

Pebeo Deco Paint

Jasco Pty Limited

Chemwatch Hazard Alert Code: 1

Chemwatch: 5423-15

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Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

L.GHS.AUS.EN

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

Product Identifier

Product name	Pebeo Deco Paint
Chemical Name	Not Applicable
Synonyms	EN-FDS179 Deco Paint
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	Paints & Varnishes for artists. Use according to manufacturer's directions.
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Details of the manufacturer or supplier of the safety data sheet

Registered company name	Jasco Pty Limited
Address	1-5 Commercial Road Kingsgrove NSW 2208 Australia
Telephone	+61 2 9807 1555
Fax	Not Available
Website	www.jasco.com.au
Email	quickinfo@jasco.com.au

Emergency telephone number

Association / Organisation	Australian Poisons Centre	CHEMWATCH EMERGENCY RESPONSE (24/7)
Emergency telephone numbers	13 11 26 (24/7)	+61 1800 951 288
Other emergency telephone numbers	Not Available	+61 3 9573 3188

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

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification ^[1]	Not Applicable

Label elements

Hazard pictogram(s)	Not Applicable
Signal word	Not Applicable

Hazard statement(s)

Not Applicable

Precautionary statement(s) Prevention

Not Applicable

Precautionary statement(s) Response

Not Applicable

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

Not Applicable

SECTION 3 Composition / information on ingredients**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
2634-33-5	<0.002	<u>1,2-benzisothiazoline-3-one</u>
55965-84-9	<0.0006	<u>5-chloro-2-methyl-4-isothiazolin-3-one</u>
128-37-0	NotSpec	<u>2,6-di-tert-butyl-4-methylphenol</u>
100-42-5	NotSpec	<u>styrene</u>
79-06-1	NotSpec	<u>acrylamide</u>
141-32-2	NotSpec	<u>butyl acrylate</u>
79-10-7	NotSpec	<u>acrylic acid</u>
7631-86-9	NotSpec	<u>silica amorphous</u>
13463-67-7	NotSpec	<u>titanium dioxide</u>

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	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ 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	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▶ Other measures are usually unnecessary.
Ingestion	<ul style="list-style-type: none"> ▶ 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.

SECTION 5 Firefighting measures**Extinguishing media**

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	<ul style="list-style-type: none"> ▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ When silica dust is dispersed in air, firefighters should wear inhalation protection as hazardous substances from the fire may be adsorbed on the silica particles. ▶ When heated to extreme temperatures, (>1700 deg.C) amorphous silica can fuse. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves in the event of a fire. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Use fire fighting procedures suitable for surrounding area. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ 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. <p>carbon dioxide (CO₂) nitrogen oxides (NO_x) silicon dioxide (SiO₂) metal oxides other pyrolysis products typical of burning organic material. May emit corrosive fumes.</p>
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	<ul style="list-style-type: none"> ▶ 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	<p>Moderate hazard.</p> <ul style="list-style-type: none"> ▶ 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. ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Neutralise/decontaminate residue (see Section 13 for specific agent). ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage**Precautions for safe handling**

Safe handling	<ul style="list-style-type: none"> ▶ 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. ▶ Avoid contact with moisture. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke.
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	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Polyethylene or polypropylene container. ▶ Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Avoid reaction with oxidising agents, bases and strong reducing agents. ▶ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

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	2,6-di-tert-butyl-4-methylphenol	2,6-Di-tert-butyl-p-cresol	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	styrene	Styrene, monomer	50 ppm / 213 mg/m3	426 mg/m3 / 100 ppm	Not Available	Not Available
Australia Exposure Standards	acrylamide	Acrylamide	0.03 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	butyl acrylate	n-Butyl acrylate	1 ppm / 5 mg/m3	26 mg/m3 / 5 ppm	Not Available	Not Available
Australia Exposure Standards	acrylic acid	Acrylic acid	2 ppm / 5.9 mg/m3	Not Available	Not Available	Not Available
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	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 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: Fumed silica (respirable dust)	2 mg/m3	Not Available	Not Available	Not Available
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	Fumed silica (respirable dust)	2 mg/m3	Not Available	Not Available	Not Available
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	Diatomaceous earth (uncalcined)	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.

Pebeo Deco Paint

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	titanium dioxide	Titanium dioxide	10 mg/m ³	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
5-chloro-2-methyl-4-isothiazolin-3-one	0.6 mg/m ³	6.6 mg/m ³	40 mg/m ³
styrene	Not Available	Not Available	Not Available
acrylamide	0.09 mg/m ³	44 mg/m ³	100 mg/m ³
butyl acrylate	Not Available	Not Available	Not Available
acrylic acid	Not Available	Not Available	Not Available
silica amorphous	18 mg/m ³	200 mg/m ³	1,200 mg/m ³
silica amorphous	18 mg/m ³	100 mg/m ³	630 mg/m ³
silica amorphous	120 mg/m ³	1,300 mg/m ³	7,900 mg/m ³
silica amorphous	45 mg/m ³	500 mg/m ³	3,000 mg/m ³
silica amorphous	18 mg/m ³	740 mg/m ³	4,500 mg/m ³
titanium dioxide	30 mg/m ³	330 mg/m ³	2,000 mg/m ³

Ingredient	Original IDLH	Revised IDLH
1,2-benzisothiazoline-3-one	Not Available	Not Available
5-chloro-2-methyl-4-isothiazolin-3-one	Not Available	Not Available
2,6-di-tert-butyl-4-methylphenol	Not Available	Not Available
styrene	700 ppm	Not Available
acrylamide	60 mg/m ³	Not Available
butyl acrylate	Not Available	113 ppm
acrylic acid	Not Available	Not Available
silica amorphous	3,000 mg/m ³	Not Available
titanium dioxide	5,000 mg/m ³	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
1,2-benzisothiazoline-3-one	E	≤ 0.01 mg/m ³
5-chloro-2-methyl-4-isothiazolin-3-one	E	≤ 0.01 mg/m ³

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

NOTE D: Certain substances which are susceptible to spontaneous polymerisation or decomposition are generally placed on the market in a stabilised form. It is in this form that they are listed on Annex I

When they are placed on the market in a non-stabilised form, the label must state the name of the substance followed by the words "non-stabilised" European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

Exposure controls

Appropriate engineering controls	
	<p>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.</p> <p>The basic types of engineering controls are:</p> <ul style="list-style-type: none"> 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. <p>General exhaust is adequate under normal operating conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air</p>

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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

Natural latex and nitrile rubber are suitable.

- ▶ 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,
- 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

	<ul style="list-style-type: none"> · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades <p>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:</p> <ul style="list-style-type: none"> · 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 <p>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.</p>
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ 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:

Pebeo Deco Paint

Material	CPI
BUTYL	C
NATURAL RUBBER	C
NITRILE	C
NITRILE+PVC	C
PE	C
PE/EVAL/PE	C
PVA	C
PVC	C
SARANEX-23	C
TEFLON	C
VITON	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.

Ansell Glove Selection

Glove — In order of recommendation
AlphaTec® 38-612
AlphaTec® 58-005
AlphaTec® 53-001
AlphaTec® Solvex® 37-175
BioClean™ Emerald BENS
BioClean™ Extra BLAS
BioClean™ Fusion (Sterile) S-BFAP
BioClean™ N-Plus BNPS

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required. Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

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

^ - Full-face

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

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

BioClean™ Ultimate BUPS

MICROFLEX® 93-732

The suggested gloves for use should be confirmed with the glove supplier.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Viscous liquid; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	1.24
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable
pH (as supplied)	8.8	Decomposition temperature (°C)	Not Applicable
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Applicable
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
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 Applicable
Vapour pressure (kPa)	Not Applicable	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	40.68

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ 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	The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.
Ingestion	The material has NOT been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.
Skin Contact	The material is not thought to produce adverse health effects or skin irritation following contact (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting.
Eye	Although the liquid is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).

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Chronic	<p>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.</p> <p>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.</p> <p>Substances that can cause 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</p> <p>Wherever it is reasonably practicable, exposure to substances that can cause 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.</p> <p>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.</p> <p>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.</p> <p>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</p> <p>Repeated exposure to synthetic amorphous silicas may produce skin dryness and cracking.</p> <p>Available data confirm the absence of significant toxicity by oral and dermal routes of exposure.</p> <p>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.</p> <p>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.</p>
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Pebeo Deco Paint	TOXICITY	IRRITATION
	Not Available	Not Available
1,2-benzisothiazoline-3-one	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1] Oral (Rat) LD50: 454 mg/kg ^[1]	Eye: adverse effect observed (irreversible damage) ^[1] Skin: no adverse effect observed (not irritating) ^[1]
5-chloro-2-methyl-4-isothiazolin-3-one	TOXICITY	IRRITATION
	dermal (rat) LD50: >1008 mg/kg ^[2] Inhalation (Rat) LC50: 1.23 mg/l4h ^[2] Oral (Rat) LD50: 53 mg/kg ^[2]	Eye: adverse effect observed (irreversible damage) ^[1] Skin: adverse effect observed (corrosive) ^[1] Skin: adverse effect observed (irritating) ^[1]
2,6-di-tert-butyl-4-methylphenol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2] Oral (Rat) LD50: 890 mg/kg ^[2]	Eye (rabbit): 100 mg/24h-moderate Eye: no adverse effect observed (not irritating) ^[1] Skin (human): 500 mg/48h - mild Skin (rabbit):500 mg/48h-moderate Skin: no adverse effect observed (not irritating) ^[1]
styrene	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1] Inhalation (Mouse) LC50: 9.5 mg/L4h ^[2] Oral (Mouse) LD50; 316 mg/kg ^[2]	Eye (rabbit): 100 mg/24h - moderate Eye (rabbit): 100 mg/24h - moderate Skin (rabbit): 500 mg - mild Skin (rabbit): 500 mg - mild
acrylamide	TOXICITY	IRRITATION
	dermal (rat) LD50: 400 mg/kg ^[2] Oral (Rat) LD50: 124 mg/kg ^[2]	Eye (rabbit): 100mg/24h - moderate Eye (rabbit): 10mg/30s rinse-mild Skin (rabbit): 50 mg/3d - mild Skin (rabbit): 500 mg/24h - mild

	TOXICITY	IRRITATION
butyl acrylate	Dermal (rabbit) LD50: 750 mg/kg ^[2]	Eye (rabbit) 50 mg - mild
	Inhalation (Rat) LC50: >5.24 mg/l4h ^[1]	Eye: adverse effect observed (irritating) ^[1]
	Oral (Rat) LD50: 900 mg/kg ^[2]	Skin (rabbit) 10 mg/24h open mild
		Skin (rabbit) 500 mg open - mild
		Skin: adverse effect observed (irritating) ^[1]
acrylic acid	Dermal (rabbit) LD50: >2000 mg/kg ^[1]	Eye: adverse effect observed (irreversible damage) ^[1]
	Inhalation (Rat) LC50: >1.078 mg/l4h ^[1]	Skin: adverse effect observed (corrosive) ^[1]
	Oral (Rat) LD50: >=146<=468 mg/kg ^[1]	Skin: adverse effect observed (irritating) ^[1]
silica amorphous	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]
titanium dioxide	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]

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

1,2-BENZISOTHIAZOLINE-3-ONE	<p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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 sternbrae) but not external or visceral abnormalities.</p> <p>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.</p>
5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE	<p>Considered to be the major sensitiser in Kathon CG (1) (1). Bruze et al - Contact Dermatitis 20: 219-39, 1989</p> <p>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.</p> <p>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.</p> <p>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</p>

Continued...

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% (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.

The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

2,6-DI-TERT-BUTYL-4-METHYLPHENOL

* Degussa SDS Effects such as behavioral changes, reduction in body weight gain, and decrement in body weight have been observed after long-term administration of BHT to mice and rats. Toxic effects may be attributed more to BHT metabolites than to their parent compound, only a few studies have focused on their carcinogenicity and toxicity, and not only on that of BHT. The metabolite BHT-QM (syn: 2,6-di-tert-butyl-1,4-methylene-2,5-cyclohexadien-1-one, CAS RN: 2607-52-5) is a very reactive compound which is considered to play a significant role in hepatotoxicity, pneumotoxicity, and skin tumor promotion in mice. In addition, it was reported that another quinone derivative, BHT-OH(t)QM (syn 2-tert-butyl-6-(2-hydroxy-tert-butyl-4-methylene-2,5-cyclohexadien-1-one, CAS RN: 124755-19-7), is chemically more reactive than BHT-QM, and it has been recognized as the principal metabolite responsible for lung tumor promotion activity of BHT in mice. BHT has been reported to exert prooxidant effects under certain conditions. Thus, when BHT was added in excess to a wheat seedling medium in aerobic conditions, an enhancement of the generation rate of superoxide anion was observed. This is a reactive particle that may damage cellular structures at high concentrations. In addition, an increase in hepatic microsomal lipid peroxidation was observed in rats fed with diets containing 0.2% of BHT for 30 days. Due to this ability of BHT to exert prooxidant effects at high concentrations, it has been used to induce experimental models of oxidative stress in several animals and fungi in order to study the protective effects of other compounds. Quinone methide derivatives form adducts with several proteins, including enzymes that protect cells from oxidative stress; this prooxidant state can also lead to cell oxidative damage. It must be noted that relationships between chronic oxidative stress and tumor promotion are well known. Some authors have reported that at high aeration rate, BHT can react with molecular oxygen rather than with the reactive oxygen species present, yielding BHT-phenoxy radical and superoxide anion. In addition, the phenolic radical itself may undergo redox recycling which can be a critical factor depending on the reductant involved. However, it has to be noted that BHT-phenoxy radical has been reported to be relatively stable. Furthermore, the potential reactivity of BHT-derived metabolites should be taken into account; some studies reported that not only BHT but also its metabolites, such as BHT-Q and BHT-QM, can act as prooxidant. As BHT undergoes several reactions during biotransformation, a large number of intermediate metabolites have been identified. However, their nature and concentration depend on the environmental conditions and on the animal species. Although the changes undergone by BHT during in vivo digestion processes have not been studied, after submission of a fluid deep-frying fat containing BHT and BHT-QM to an in vitro gastrointestinal digestion model, both these were detected in the digested samples. These results indicate that BHT and its toxic metabolite could remain bioaccessible for intestinal absorption. Studies concerning BHT metabolism have shown that, unlike other synthetic antioxidants, BHT is a potent inducer of the microsomal monooxygenase system and its major route of degradation is oxidation catalyzed by cytochrome P450. Studies have reported potential toxicity derived from the ingestion or administration of BHT. As for acute oral toxicity, although this is considered low in animals, it must be noted that 2 clinical cases were reported in patients who suffered acute neurotoxicity and gastritis after ingesting a high dose of BHT (4 and 80 g without medical prescription) to cure recurrent genital herpes. Regarding short-term subchronic toxicity studies, it has been reported that BHT causes dose-related increase in the incidence and severity of toxic nephrosis in mice, nephrotoxicity and pneumotoxicity in rats, and in chicken a marked congestion of the liver and kidney, as well as diffuse enlargement of the liver with rounded borders and rupture with hemorrhaging. It has to be noted that the EFSA Panel (2012) pointed out certain inconsistencies in the findings obtained from the short-term and subchronic toxicity studies. Several genotoxicity studies on BHT concluded that BHT does not represent a genotoxic risk, because most of the studies carried out to that date had shown BHT was not able to induce mutations or to damage deoxyribonucleic acid (DNA). Nevertheless, it must be mentioned that other studies reported contrary results. The effect of BHT and 7 of its metabolites on in vitro DNA cleavage was studied and the metabolites BHT-Q (syn: 2,6-di-tert-butyl-2,5-cyclohexadiene-1,4-dione, CAS RN: 719-22-2), BHT-CHO (syn: 3,5-di-tert-butyl-4-hydroxybenzaldehyde, CAS RN: 1620-98-0) and BHT-OOH (syn: 2,6-di-tert-butyl-4-methyl-4-hydroperoxy-2,5-cyclohexadien-1-one, CAS RN: 6485-57-0) were able to cleave DNA. The Panel on Food Additives and Nutrient Sources Added to Food of the European Food Safety Authority (EFSA)

recognized that these positive genotoxicity results may be due to the prooxidative chemistry of BHT, which gives rise to reactive metabolites. Some studies addressed the carcinogenicity and chronic toxicity of BHT and its metabolites in rodents with contradictory results. Thus, mice-fed dietary BHT for a year developed marked hyperplasia of the hepatic bile ducts with an associated subacute cholangitis. Moreover, after 104 wk of administration of BHT, the formation of hepatocellular tumors in male mice was observed. After 10 months of feeding mice with a diet containing different amounts of BHT, an increased incidence of liver tumors in male, but not female, animals was also reported. However, in a similar study no evidence of the carcinogenicity of BHT administered to mice was observed. Studies performed in rats also reported dose-related increases in hepatocellular adenomas and carcinomas; nevertheless, other studies carried out with rats showed no consistent carcinogenic effects. Several studies have demonstrated the potential of BHT to act either as a tumor promoter or as a tumor suppressor, modulating the carcinogenicity of some well-known carcinogens. Barbara Nieva-Echevarria et al: Comprehensive reviews in Food Science and Food Safety, Vol 14, Dec 2014 <https://onlinelibrary.wiley.com/doi/10.1111/1541-4337.12121/pdf> for bridged alkyl phenols:

Acute toxicity: Acute oral and dermal toxicity data are available for all but two of the substances in the group. The data show that acute toxicity of these substances is low. The testing for acute toxicity spans five decades

Repeat dose toxicity: Repeat dose studies on the members of this category include both subchronic and chronic exposures. The liver is identified as the target organ in rats for all of the substances tested. NOAEL s or NOEL s in rats for 13- week studies ranged from 100 ppm (approximately 5 mg/kg/day) to 500 ppm (approximately 25 mg/kg/day) while NOAEL s or NOEL s in rats for chronic studies were the same, 25 mg/kg/day (500 ppm).

Reproductive toxicity: Evaluation of effects on reproduction for the bridged alkyl phenols is supplemented by histopathological data on male and female reproductive organs in repeated dose studies. The data on the effects of bridged alkyl phenols on reproduction and reproductive organs span the range of structures and molecular weights. While not all of the data for reproductive effects are from reproduction studies, microscopic evaluations of reproductive organs along with other short-term tests for reproductive effects provide adequate data to evaluate the effects of these bridged alkyl phenols on reproduction. It can be concluded that reproductive toxicity is low.

Typically a two-year chronic feeding study provides data for 4,4'-thiobis-6-(t-butyl-m-cresol) (96-69-5). No adverse effects were noted on reproductive organs

Genotoxicity: Data from bacterial reverse mutation assays and *in vitro* and *in vivo* chromosome aberration studies were reviewed. Adequate bacterial gene mutation assays have been conducted with all of the category chemicals except two. Chromosome aberration studies, *in vitro* and/or *in vivo*, are available for all but two substances. The mutagenicity data span the range of structures and molecular weights and data can be bridged from other members of the group to meet any outstanding requirements. The weight of evidence for mutagenic potential for this category indicates these substances are not mutagenic.

Carcinogenicity: The mutagenicity data combined with the animal data plus the long historical use of BHT (128-37-0) indicate that the chemicals in this class are not expected to exhibit any significant potential to cause cancer. The weight of the evidence indicates that these chemicals are not genotoxic.

The Bridged Alkyl Phenols Category consists of a group of chemicals in which two molecules of mono or di-substituted alkyl (C1, C4, and/or C9) phenols are "bridged" or linked by a single atom (carbon or sulfur). The carbon atom linking the alkyl phenol groups contains hydrogen, propyl, or methyl substitutions. CAS No. 128-37-0 (BHT) is included in this category for data purposes because it is an alkyl phenol with a single carbon group such as the ones that link the phenol groups. ferroptosis inhibitors are currently being treated systemically rather than specifically, which may have multiple side effects. For example, Desferoxamin (DFO), an iron chelating agent, is known to have a short half-life, need long-term subcutaneous infusions, and provoke ototoxicity and neurotoxicity. Deferasirox (DFX), an iron chelator, is associated with gastrointestinal and renal toxicity.

For hindered phenols:

Available data shows that acute toxicity of these substances is low.

Mutagenicity. Data from bacterial reverse mutation assays and *in vitro* and *in vivo* chromosome aberration studies were reviewed. All assays, with and without metabolic activation, were negative. The weight of evidence for mutagenic potential for this category indicates these substances are not mutagenic.

In Vitro Chromosome Aberration Studies. *In vitro* chromosome aberration studies are available for several members. All except 2,6-di-tert-butyl-p-cresol were negative.

In Vivo Chromosome Aberration Studies. *In vivo* studies evaluating chromosome damage are available for six of the hindered phenols. All *in vivo* evaluations were negative.

Repeated Dose Toxicity. Repeated dose toxicity data of approximately three months (90-day, 12- and 13-week) are available for some of the substances in this group. The liver was the target organ in rats for almost all of the substances with subchronic toxicity data in that species. Other target organs included thyroid and kidney and mesenteric lymph nodes. NOAELs in rats ranged from 100 ppm (approximately 5 mg/kg/day) to 10,000 ppm (500 mg/kg/day).

Carcinogenicity: Data is available for 2,6-di-tert-butyl-p-cresol (128-37-0); and 4,4'-thiobis-6-(t-butyl-m-cresol) (96-69-5). Liver adenomas were reported for 2,6-di-tert-butyl-p-cresol (128-37-0) and a NOAEL was established for the study at 25 mg/kg/day. 4,4'-Thiobis-6-(t-butyl-m-cresol) (96-69-5) was not carcinogenic in rats or mice, but the kidney was identified as a target organ in female rats

ACRYLAMIDE

WARNING: This substance has been classified by the IARC as Group 2A: Probably Carcinogenic to Humans. Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [National Toxicology Program: U.S. Dep. of Health & Human Services 2002]

BUTYL ACRYLATE

Where no "official" classification for acrylates and methacrylates exists, there has been cautious attempts to create classifications in the absence of contrary evidence. For example

Monoalkyl or monoarylestere of acrylic acids should be classified as R36/37/38 and R51/53

Monoalkyl or monoaryl estere of methacrylic acid should be classified as R36/37/38

for n-butyl acrylate

Acute toxicity: After oral administration, n-butyl acrylate is rapidly absorbed and metabolized in male rats (75% was eliminated as CO₂, approximately 10% via urine and 2% via feces). The major portion of n-butyl acrylate was hydrolysed by carboxyesterase to acrylic acid and butanol.

Following acute exposure, n-butyl acrylate exhibits low toxicity. n-Butyl acrylate has oral LD50s of 3143 mg/kg bw (rats) and 9050 mg/kg bw (male rats), an inhalation LC50 (4-hour, rat) of 10.3 mg/L and a dermal LD50 (rabbit)

of 2000 to 3024 mg/kg. n-Butyl acrylate is irritating to skin and eyes and showed a skin sensitising potential in animals. In humans, skin sensitisation to butyl acrylate was reported. Patch test concentration ranged from 0.1 to 0.5%. 6 out of 124 patients

were positive, but the author stated that those results should be interpreted with caution, due to clinical history of the patients and purity of the different tested acrylates. Another publication describes that a data collection of 82 patients between 1987 and 1992 suspected of occupational acrylic sensitisation, showed in the patch test with 1% in petrolatum 2 patients to be sensitised to n-butyl acrylate

Repeat dose toxicity: In an oral (drinking water) 90-day study in rats, using a satellite group (gavage) at 150 mg/kg bw/day, the only effects reported were a slight reduction in water consumption in all dose groups and a decrease in weight gain in the highest dose group. The NOAEL (males) = 84 mg/kg/bw/day and NOAEL (females) = 111 mg/kg/bw/day. The NOAEL (gavage) (males and females) = 150 mg/kg/bw/day.

In a 90-day inhalation study, rats were exposed to 0, 21, 108, 211, and 546 ppm (0, 0.11, 0.57, 1.12, 2.90 mg/L) n-butyl acrylate. The primary effects at 211 ppm (1.12 mg/L) were irritation of eyes and nasal mucosa, reduced body weights (13.3 percent in males and 3.76 percent in females compared with controls), decreased potassium values (females) and an increase in alkaline phosphatase activity (females.) At the highest dose of 546 ppm (2.90 mg/L) 31 of 40 animals died. The primary cause of death was due to the strong irritation of the substance on the respiratory tract. The NOAEL = 108 ppm (0.57 mg/L/day) and the LOAEL = 211 ppm (1.12 mg/L/day).

In a two-year inhalation study, rats (male/female) received whole body exposures of 0, 15, 45, or 135 ppm (0, 0.086, 0.258, 0.773 mg/L). There was a slight decrease in food consumption and slightly lower relative heart, kidney, liver and thyroid weights at the highest dose. A NOAEL was determined to be 45 ppm (0.258 mg/L/day) based upon localized and diffuse stippling of the corneal epithelium, cloudiness of the cornea, and various degrees of vascularization. The severity of nasal mucosa effects increased with dose and occurred at all doses in males and females. Effects ranged from slight atrophy of the neurogenic part of the olfactory epithelium at 15 ppm (0.086 mg/L) to partial loss of the columnar cell layer and stratified reserve-cell hyperplasia at 45 (0.258 mg/L) and 135 ppm (0.773 mg/L).

Reproductive toxicity: In repeated-dose studies (noted above), no effects were seen in the reproductive organs.

Developmental toxicity: In developmental toxicity studies with rats via inhalation, n-butyl acrylate caused foetotoxic effects (resorptions and reduced number of live fetuses at >135 ppm) at maternally toxic concentrations.

At exposures of 25, 135 and 250 ppm (0.13, 0.72 and 1.33 mg/L/day), the NOAEL (maternal) = 25 ppm (0.13 mg/L/day) based on reduced body weights and irritation to the eyes and nose. The NOAEL (developmental) = 25 ppm (0.13 mg/L/day), based on post-implantation loss and the NOAEL (teratogenicity) = 250 ppm. In a separate study, female rats were given 100, 200 and 300 ppm. A maternal NOAEL could not be determined based on a reduction of absolute body weight gain at all doses; the maternal LOAEL was set at 100 ppm. At 200 and 300 ppm there was a reduction in foetal body weights. Sporadic malformations occurred at 300 ppm and in the control group.

The NOAEL (developmental) was 100 ppm and the NOAEL (teratogenicity) was 300 ppm (highest dose tested).

Genotoxicity: n-Butyl acrylate was negative in the Ames test with Salmonella typhimurium TA98, TA100, TA1535 and TA1537 with and without metabolic activation tested up to 10,000 µg/plate. In a cytogenetic assay with Chinese Hamster Ovary Cells, n-butyl acrylate showed no clastogenic potential in concentrations where no cytotoxicity occurred.

Without metabolic activation an increase of aberrant cells was observed at cytotoxic concentrations. No genotoxic effects were found in an in vitro micronucleus test and an UDS-test with Syrian hamster fibroblasts. In an in vivo cytogenetic assay, n-butyl acrylate showed no clastogenic effect in rats and hamsters after inhalation exposure.

Carcinogenicity: n-Butyl acrylate was not carcinogenic to rats via inhalation up to 135 ppm (0.773 mg/L/day), the highest dose tested.

Based on the available oncogenicity data and without a better understanding of the carcinogenic mechanism the Health and Environmental Review Division (HERD), Office of Toxic Substances (OTS), of the US EPA previously concluded that all chemicals that contain the acrylate or methacrylate moiety ($\text{CH}_2=\text{CHCOO}$ or $\text{CH}_2=\text{C}(\text{CH}_3)\text{COO}$) should be considered to be a carcinogenic hazard unless shown otherwise by adequate testing.

This position has now been revised and acrylates and methacrylates are no longer *de facto* carcinogens.

ACRYLIC ACID

For acrylic acid:

Acute toxicity: Acrylic acid is absorbed via the lungs in animals and humans, absorption via the oral and dermal routes of exposure is demonstrated. In animals with solely nasal respiration, it is resorbed at the nasal mucosa. The extent of absorption depends on pH and solvent with direct dependence on substance concentration. In mice acrylic acid is rapidly and completely metabolised mainly in liver and kidney via the normal catabolic pathways of beta-oxidation. Elimination preferably occurs as carbon dioxide.

Pure acrylic acid is a very reactive chemical and accordingly exhibits severe corrosive properties in contact with biological material. Thus, acrylic acid causes acute harmful effects by oral and dermal exposure. Oral LD50 values for rats cover a range from 140 up to 1400 mg/kg bw depending on the concentration of the test substance. An oral LD50 of 1350 mg/kg bw was detected for male rats with a 10% aqueous solution of acrylic acid (pH 2.5) thus indicating that corrosive effects are not caused by the pH of the test substance. A dermal LD50 of 640 mg/kg bw was determined for rabbits (with undiluted acrylic acid). Acute inhalation toxicity is low because acrylic acid interacts with humidity of the air prior to reaching the depth of the respiratory tract. LC50 values of 3.6 to >5.1 mg/l/4 hours have been determined.

Workplace data demonstrate that acrylic acid causes skin corrosion and irritation of the respiratory tract in humans.

In tests with rabbits the pure acid caused severe burns to skin and eyes. Severe ocular damage caused by acrylic acid cannot be avoided by neutralizing the acid.

Pure acrylic acid does not show skin sensitizing properties in animal sensitization tests. However, skin sensitization was observed in humans. This was attributed to oligomeric impurities in the raw material. Respiratory sensitization has not been observed in humans.

Repeat dose toxicity: Repeated oral and inhalation exposure of acrylic acid to rats and mice resulted in dose related severe effects. Gavage on 90 days revealed dose-dependent mortality, irritation and ulceration of the stomach, and renal tubular necrosis in rats (LOAEL 150 mg/kg bw/d). No specific toxic effects were noted in subchronic and chronic drinking water studies. Reduced palatability (decreased water consumption) and unspecific signs of toxicity (decreased food consumption, body weight gain) at dosages >2000 ppm (100 mg/kg bw/d in male rats, 150 mg/kg bw/d in females) were observed. In a 90-day inhalation study, acrylic acid induced degenerative lesions on the olfactory mucosa in mice at 5 ppm (0.015 mg/l) and in rats at 75 ppm (0.221 mg/l). Mice seemed to be more sensitive than rats, thus a LOAEC of 5 ppm (0.015 mg/l) was derived for local effects. Long term dermal exposure at concentrations >1 % resulted in skin irritation.

Genotoxicity: Acrylic acid did not induce gene mutations in Salmonella or CHO cells (HPRT locus) but was clearly positive in the mouse lymphoma assay and in the in vitro chromosomal aberration test. In the mouse lymphoma assay small colonies were induced preferentially, thus the mutagenic potential of acrylic acid seems to be limited to clastogenicity. In vivo, acrylic acid did not induce mutagenic effects in either rat bone marrow cells or mouse germ cells after oral administration.

Carcinogenicity: There is no evidence that acrylic acid administered orally to rats or applied dermally to mice is carcinogenic. There are no cancer data available with respect to human exposure.

Continued...

	<p>Reproductive and developmental toxicity: In oral studies on rats no effects on reproductive function (fertility) were observed. Some signs of postnatal developmental toxicity (retarded body weight gain of the pups) were seen following exposure of the parental generation at dose levels that led to reduced food intake and weight gain in the dams. No gross abnormalities were observed in the offspring. No prenatal developmental toxicity was observed in rats and rabbits following inhalation exposure. The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.</p>
<p>SILICA AMORPHOUS</p>	<p>Reports indicate high/prolonged exposures to amorphous silicas induced lung fibrosis in experimental animals; in some experiments these effects were reversible. [PATTYS]</p> <p>For silica amorphous: Derived No Adverse Effects Level (NOAEL) in the range of 1000 mg/kg/d.</p> <p>In humans, synthetic amorphous silica (SAS) is essentially non-toxic by mouth, skin or eyes, and by inhalation. Epidemiology studies show little evidence of adverse health effects due to SAS. Repeated exposure (without personal protection) may cause mechanical irritation of the eye and drying/cracking of the skin.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>For silane treated synthetic amorphous silica: Repeated dose toxicity: oral (rat), 28-d, diet, no significant treatment-related adverse effects at the doses tested.</p> <p>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.</p>
<p>TITANIUM DIOXIDE</p>	<p>* IUCLID</p> <p>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.</p> <p>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.</p> <p>No data were available on genotoxic effects in titanium dioxide-exposed humans.</p> <p>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-</p>

	<p>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.</p> <p>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.</p> <p>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.</p> <p>Animal carcinogenicity data</p> <p>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.</p> <p>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.</p> <p>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.</p>
<p>1,2-BENZISOTHIAZOLINE-3-ONE & 5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE & 2,6-DI-TERT-BUTYL-4-METHYLPHENOL & ACRYLAMIDE & BUTYL ACRYLATE</p>	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>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.</p>
<p>1,2-BENZISOTHIAZOLINE-3-ONE & 5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE</p>	<p>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.</p>
<p>1,2-BENZISOTHIAZOLINE-3-ONE & 5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE & TITANIUM DIOXIDE</p>	<p>No significant acute toxicological data identified in literature search.</p>
<p>5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE & 2,6-DI-TERT-BUTYL-4-METHYLPHENOL & BUTYL ACRYLATE & ACRYLIC ACID & TITANIUM DIOXIDE</p>	<p>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.</p>
<p>5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE & ACRYLAMIDE & TITANIUM DIOXIDE</p>	<p>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.</p>
<p>2,6-DI-TERT-BUTYL-4-METHYLPHENOL & TITANIUM DIOXIDE</p>	<p>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.</p>
<p>2,6-DI-TERT-BUTYL-4-METHYLPHENOL & BUTYL</p>	<p>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.</p>

ACRYLATE & SILICA AMORPHOUS	
2,6-DI-TERT-BUTYL-4-METHYLPHENOL & STYRENE	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 of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
STYRENE & TITANIUM DIOXIDE	WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
ACRYLAMIDE & TITANIUM DIOXIDE	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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

	Endpoint	Test Duration (hr)	Species	Value	Source
Pebeo Deco Paint	Not Available	Not Available	Not Available	Not Available	Not Available
1,2-benzisothiazoline-3-one	EC50	72h	Algae or other aquatic plants	0.07mg/L	2
	EC50	48h	Crustacea	0.097mg/L	4
	LC50	96h	Fish	0.067-0.29mg/L	4
	NOEC(ECx)	72h	Algae or other aquatic plants	0.04mg/L	2
5-chloro-2-methyl-4-isothiazolin-3-one	EC50	72h	Algae or other aquatic plants	0.018-0.026mg/L	4
	EC50	48h	Crustacea	4.71mg/l	1
	LC50	96h	Fish	0.13-0.31mg/L	4
	NOEC(ECx)	504h	Crustacea	0.172mg/l	1
	EC50	96h	Algae or other aquatic plants	0.03-0.13mg/L	4
2,6-di-tert-butyl-4-methylphenol	BCF	1344h	Fish	220-2800	7
	EC50	72h	Algae or other aquatic plants	>0.42mg/l	1
	ErC50	72h	Algae or other aquatic plants	>0.42mg/l	1
	EC50	48h	Crustacea	>0.17mg/l	2
	EC0(ECx)	48h	Crustacea	>=0.31mg/l	1
	LC50	96h	Fish	0.199mg/l	2
	EC50	96h	Algae or other aquatic plants	0.758mg/l	2
styrene	NOEC(ECx)	96h	Algae or other aquatic plants	0.063mg/l	1
	LC50	96h	Fish	3.29-5.05mg/L	4
	EC50	72h	Algae or other aquatic plants	1.4mg/l	1
	EC50	48h	Crustacea	4.7mg/l	1
	EC50	96h	Algae or other aquatic plants	0.72mg/l	1

Continued...

	Endpoint	Test Duration (hr)	Species	Value	Source
acrylamide	EC50	48h	Crustacea	98mg/l	1
	LC50	96h	Fish	74-150mg/L	4
	NOEC(ECx)	672h	Crustacea	0.06mg/l	1
butyl acrylate	EC50	72h	Algae or other aquatic plants	1.71mg/l	2
	EC50	48h	Crustacea	1.3mg/l	2
	LC50	96h	Fish	1.1mg/l	2
	NOEC(ECx)	504h	Crustacea	0.136mg/l	2
	EC50	96h	Algae or other aquatic plants	2.65mg/l	2
acrylic acid	ErC50	72h	Algae or other aquatic plants	0.06mg/l	1
	EC50	72h	Algae or other aquatic plants	0.04mg/l	1
	EC50	48h	Crustacea	47mg/l	1
	LC50	96h	Fish	11mg/l	1
	EC50	96h	Algae or other aquatic plants	0.17mg/l	1
	NOEC(ECx)	72h	Algae or other aquatic plants	0.008mg/l	1
silica amorphous	EC50	72h	Algae or other aquatic plants	14.1mg/l	2
	EC50	48h	Crustacea	>86mg/l	2
	LC50	96h	Fish	1033.016mg/l	2
	EC50	96h	Algae or other aquatic plants	217.576mg/l	2
	EC0(ECx)	24h	Crustacea	>=10000mg/l	1
titanium dioxide	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
	LC50	96h	Fish	1.85-3.06mg/l	4
	NOEC(ECx)	672h	Fish	>=0.004mg/L	2
	EC50	96h	Algae or other aquatic plants	179.05mg/l	2

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

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
5-chloro-2-methyl-4-isothiazolin-3-one	HIGH	HIGH
2,6-di-tert-butyl-4-methylphenol	HIGH	HIGH
styrene	HIGH (Half-life = 210 days)	LOW (Half-life = 0.3 days)
acrylamide	LOW	LOW
butyl acrylate	LOW (Half-life = 14 days)	LOW (Half-life = 0.96 days)
acrylic acid	HIGH (Half-life = 180 days)	LOW (Half-life = 0.99 days)
silica amorphous	LOW	LOW
titanium dioxide	HIGH	HIGH

Bioaccumulative potential

Continued...

Ingredient	Bioaccumulation
5-chloro-2-methyl-4-isothiazolin-3-one	LOW (LogKOW = 0.0444)
2,6-di-tert-butyl-4-methylphenol	HIGH (BCF = 2500)
styrene	LOW (BCF = 77)
acrylamide	LOW (BCF = 1.65)
butyl acrylate	LOW (LogKOW = 2.36)
acrylic acid	LOW (LogKOW = 0.35)
silica amorphous	LOW (LogKOW = 0.5294)
titanium dioxide	LOW (BCF = 10)

Mobility in soil

Ingredient	Mobility
5-chloro-2-methyl-4-isothiazolin-3-one	LOW (Log KOC = 45.15)
2,6-di-tert-butyl-4-methylphenol	LOW (Log KOC = 23030)
styrene	LOW (Log KOC = 517.8)
acrylamide	LOW (Log KOC = 10.46)
butyl acrylate	LOW (Log KOC = 40.3)
acrylic acid	HIGH (Log KOC = 1.201)
silica amorphous	LOW (Log KOC = 23.74)
titanium dioxide	LOW (Log KOC = 23.74)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product. <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> ▶ 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. ▶ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▶ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible material). ▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.
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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

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

Not Applicable

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

Product name	Group
1,2-benzisothiazoline-3-one	Not Available
5-chloro-2-methyl-4-isothiazolin-3-one	Not Available
2,6-di-tert-butyl-4-methylphenol	Not Available
styrene	Not Available
acrylamide	Not Available
butyl acrylate	Not Available
acrylic acid	Not Available
silica amorphous	Not Available
titanium dioxide	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
1,2-benzisothiazoline-3-one	Not Available
5-chloro-2-methyl-4-isothiazolin-3-one	Not Available
2,6-di-tert-butyl-4-methylphenol	Not Available
styrene	Not Available
acrylamide	Not Available
butyl acrylate	Not Available
acrylic acid	Not Available
silica amorphous	Not Available
titanium dioxide	Not Available

SECTION 15 Regulatory information

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

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)

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

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

Australian Inventory of Industrial Chemicals (AIIC)

2,6-di-tert-butyl-4-methylphenol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

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)

styrene is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

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

Continued...

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2A: Probably carcinogenic to humans

acrylamide is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2A: Probably carcinogenic to humans

butyl acrylate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

acrylic acid is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

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)

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)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (1,2-benzisothiazoline-3-one; 5-chloro-2-methyl-4-isothiazolin-3-one; styrene; acrylamide; butyl acrylate; acrylic acid)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
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	23/12/2022
Initial Date	01/09/2020

Continued...

Pebeo Deco Paint

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	01/09/2020	Composition / information on ingredients - Ingredients
3.1	23/12/2022	Classification review due to GHS Revision change.

Other information

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

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

Definitions and abbreviations

- PC - TWA: Permissible Concentration-Time Weighted Average
- PC - STEL: Permissible Concentration-Short Term Exposure Limit
- IARC: International Agency for Research on Cancer
- ACGIH: American Conference of Governmental Industrial Hygienists
- STEL: Short Term Exposure Limit
- TEEL: Temporary Emergency Exposure Limit,
- IDLH: Immediately Dangerous to Life or Health Concentrations
- ES: Exposure Standard
- OSF: Odour Safety Factor
- NOAEL: No Observed Adverse Effect Level
- LOAEL: Lowest Observed Adverse Effect Level
- TLV: Threshold Limit Value
- LOD: Limit Of Detection
- OTV: Odour Threshold Value
- BCF: BioConcentration Factors
- BEI: Biological Exposure Index
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration

- 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

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