blood. Repeat dietary administration of bentonite did not affect calcium or phosphorus metabolism. However, larger amounts caused decreased growth, muscle weakness, and death with marked changes in both calcium and phosphorus metabolism. Bentonite did not cause fibrosis after 1 year exposure of 60 mg dust (<5 um) in a rat study. However, in a second rat study, where 5 um particles were intratracheally instilled at 5, 15 and 45 mg/rat, dose-related fibrosis was observed. Bentonite clay dust is believed to be responsible for bronchial asthma in workers at a processing plant in USA. Ingestion of bentonite without adequate liquids may result in intestinal obstruction in humans. Hypokalaemia and microcytic iron-deficiency anaemia may occur in patients after repeat doses of clay. Chronic ingestion has been reported to cause myositis.
SILICA AMORPHOUS	Reports indicate high/prolonged exposures to amorphous silicas induced lung fibrosis in experimental animals; in some experiments these effects were reversible. [PATTYS] 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

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	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, 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.
GAMMA-GLYCIDOXYPROPYLTRIMETHOXYSILANE	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. 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. For gamma-glycidopropyltrimethoxysilane (GPTMS) GPTMS is subject to rapid hydrolysis, and the observed toxicity is expected to be due primarily to methanol and silanetriols. GPTMS has been tested for acute toxicity by the oral, dermal, and inhalation routes of exposure. Reported acute oral LD50s in rats range from 7010 to 16900 mg/kg bw and > 5 ml/kg bw to 22.6 ml/kg bw. The dermal LD50s are 6800 mg/kg bw and 4.0 ml/kg bw. The 4-hour inhalation LC50 was greater than 2.7 mg/L in one study and greater than 5.3 mg/L in another study. GPTMS is mildly irritating to the skin and eyes and is not a known skin sensitiser in humans or in animals. Following inhalation exposures of rats to target aerosol concentrations of 0, 75, 225 and 750 mg/m3 (actual concentrations were 0, 77, 226, 707 mg/m3 (males) and 0, 73, 226, 734 mg/m3 (females)), GPTMS in 9 repeated exposures administered over two weeks, 6 animals in the high dose group died or were sacrificed from three to five days after initiation of the study. These animals had signs of inanition but no acute tissue toxicity. At both the mid and high doses, rats exhibited some clinical signs including a dose-related decrease in body weight. Under the conditions of this study, the No Observed Adverse Effect Concentration is 225 mg/m3. Repeated exposure of rats by gavage to GPTMS doses of 40, 400 and 1000 mg/kg bw/day for 5 days/week for 4 weeks resulted in no test substance-related organ weights effects or gross or microscopic pathological changes. Under the conditions of this study, the NOAEL for the test substance was found to be 1000 mg/kg bw/day. **Genotoxicity:** GPTMS did not induce chromosomal damage in mouse bone marrow cells by gavage at doses of 500, 1670 and 5000 mg/kg bw/day, or when administered by intraperitoneal (i.p.) injection at 1600 mg/kg bw/day. However, chromosomal damage was induced in mouse bone marrow cells when administered by i.p. in water at doses of 500, 1000 and 2000 mg/kg bw/day. GPTMS induced gene mutations in bacteria. GPTMS induced gene mutations in mouse lymphoma L1578Y TK cells but did not induce forward mutations in CHO cells. GPTMS induced SCE in vitro. There are no in vivo gene mutation data. **Carcinogenicity:** GPTMS was not considered tumourigenic when applied to the clipped skin of mice (25 ul dose of 25% GPTMS in acetone) three times per week for approximately 78 weeks. Note that there was only one dose level, and this dose was relatively low. **Reproductive toxicity:** In a one-generation reproduction toxicity study in rats, no reproductive effects were observed at any of the doses tested (250, 500, or 1000 mg/kg bw/day). At 1000 mg/kg bw/day, treatment with GPTMS resulted in the following signs in parental animals: discomfort after dosing (noted for females from early/mid gestation onwards), decreased body weight gain (males), increased mean relative liver and kidney weights (noted for males and females), and histopathological effects on livers and kidneys (males). Based on these data, a NOAEL for parental animals was established at 500 mg/kg bw/day. A NOAEL for reproductive effects was established at 1000 mg/kg bw/day. **Developmental toxicity:** Three developmental studies have been conducted using GPTMS. In a rabbit study, the maternal NOAEL was 200 mg/kg bw/day and the developmental NOAEL was 400 mg/kg bw/day (the highest dose tested). In a rat study, the NOAELs for both maternal and developmental toxicity were also at the highest dose tested (1000 mg/kg bw/day). In another rat study, developmental effects were observed at the maternally toxic dose of 3000 mg/kg bw/day (again, the highest dose tested). Oxiranes (including glycidyl ethers and alkyl oxides, and epoxides) exhibit many common characteristics with respect to animal toxicology. One such oxirane is ethyloxirane; data presented here may be taken as representative. for 1,2-butylene oxide (ethyloxirane):

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	Ethyloxirane increased the incidence of tumours of the respiratory system in male and female rats exposed via inhalation. Significant increases in nasal papillary adenomas and combined alveolar/bronchiolar adenomas and carcinomas were observed in male rats exposed to 1200 mg/m3 ethyloxirane via inhalation for 103 weeks. There was also a significant positive trend in the incidence of combined alveolar/bronchiolar adenomas and carcinomas. Nasal papillary adenomas were also observed in 2/50 high-dose female rats with none occurring in control or low-dose animals. In mice exposed chronically via inhalation, one male mouse developed a squamous cell papilloma in the nasal cavity (300 mg/m3) but other tumours were not observed. Tumours were not observed in mice exposed chronically via dermal exposure. When trichloroethylene containing 0.8% ethyloxirane was administered orally to mice for up to 35 weeks, followed by 0.4% from weeks 40 to 69, squamous-cell carcinomas of the forestomach occurred in 3/49 males (p=0.029, age-adjusted) and 1/48 females at week 106. Trichloroethylene administered alone did not induce these tumours and they were not observed in control animals . Two structurally related substances, oxirane (ethylene oxide) and methyloxirane (propylene oxide), which are also direct-acting alkylating agents, have been classified as carcinogenic
TITANIUM DIOXIDE	* IUCLID Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies. For titanium dioxide: Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin. Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica. No data were available on genotoxic effects in titanium dioxide-exposed humans. Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts. Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium. Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light. **Animal carcinogenicity data** Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats. In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative. Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice. In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative. The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
SILICA CRYSTALLINE - QUARTZ	**WARNING:** For inhalation exposure ONLY: This substance has been classified by the IARC as Group 1: **CARCINOGENIC TO HUMANS** The International Agency for Research on Cancer (IARC) has classified occupational exposures to **respirable** (<5 um) crystalline silica as being carcinogenic to humans . This classification is based on what IARC considered sufficient evidence from epidemiological studies of humans for the carcinogenicity of inhaled silica in the forms of quartz and cristobalite. Crystalline silica is also known to cause silicosis, a non-cancerous lung disease. Intermittent exposure produces; focal fibrosis, (pneumoconiosis), cough, dyspnoea, liver tumours. * Millions of particles per cubic foot (based on impinger samples counted by light field techniques). NOTE : the physical nature of quartz in the product determines whether it is likely to present a chronic health problem. To be a hazard the material must enter the breathing zone as respirable particles.
DIURON	Note: Equivocal animal tumorigenic agent by RTECS criteria. NOTE: This substance may contain impurities (tetrachlorazobenzene and tetrachloroazoxybenzene). Maximum impurity levels are proscribed under various jurisdictions ADI: 0.006 mg/kg/day NOEL: 0.625 mg/kg/day Diuron is absorbed readily through the gut and lungs while uptake through the skin is more limited. It is slightly toxic to mammals but juveniles are more susceptible than adults(18). The oral LD50 in rats is 3-4 g/kg and the dermal LD50 is > 2 g/kg(19). An early study indicated that animals fed protein-deficient diets were considerably more vulnerable to diuron toxicity; rats fed a diet of 3% protein

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	were five times more sensitive to diuron. Exposure to sub-lethal doses of diuron causes formation of methaemoglobin, an abnormal form of the protein haemoglobin which carries oxygen in the blood. Diuron can decrease the number of red blood cells (RBCs), increase the number of abnormally shaped RBCs, and increase the number of white blood cells. Diuron may cause the spleen to become congested due to the increased demand to remove damaged RBCs. Increases in liver size are also observed and are indicative of the extra load placed on this organ, the body s major site of detoxification. Diuron can also cause eye and skin irritation. Diuron contains two significant impurities from the manufacturing process 3,3',4,4'-tetrachloroazobenzene (TCAB) and 3,3',4,4'-tetrachloroazoxybenzene (TCAOB), both potent 'dioxin-like substances. TCAB levels between 0.15 and 28 ppm have been found in diuron samples tested. TCAOB is present at lower levels. Both TCAB and TCAOB cause chloracne a serious skin disease **Carcinogenicity:** The US Environmental Protection Agency (EPA) has classified diuron as a 'known/likely' carcinogen since 1997 based on the results of two studies. One study on rats indicated that both males and females fed diuron had a higher incidence of bladder cancer than control animals. The male rats in this study also had a higher incidence of kidney cancer than the control animals. In a study of mice animals with higher exposures had more breast cancer **Mutagenicity:** There is conflicting evidence on whether diuron can cause mutations **Developmental Toxicity:** Rats fed relatively high levels (125 mg/kg/day) of diuron produced offspring with delayed bone formation(26) and other studies indicate that similar levels of diuron reduce birth weight. The US Toxics Release Inventory list diuron as a developmental toxin In mammals, metabolism principally occurs through hydroxylation and dealkylation. **Metabolites**. Breakdown of this compound is similar in animals, plants and soil. The first step is N-demethylation followed by ring cleavage. The main breakdown product of diuron is 3,4-dichloroaniline(3,4-DCA). The oral LD50 of 3,4-DCA in rats is around 60 mg/kg and by the inhalation route the LC50 ranges from 2.8 to 4.7 mg/l/4hrs indicating that 3,4-DCA is considerably more toxic than diuron itself. Dermal and inhalation absorption is rapid leading to formation of methaemoglobin. A marked species difference is dermal toxicity is noted with rabbit considerably more sensitive than rats. No human data is available but by extrapolating from other aromatic amines humans could be considerably more sensitive to methaemoglobin formation than rats. 3,4-DCA should be regarded as a potential respiratory sensitiser. The carcinogenic potential of 3,4-DCA remains uncertain.
1,2-BENZISOTHIAZOLINE-3-ONE	The predominant fate of the thiazole ring is oxidative ring scission catalysed by cytochrome P450 (CYP) and formation of the corresponding alpha-dicarbonyl metabolites and thioamide derivatives. The well-established toxicity associated with thioamides and thioureas has led to the speculation that thiazole toxicity is attributed to ring scission yielding the corresponding thioamide metabolite. Ring opening has also been observed in benzothiazoles. For instance, benzothiazole itself is converted to S-methylmercaptoaniline. **Acute toxicity** data show that 1,2-benzisothiazoline-3-one (BIT) is moderately toxic by the oral and dermal routes but that this chemical is a severe eye irritant. Irritation to the skin from acute data show only mild skin irritation , but repeated dermal application indicated a more significant skin irritation response. The neurotoxicity observed in the rat acute oral toxicity study (piloerection and upward curvature of the spine at 300 mg/kg and above; decreased activity, prostration, decreased abdominal muscle tone, reduced righting reflex, and decreased rate and depth of breathing at 900 mg/kg) and the acute dermal toxicity study (upward curvature of the spine was observed in increased incidence, but this was absent after day 5 post-dose at a dose of 2000 mg/kg) were felt to be at exposures in excess of those expected from the use pattern of this pesticide and that such effects would not be observed at estimated exposure doses. **Subchronic oral toxicity** studies showed systemic effects after repeated oral administration including decreased body weight, increased incidence of forestomach hyperplasia, and non-glandular stomach lesions in rats. In dogs, the effects occurred at lower doses than in rats, and included alterations in blood chemistry (decreased plasma albumin, total protein, and alanine aminotransferase) and increased absolute liver weight. **Developmental toxicity** studies were conducted in rats with maternal effects including decreased body weight gain, decreased food consumption, and clinical toxicity signs (audible breathing, haircoat staining of the anogenital region, dry brown material around the nasal area) as well as increased mortality. Developmental effects consisted of increases in skeletal abnormalities (extra sites of ossification of skull bones, unossified sternebrae) but not external or visceral abnormalities. **Reproductive toxicity:** In a two- generation reproduction study, parental toxicity was observed at 500 ppm and was characterized by lesions in the stomach. In pups, toxic effects were reported at 1000 ppm and consisted of preputial separation in males and impaired growth and survival in both sexes. The reproduction study did not show evidence of increased susceptibility of offspring.
2-METHYL-4-ISOTHIAZOLIN-3-ONE	Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies with similar materials using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies. The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The European Union has reclassified several formaldehyde-releasing agents (FRAs) such as methylenedimorpholine (MBM), oxazolidine (MBO) and hydroxypropylamine (HPT) as category 1B carcinogens. Previously, formaldehyde itself was classed as a carcinogen – but formaldehyde-releasing agents were not. This is no longer the case. Based on this regulation, formulations for which the maximum theoretical concentration of releasable formaldehyde is more than > 1000 ppm (>0.1%), have to be labelled as carcinogenic. Water mix metalworking fluids are subject to contamination by bacteria and fungi, and the control of this is an essential part of good fluid maintenance. The use of preservatives both within the formulation and tank-side treatment plays a significant contribution in the protection of potentially harmful microbes that could cause health problems for workers. A large proportion of bactericides on the market today are classed as formaldehyde releasing biocides which means that under specific conditions they release small amounts of formaldehyde – this is their mode of action in the presence of bacteria. Although they are effective as a biocide their use may become restricted or unfavourable due to potential changes in legislation. A decision by the ECHA (European Chemicals Agency) was made to re-classify formaldehyde as a category 1b H350 carcinogen and category 2 mutagen in June 2015. It has also been proposed by the ECHA Risk Assessment Committee (RAC) that formaldehyde release biocides should be classified the same as formaldehyde because formaldehyde is released when these substances come into contact under favorable conditions (i.e. interaction with microorganisms). Formaldehyde generators (releasers) are often used as preservatives (antimicrobials, biocides, microbiocides). Formaldehyde may be generated following hydrolysis. The most widely used antimicrobial compounds function by releasing formaldehyde once inside the microbe cell. Some release detectable levels of formaldehyde into the air space, above working solutions, especially when pH has dropped. Many countries are placing regulatory pressure on suppliers and users to replace formaldehyde generators. Formaldehyde generators are a diverse group of chemicals that can be recognised by a small, easily detachable formaldehyde moiety, prepared by reacting an amino alcohol with formaldehyde ("formaldehyde-condensates"), There is concern that when formaldehyde-releasing preservatives are present in a formulation that also includes amines, such as triethanolamine (TEA), diethanolamine (DEA), or monoethanolamine (MEA), nitrosamines can be formed,; nitrosamines are carcinogenic substances that can potentially penetrate skin. One widely-discussed hypothesis states that formaldehyde-condensate biocides, such as triazines and oxazolidines, may cause an imbalance in the microbial flora of in-use metalworking fluids (MWFs).The hypothesis further asserts that this putative microbial imbalance favours the proliferation of certain nontuberculosis mycobacteria (NTM) in MWFs and that the subsequent inhalation of NTM-containing aerosols can cause hypersensitivity pneumonitis (HP), also known as extrinsic allergic alveolitis, in a small percentage of susceptible workers. Symptoms of HP include flu-like illness accompanied by chronic dyspnea, i.e., difficult or laboured respiration According to Annex VI of the Cosmetic Directive 76/768/EC, the maximum authorised concentration of free formaldehyde is 0.2%

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	(2000 ppm). In addition, the provisions of Annex VI state that, *All finished products containing formaldehyde or substances in this Annex and which release formaldehyde must be labelled with the warning "contains formaldehyde" where the concentration of formaldehyde in the finished product exceeds 0.05%.* Formaldehyde-releasing preservatives have the ability to release formaldehyde in very small amounts over time. The use of formaldehyde-releasing preservatives ensures that the actual level of free formaldehyde in the products is always very low but at the same time sufficient to ensure absence of microbial growth. The formaldehyde reacts most rapidly with organic and inorganic anions, amino and sulfide groups and electron-rich groups to disrupt metabolic processes, eventually causing death of the organism. **NOTE:** Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA. Considered to be a minor sensitiser in Kathon CG (1) (1). Bruze etal - Contact Dermatitis 20: 219-39, 1989
2-OCTYL-4-ISOTHIAZOLIN-3-ONE	ROHM & HAAS Data ADI: 0.03 mg/kg/day NOEL: 60 mg/kg/day
TALC & TITANIUM DIOXIDE & 2-METHYL-4-ISOTHIAZOLIN-3-ONE & 2-OCTYL-4-ISOTHIAZOLIN-3-ONE	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. 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. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
TALC & BARIUM SULFATE & KAOLIN & TITANIUM DIOXIDE & DIURON & 1,2-BENZISOTHIAZOLINE-3-ONE & 2-METHYL-4-ISOTHIAZOLIN-3-ONE	No significant acute toxicological data identified in literature search.
TALC & SILICA AMORPHOUS	The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
TITANIUM DIOXIDE & 2-METHYL-4-ISOTHIAZOLIN-3-ONE	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.
1,2-BENZISOTHIAZOLINE-3-ONE & 2-METHYL-4-ISOTHIAZOLIN-3-ONE & 2-OCTYL-4-ISOTHIAZOLIN-3-ONE	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.
1,2-BENZISOTHIAZOLINE-3-ONE & 2-METHYL-4-ISOTHIAZOLIN-3-ONE	In light of potential adverse effects, and to ensure a harmonised risk assessment and management, the EU regulatory framework for biocides has been established with the objective of ensuring a high level of protection of human and animal health and the environment. To this aim, it is required that risk assessment of biocidal products is carried out before they can be placed on the market. A central element in the risk assessment of the biocidal products are the utilization instructions that defines the dosage, application method and amount of applications and thus the exposure of humans and the environment to the biocidal substance. Humans may be exposed to biocidal products in different ways in both occupational and domestic settings. Many biocidal products are intended for industrial sectors or professional uses only, whereas other biocidal products are commonly available for private use by non-professional users. In addition, potential exposure of non-users of biocidal products (i.e. the general public) may occur indirectly via the environment, for example through drinking water, the food chain, as well as through atmospheric and residential exposure. Particular attention should be paid to the exposure of vulnerable sub-populations, such as the elderly, pregnant women, and children. Also pets and other domestic animals can be exposed indirectly following the application of biocidal products. Furthermore, exposure to biocides may vary in terms of route (inhalation, dermal contact, and ingestion) and pathway (food, drinking water, residential, occupational) of exposure, level, frequency and duration.

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

Dunlop Bond Coat

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

talc

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	96h	Algae or other aquatic plants	7202.7mg/l	2
LC50	96h	Fish	89581.016mg/l	2
NOEC(ECx)	720h	Algae or other aquatic plants	918.089mg/l	2

barium sulfate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>1.15mg/l	2

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	EC50	48h	Crustacea	32mg/L	2
	NOEC(ECx)	72h	Algae or other aquatic plants	>=1.15mg/l	2
	LC50	96h	Fish	>3.5mg/l	2

kaolin

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

silica amorphous

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	14.1mg/l	2
EC50	48h	Crustacea	>86mg/l	2
EC50	96h	Algae or other aquatic plants	217.576mg/l	2
LC50	96h	Fish	1033.016mg/l	2
EC0(ECx)	24h	Crustacea	>=10000mg/l	1

gamma-glycidoxypropyltrimethoxysilane

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>420mg/l	2
EC50	48h	Crustacea	473mg/l	2
EC50	96h	Algae or other aquatic plants	250mg/l	2
NOEC(ECx)	96h	Fish	1.5mg/l	2
LC50	96h	Fish	4.9mg/l	2

titanium dioxide

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1008h	Fish	<1.1-9.6	7
EC50	72h	Algae or other aquatic plants	3.75-7.58mg/l	4
EC50	48h	Crustacea	1.9mg/l	2
EC50	96h	Algae or other aquatic plants	179.05mg/l	2
LC50	96h	Fish	1.85-3.06mg/l	4
NOEC(ECx)	672h	Fish	>=0.004mg/L	2

silica crystalline - quartz

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

diuron

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1008h	Fish	<2.9-14	7
EC50	72h	Algae or other aquatic plants	0.00055mg/l	4
EC50	48h	Crustacea	>0.677mg/l	4
EC50	96h	Algae or other aquatic plants	0.001mg/l	4
LC50	96h	Fish	0.5mg/l	4
EC10(ECx)	48h	Algae or other aquatic plants	0.00004mg/l	4

1,2-benzisothiazoline-3-one

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.07mg/L	2
EC50	48h	Crustacea	0.097mg/L	4
NOEC(ECx)	72h	Algae or other aquatic plants	0.04mg/L	2
LC50	96h	Fish	0.067-0.29mg/L	4

2-methyl-4-isothiazolin-3-one

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.057mg/L	2
EC50	48h	Crustacea	0.189-0.257mg/L	4
EC50	96h	Algae or other aquatic plants	0.061mg/L	2
LC50	96h	Fish	0.081-0.122mg/L	4
NOEC(ECx)	96h	Algae or other aquatic plants	0.01mg/l	2

2-octyl-4-isothiazolin-3-one

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	0.057-0.178mg/L	4
EC50	96h	Algae or other aquatic plants	0.15mg/l	2
NOEC(ECx)	840h	Fish	0.009mg/L	4
LC50	96h	Fish	0.041-0.104mg/l	4

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 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*

Continued...

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
silica amorphous	LOW	LOW
gamma-glycidoxypropyltrimethoxysilane	HIGH	HIGH
titanium dioxide	HIGH	HIGH
diuron	HIGH	HIGH
2-methyl-4-isothiazolin-3-one	HIGH	HIGH
2-octyl-4-isothiazolin-3-one	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
silica amorphous	LOW (LogKOW = 0.5294)
gamma-glycidoxypropyltrimethoxysilane	LOW (LogKOW = -0.9152)
titanium dioxide	LOW (BCF = 10)
diuron	LOW (BCF = 14)
2-methyl-4-isothiazolin-3-one	LOW (LogKOW = -0.8767)
2-octyl-4-isothiazolin-3-one	LOW (LogKOW = 2.561)

Mobility in soil

Ingredient	Mobility
silica amorphous	LOW (KOC = 23.74)
gamma-glycidoxypropyltrimethoxysilane	LOW (KOC = 90.22)
titanium dioxide	LOW (KOC = 23.74)
diuron	LOW (KOC = 136)
2-methyl-4-isothiazolin-3-one	LOW (KOC = 27.88)
2-octyl-4-isothiazolin-3-one	LOW (KOC = 2120)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ **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
talc	Not Available
barium sulfate	Not Available
kaolin	Not Available
silica amorphous	Not Available

Continued...

Product name	Group
gamma-glycidoxypropyltrimethoxysilane	Not Available
titanium dioxide	Not Available
silica crystalline - quartz	Not Available
diuron	Not Available
1,2-benzisothiazoline-3-one	Not Available
2-methyl-4-isothiazolin-3-one	Not Available
2-octyl-4-isothiazolin-3-one	Not Available

Transport in bulk in accordance with the IGC Code

Product name	Ship Type
talc	Not Available
barium sulfate	Not Available
kaolin	Not Available
silica amorphous	Not Available
gamma-glycidoxypropyltrimethoxysilane	Not Available
titanium dioxide	Not Available
silica crystalline - quartz	Not Available
diuron	Not Available
1,2-benzisothiazoline-3-one	Not Available
2-methyl-4-isothiazolin-3-one	Not Available
2-octyl-4-isothiazolin-3-one	Not Available

SECTION 15 Regulatory information

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

talc is found on the following regulatory lists

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 Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

barium sulfate 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)

kaolin is found on the following regulatory lists

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

silica amorphous is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring
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
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

gamma-glycidoxypropyltrimethoxysilane is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

titanium dioxide is found on the following regulatory lists

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)

silica crystalline - quartz is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring
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 1: Carcinogenic to humans

diuron 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 WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

Continued...

1,2-benzisothiazoline-3-one is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals | Australian Inventory of Industrial Chemicals (AIIC)

2-methyl-4-isothiazolin-3-one is found on the following regulatory lists

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

2-octyl-4-isothiazolin-3-one is found on the following regulatory lists

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

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (talc; barium sulfate; kaolin; gamma-glycidoxypropyltrimethoxysilane; silica crystalline - quartz; diuron; 1,2-benzisothiazoline-3-one; 2-methyl-4-isothiazolin-3-one; 2-octyl-4-isothiazolin-3-one)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (kaolin)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (gamma-glycidoxypropyltrimethoxysilane)
Vietnam - NCI	Yes
Russia - FBEPH	Yes
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 exempt or will require registration.*

SECTION 16 Other information

Revision Date	10/03/2023
Initial Date	29/04/2021

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	29/04/2021	Toxicological information - Acute Health (eye), Toxicological information - Acute Health (inhaled), Toxicological information - Acute Health (skin), Toxicological information - Acute Health (swallowed), First Aid measures - Advice to Doctor, Toxicological information - Chronic Health, Hazards identification - Classification, Disposal considerations - Disposal, Ecological Information - Environmental, Firefighting measures - Fire Fighter (extinguishing media), Firefighting measures - Fire Fighter (fire/explosion hazard), Firefighting measures - Fire Fighter (fire fighting), Firefighting measures - Fire Fighter (fire incompatibility), First Aid measures - First Aid (inhaled), First Aid measures - First Aid (skin), Handling and storage - Handling Procedure, Composition / information on ingredients - Ingredients, Exposure controls / personal protection - Personal Protection (hands/feet), Handling and storage - Storage (storage incompatibility), Name
3.1	10/03/2023	Classification change due to full database hazard calculation/update.

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

Continued...

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