DuoTEMP

Coltène/Whaledent AG

Version No: **1.1**
Safety data sheet according to REACH Regulation (EC) No 1907/2006, as amended by UK REACH Regulations SI 2019/758

Issue Date: **21/04/2022**
Print Date: **24/01/2023**
L.REACH.GB.EN

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

1.1. Product Identifier

Product name	DuoTEMP
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains zinc oxide)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Medical device, for dental use only Use according to manufacturer's directions.
Uses advised against	Not Applicable

1.3. Details of the manufacturer or supplier of the safety data sheet

Registered company name	**Coltène/Whaledent AG**
Address	Feldwiesenstrasse 20 Altstätten CH-9450 Switzerland
Telephone	+41 (71) 75 75 300
Fax	+41 (71) 75 75 301
Website	www.coltene.com
Email	msds@coltene.com

1.4. Emergency telephone number

Association / Organisation	**CHEMWATCH EMERGENCY RESPONSE**
Emergency telephone numbers	+44 20 3901 3542
Other emergency telephone numbers	+44 808 164 9592

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

SECTION 2 Hazards identification

2.1. Classification of the substance or mixture

Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567 [1]	H400 - Hazardous to the Aquatic Environment Acute Hazard Category 1, H318 - Serious Eye Damage/Eye Irritation Category 1, H317 - Sensitisation (Skin) Category 1, H410 - Hazardous to the Aquatic Environment Long-Term Hazard Category 1
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567*

2.2. Label elements

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

Hazard statement(s)

H318	Causes serious eye damage.
H317	May cause an allergic skin reaction.
H410	Very toxic to aquatic life with long lasting effects.

Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

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

2.3. Other hazards

Ingestion may produce health damage*.

REACH - Art.57-59: The mixture does not contain Substances of Very High Concern (SVHC) at the SDS print date.

SECTION 3 Composition / information on ingredients

3.1.Substances

See 'Composition on ingredients' in Section 3.2

3.2.Mixtures

1.CAS No 2.EC No 3.Index No 4.REACH No	**%[weight]**	**Name**	**Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567**	**SCL / M-Factor**	**Nanoform Particle Characteristics**
1.72869-86-4* 2.276-957-5 3.Not Available 4.Not Available	15-20	diurethane dimethacrylate	Hazardous to the Aquatic Environment Long-Term Hazard Category 2, Sensitisation (Skin) Category 1; H411, H317 [1]	Not Available	Not Available
1.1314-13-2 2.215-222-5 3.030-013-00-7 4.Not Available	25-35	zinc oxide	Hazardous to the Aquatic Environment Acute Hazard Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 1; H400, H410 [2]	Not Available	Not Available

1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567	SCL / M-Factor	Nanoform Particle Characteristics
1.7446-19-7 2.Not Available 3.030-006-00-9 4.Not Available	10-15	zinc sulfate monohydrate	Acute Toxicity (Oral) Category 4, Serious Eye Damage/Eye Irritation Category 1, Hazardous to the Aquatic Environment Acute Hazard Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 1; H302, H318, H400, H410 [2]	Not Available	Not Available
1.8006-90-4 2.Not Available 3.Not Available 4.Not Available	<1	peppermint oil	Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 2; H315, H317, H411, EUH019 [3]	Not Available	Not Available

Legend:	*1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567; 3. Classification drawn from C&L; * EU IOELVs available; [e] Substance identified as having endocrine disrupting properties*

SECTION 4 First aid measures

4.1. Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice.

4.2 Most important symptoms and effects, both acute and delayed

See Section 11

4.3. Indication of any immediate medical attention and special treatment needed

- Absorption of zinc compounds occurs in the small intestine.
- The metal is heavily protein bound.
- Elimination results primarily from faecal excretion.
- The usual measures for decontamination (Ipecac Syrup, lavage, charcoal or cathartics) may be administered, although patients usually have sufficient vomiting not to require them.
- CaNa2EDTA has been used successfully to normalise zinc levels and is the agent of choice.

[Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 Firefighting measures

5.1. Extinguishing media

- Foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.

- Water spray or fog - Large fires only.

5.2. Special hazards arising from the substrate or mixture

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

5.3. Advice for firefighters

Fire Fighting	‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear breathing apparatus plus protective gloves. ‣ Prevent, by any means available, spillage from entering drains or water courses. ‣ Use water delivered as a fine spray to control fire and cool adjacent area. ‣ **DO NOT** approach containers suspected to be hot. ‣ Cool fire exposed containers with water spray from a protected location. ‣ If safe to do so, remove containers from path of fire. ‣ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	Combustible. Will burn if ignited. Combustion products include: , carbon monoxide (CO) , carbon dioxide (CO2) , sulfur oxides (SOx) , sulfur dioxide (SO2) , metal oxides , other pyrolysis products typical of burning organic material.

SECTION 6 Accidental release measures

6.1. Personal precautions, protective equipment and emergency procedures

See section 8

6.2. Environmental precautions

See section 12

6.3. Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. ‣ Clean up all spills immediately. ‣ Avoid contact with skin and eyes. ‣ Wear impervious gloves and safety goggles. ‣ Trowel up/scrape up. ‣ Place spilled material in clean, dry, sealed container. ‣ Flush spill area with water.
Major Spills	‣ 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. Environmental hazard - contain spillage.

6.4. Reference to other sections

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

SECTION 7 Handling and storage

7.1. Precautions for safe handling

Safe handling	‣ Avoid all personal contact, including inhalation. ‣ Wear protective clothing when risk of exposure occurs. ‣ Use in a well-ventilated area. ‣ Prevent concentration in hollows and sumps. ‣ Avoid contact with incompatible materials. ‣ 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.
Fire and explosion protection	See section 5
Other information	‣ Store in original containers. ‣ Keep containers securely sealed. ‣ Store in a cool, dry, well-ventilated area. ‣ Store away from incompatible materials and foodstuff containers. ‣ Protect containers against physical damage and check regularly for leaks. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS.

7.2. Conditions for safe storage, including any incompatibilities

Suitable container	Recommended storage temperature: 15 - 23 °C ‣ Metal can or drum ‣ Packaging as recommended by manufacturer. ‣ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Zinc oxide: ‣ slowly absorbs carbon dioxide from the air. ‣ may react, explosively with magnesium and chlorinated rubber when heated ‣ is incompatible with linseed oil (may cause ignition) ‣ WARNING: Avoid or control reaction with peroxides. All *transition meta*l peroxides should be considered as potentially explosive. For example transition metal complexes of alkyl hydroperoxides may decompose explosively. ‣ The pi-complexes formed between chromium(0), vanadium(0) and other transition metals (haloarene-metal complexes) and mono-or poly-fluorobenzene show extreme sensitivity to heat and are explosive. ‣ Avoid reaction with borohydrides or cyanoborohydrides ‣ Avoid strong acids, bases. ‣ Avoid reaction with oxidising agents
Hazard categories in accordance with Regulation (EC) No 1272/2008	E1: Hazardous to the Aquatic Environment in Category Acute 1 or Chronic 1
Qualifying quantity (tonnes) of dangerous substances as referred to in Article 3(10) for the application of	E1 Lower- / Upper-tier requirements: 100 / 200

7.3. Specific end use(s)

See section 1.2

SECTION 8 Exposure controls / personal protection

8.1. Control parameters

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
diurethane dimethacrylate	Dermal 1.3 mg/kg bw/day (Systemic, Chronic) Inhalation 3.3 mg/m³ (Systemic, Chronic) *Dermal 0.7 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 0.6 mg/m³ (Systemic, Chronic) ** *Oral 0.3 mg/kg bw/day (Systemic, Chronic) **	0.01 mg/L (Water (Fresh)) 0.001 mg/L (Water - Intermittent release) 0.1 mg/L (Water (Marine)) 4.56 mg/kg sediment dw (Sediment (Fresh Water)) 0.46 mg/kg sediment dw (Sediment (Marine)) 0.91 mg/kg soil dw (Soil) 3.61 mg/L (STP)

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
zinc oxide	Dermal 83 mg/kg bw/day (Systemic, Chronic) Inhalation 2 mg/m³ (Systemic, Chronic) Inhalation 0.5 mg/m³ (Local, Chronic) Inhalation 2 mg/m³ (Systemic, Acute) *Dermal 83 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 1 mg/m³ (Systemic, Chronic) ** *Oral 0.83 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 1 mg/m³ (Systemic, Acute) **	0.19 µg/L (Water (Fresh)) 1.14 µg/L (Water - Intermittent release) 1.2 µg/L (Water (Marine)) 18 mg/kg sediment dw (Sediment (Fresh Water)) 6.4 mg/kg sediment dw (Sediment (Marine)) 0.7 mg/kg soil dw (Soil) 20 µg/L (STP) 0.16 mg/kg food (Oral)
zinc sulfate monohydrate	Dermal 8.3 mg/kg bw/day (Systemic, Chronic) Inhalation 1 mg/m³ (Systemic, Chronic) *Dermal 8.3 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 1.25 mg/m³ (Systemic, Chronic) ** *Oral 0.83 mg/kg bw/day (Systemic, Chronic) **	20.6 µg/L (Water (Fresh)) 6.1 µg/L (Water - Intermittent release) 117.8 mg/kg sediment dw (Sediment (Fresh Water)) 56.5 mg/kg sediment dw (Sediment (Marine)) 35.6 mg/kg soil dw (Soil) 100 µg/L (STP)
peppermint oil	Dermal 5 mg/kg bw/day (Systemic, Chronic) Inhalation 35.3 mg/m³ (Systemic, Chronic) *Dermal 2.5 mg/kg bw/day (Systemic, Chronic) ** *Inhalation 8.7 mg/m³ (Systemic, Chronic) ** *Oral 2.5 mg/kg bw/day (Systemic, Chronic) **	5.4 µg/L (Water (Fresh)) 0.54 µg/L (Water - Intermittent release) 5.77 µg/L (Water (Marine)) 1.3 mg/kg sediment dw (Sediment (Fresh Water)) 0.13 mg/kg sediment dw (Sediment (Marine)) 0.29 mg/kg soil dw (Soil) 1.8 mg/L (STP)

* Values for General Population

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Not Available	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available

Not Applicable

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
diurethane dimethacrylate	120 mg/m3	1,300 mg/m3	7,900 mg/m3
zinc oxide	10 mg/m3	15 mg/m3	2,500 mg/m3
zinc sulfate monohydrate	15 mg/m3	97 mg/m3	580 mg/m3

Ingredient	Original IDLH	Revised IDLH
diurethane dimethacrylate	Not Available	Not Available
zinc oxide	500 mg/m3	Not Available
zinc sulfate monohydrate	Not Available	Not Available
peppermint oil	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
diurethane dimethacrylate	E	≤ 0.1 ppm
zinc oxide	E	≤ 0.01 mg/m³
zinc sulfate monohydrate	E	≤ 0.01 mg/m³
peppermint oil	E	≤ 0.1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

Sensory irritants are chemicals that produce temporary and undesirable side-effects on the eyes, nose or throat. Historically occupational exposure standards for these irritants have been based on observation of workers' responses to various airborne concentrations. Present day expectations require that nearly every individual should be protected against even minor sensory irritation and exposure standards are established using uncertainty factors or safety factors of 5 to 10 or more. On occasion animal no-observable-effect-levels (NOEL) are used to determine these limits where human results are unavailable. An additional approach, typically used by the TLV committee (USA) in determining respiratory standards for this group of chemicals, has been to assign ceiling values (TLV C) to rapidly acting irritants and to assign short-term exposure limits (TLV STELs) when the weight of evidence from irritation, bioaccumulation and other endpoints combine to warrant such a limit. In contrast the MAK Commission (Germany) uses a five-category system based on intensive odour, local irritation, and elimination half-life.

However this system is being replaced to be consistent with the European Union (EU) Scientific Committee for Occupational Exposure Limits (SCOEL); this is more closely allied to that of the USA.
OSHA (USA) concluded that exposure to sensory irritants can:

- cause inflammation
- cause increased susceptibility to other irritants and infectious agents
- lead to permanent injury or dysfunction
- permit greater absorption of hazardous substances and
- acclimate the worker to the irritant warning properties of these substances thus increasing the risk of overexposure.

Fragrance substance with is an established contact allergen in humans.
Scientific Committee on Consumer Safety SCCS OPINION on Fragrance allergens in cosmetic products 2012
for zinc oxide:
Zinc oxide intoxication (intoxication zincale) is characterised by general depression, shivering, headache, thirst, colic and diarrhoea.
Exposure to the fume may produce metal fume fever characterised by chills, muscular pain, nausea and vomiting. Short-term studies with guinea pigs show pulmonary function changes and morphologic evidence of small airway inflammation. A no-observed-adverse-effect level (NOAEL) in guinea pigs was 2.7 mg/m3 zinc oxide. Based on present data, the current TLV-TWA may be inadequate to protect exposed workers although known physiological differences in the guinea pig make it more susceptible to functional impairment of the airways than humans.

Exposed individuals are **NOT** reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

Class	OSF	Description
A	550	Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities
B	26-550	As "A" for 50-90% of persons being distracted
C	1-26	As "A" for less than 50% of persons being distracted
D	0.18-1	10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached
E	<0.18	As "D" for less than 10% of persons aware of being tested

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

8.2. Exposure controls

8.2.1. Appropriate engineering controls

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

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

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

Within each range the appropriate value depends on:

	Lower end of the range / Upper end of the range 1: Room air currents minimal or favourable to capture / 1: Disturbing room air currents 2: Contaminants of low toxicity or of nuisance value only. / 2: Contaminants of high toxicity 3: Intermittent, low production. / 3: High production, heavy use 4: Large hood or large air mass in motion / 4: Small hood-local control only Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
8.2.2. Personal protection	
Eye and face protection	‣ Safety glasses with side shields. ‣ Chemical goggles. ‣ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	‣ Wear chemical protective gloves, e.g. PVC. ‣ Wear safety footwear or safety gumboots, e.g. Rubber **NOTE:** ‣ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ‣ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.
Body protection	See Other protection below
Other protection	‣ Overalls. ‣ P.V.C apron. ‣ Barrier cream. ‣ Skin cleansing cream. ‣ Eye wash unit.

Respiratory protection

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

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

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

8.2.3. Environmental exposure controls

See section 12

SECTION 9 Physical and chemical properties

9.1. Information on basic physical and chemical properties

Appearance	White

Physical state	Free-flowing Paste	**Relative density (Water = 1)**	2.5
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available
Nanoform Solubility	Not Available	**Nanoform Particle Characteristics**	Not Available
Particle Size	Not Available		

9.2. Other information

Not Available

SECTION 10 Stability and reactivity

10.1.Reactivity	See section 7.2
10.2. Chemical stability	‣ Unstable in the presence of incompatible materials. ‣ Product is considered stable. ‣ Hazardous polymerisation will not occur.
10.3. Possibility of hazardous reactions	See section 7.2
10.4. Conditions to avoid	See section 7.2
10.5. Incompatible materials	See section 7.2
10.6. Hazardous decomposition products	See section 5.3

SECTION 11 Toxicological information

11.1. Information on toxicological effects

Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Following an oral intake of extremely high doses of zinc (where 300 mg Zn/d – 20 times the US Recommended Dietary Allowance (RDA) – is a "low intake" overdose), nausea, vomiting, pain, cramps and diarrhea may occur. There is evidence of induced copper deficiency, alterations of blood lipoprotein levels, increased levels of LDL, and decreased levels of HDL at long-term intakes of 100 mg Zn/d. The USDA RDA is 15 mg Zn/d. There is also a condition called the "zinc shakes" or "zinc chills" or metal fume fever that can be induced by the inhalation of freshly formed zinc oxide formed during the welding of galvanized materials. Supplemental zinc can prevent iron absorption, leading to iron deficiency and possible peripheral neuropathy, with loss of

	sensation in extremities. Zinc is necessary for normal fetal growth and development. Fetal damage may result from zinc deficiency. Only one report in the literature suggested adverse developmental effects in humans due to exposure to excessive levels of zinc. Four women were given zinc supplements of 0.6 mg zinc/kg/day as zinc sulfate during the third trimester of pregnancy. Three of the women had premature deliveries, and one delivered a stillborn infant. However, the significance of these results cannot be determined because very few details were given regarding the study protocol, reproductive histories, and the nutritional status of the women. Other human studies have found no developmental effects in the newborns of mothers consuming 0.3 mg zinc/kg/day as zinc sulfate or zinc citrate or 0.06 mg zinc/kg/day as zinc aspartate during the last two trimesters. There has been a suggestion that increased serum zinc levels in pregnant women may be associated with an increase in neural tube defects, but others have failed to confirm this association. The developmental toxicity of zinc in experimental animals has been evaluated in a number of investigations. Exposure to high levels of zinc in the diet prior to and/or during gestation has been associated with increased fetal resorptions, reduced fetal weights, altered tissue concentrations of fetal iron and copper, and reduced growth in the offspring. Animal studies suggest that exposure to very high levels of dietary zinc is associated with reduced fetal weight, alopecia, decreased hematocrit, and copper deficiency in offspring. For example, second generation mice exposed to zinc carbonate during gestation and lactation (260 mg/kg/day in the maternal diet), and then continued on that diet for 8 weeks, had reduced body weight, alopecia, and signs of copper deficiency (e.g., lowered hematocrit and occasional achromotrichia [loss of hair colour]. Similarly, mink kits from dams that ingested a time-weighted-average dose of 20.8 mg zinc/kg/day as zinc sulfate also had alopecia and achromotrichia. It is likely that the alopecia resulted from zinc-induced copper deficiency, which is known to cause alopecia in monkeys. However, no adverse effects were observed in parental mice or mink. No effects on reproduction were reported in rats exposed to 50 mg zinc/kg/day as zinc carbonate; however, increased stillbirths were observed in rats exposed to 250 mg zinc/kg/day. Welding or flame cutting of metals with zinc or zinc dust coatings may result in inhalation of zinc oxide fume; high concentrations of zinc oxide fume may result in "metal fume fever"; also known as "brass chills", an industrial disease of short duration. [I.L.O] Symptoms include malaise, fever, weakness, nausea and may appear quickly if operations occur in enclosed or poorly ventilated areas. Genotoxicity studies conducted in a variety of test systems have failed to provide evidence for mutagenicity of zinc. However, there are indications of weak clastogenic effects following zinc exposure.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Soluble zinc salts produces irritation and corrosion of the alimentary tract (in a manner similar to copper salts) with pain, vomiting, etc. Delayed deaths have been ascribed to inanition (weakness and extreme weight loss resulting from prolonged and severe food insufficiency) following severe strictures of the oesophagus, and pylorus. Vomiting, abdominal cramps, and diarrhea, in several cases with blood, have been observed after ingestion of zinc sulfate. Several cases of gastrointestinal disturbances have been reported after ingestion of zinc sulfate. A significant reduction in erythrocyte superoxide dismutase activity (47% decrease), hematocrit, and serum ferritin, compared to pretreatment levels, occurred in female subjects who received supplements (as capsules) of 50 mg zinc/day as zinc gluconate for 10 weeks. A 15% decrease in erythrocyte superoxide dismutase activity was reported in male volunteers receiving 50 mg zinc/day as zinc gluconate for 6 weeks. Another study reported increases in bone specific alkaline phosphatase levels (~25%) and extracellular superoxide dismutase (~15%), while significant decreases were seen in mononuclear white cell 5'-nucleotidase (~30%) and plasma 5'-nucleotidase activity (~36%) following exposure of postmenopausal women to a combined (dietary+supplemental) 53 mg zinc/day as zinc glycine chelate. Healthy men given 200 mg zinc/day as elemental zinc for 6 weeks showed a reduction in lymphocyte stimulation response to phytohemagglutinin as well as chemotaxis and phagocytosis of bacteria by polymorphonuclear leukocytes.; however, no changes in lymphocyte cell number or in the proportion of lymphocyte populations were noted. Exposure of male volunteers to 0.48 mg zinc/kg/day, as zinc glycine chelate, had no effect on markers of coagulation relative to unexposed subjects. While the changes in hematological end points following long-term zinc exposure in humans are noteworthy, they were subclinical in nature, and therefore, are generally considered to be non-adverse. In animals, following oral administration of zinc compounds, decreased hemoglobin, hematocrit, erythrocyte, and/or leukocyte levels were observed in rats, mice, rabbits , dogs, ferrets, and preruminant calves A number of intermediate-duration studies have demonstrated renal effects in animals exposed to zinc oxide, zinc sulfate, and zinc acetate. Zinc sulfate caused an increase in the absolute and relative kidney weights and regressive kidney lesions (not specified) in female mice that consumed 1,110 mg zinc/kg/day in the diet for 13 weeks, but no effects occurred in rats that consumed 565 mg zinc/kg/day or in mice that consumed 104 mg zinc/kg/day under similar conditions. Severe diffuse nephrosis was observed in ferrets exposed to 195 mg zinc/kg/day as zinc oxide in the diet . In rats exposed to 191 mg zinc/kg/day as zinc acetate for 3 months, epithelial cell damage in the glomerulus and proximal convoluted tubules and increased plasma creatinine and urea levels were observed. Zinc plays a role in the normal development and maintenance of the immune system, such as in the lymphocyte response to mitogens and as a cofactor for the thymic hormone thymulin. Oral exposure to zinc at levels much higher than the recommended daily dose has impaired immune and inflammatory responses. This was observed in in vivo investigations of the immune competence of blood components taken from 11 healthy adult men after ingestion of 4.3 mg zinc/kg/day as zinc sulfate for 6 weeks. The mitogenic response elicited from peripheral blood lymphocytes and the chemotactic and phagocytic responses of polymorphonuclear leukocytes were impaired after zinc ingestion. No effects were seen on total numbers of lymphocytes or relative numbers of T cells, T cell subsets, or B cells. The relationship between these observations and decreased levels of immune competence that might lead to increased susceptibility to disease is unknown. A later study reported no effects of supplementation of male volunteers with 30 mg zinc/day (0.43 mg zinc/kg/day assuming a reference male body weight of 70 kg) as zinc glycine chelate for 14 weeks on levels of peripheral blood leucocytes or on the frequency of lymphocyte subsets. Zinc appears to be necessary for normal brain function, but excess zinc is toxic. A 16-year-old boy who ingested .86 mg zinc/kg/day of metallic zinc over a 2-day period in an attempt to promote wound healing, developed signs and symptoms of lethargy, light-headedness, staggering, and difficulty in writing clearly . Lethargy was also observed in a 2-year-old child who ingested a zinc chloride solution (.1,000 mg zinc/kg). It is not known whether these observations represent direct effects on the nervous system. Very limited data were located regarding neurological effects in animals. Minor neuron degeneration and proliferation of oligodendroglia occurred in rats dosed with 487 mg zinc/kg/day as zinc oxide for 10 days. Rats receiving 472 mg zinc/kg/day for 10 days had increased levels of secretory material in the neurosecretory nuclei of the hypothalamus. Mice

	exposed postnatally to 0.5 mg zinc/kg/day as zinc acetate for 28 days showed no changes in memory formation, but showed a gradual decrease in learning extinction throughout the study.
Skin Contact	The material may accentuate any pre-existing dermatitis condition Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions. Open cuts, abraded or irritated skin should not be exposed to this material Repeated or excessive handling, coupled with poor personal hygiene, may result in acne-like eruptions known as "zinc oxide pox". The material may produce mild skin irritation; limited evidence or practical experience suggests, that the material either: ‣ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or ‣ produces significant, but mild, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (non allergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.

DuoTEMP	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
diurethane dimethacrylate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg *[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg *[2]	Skin: no adverse effect observed (not irritating)[1]
zinc oxide	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit) : 500 mg/24 h - mild
	Inhalation(Rat) LC50: >1.79 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >5000 mg/kg[1]	Skin (rabbit) : 500 mg/24 h- mild
		Skin: no adverse effect observed (not irritating)[1]
zinc sulfate monohydrate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Not Available
	Oral (Mouse) LD50; 200 mg/kg[2]	
peppermint oil	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >5000 mg/kg[2]	Not Available
	Oral (Rat) LD50: 2426 mg/kg[2]	

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*

DuoTEMP	Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A prehapten is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems. A prohapten is a chemical that itself is non- or low-sensitising but that is transformed into a hapten in the skin (bioactivation) usually via enzyme catalysis. It is not always possible to know whether a particular allergen that is not directly reactive acts as a prehapten or as a prohapten, or both, because air oxidation and bioactivation can often give the same product (geraniol is an example). Some chemicals might act by all three pathways. **Prohaptens** Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohaptens. In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal. The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin . These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohaptens can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity. **QSAR prediction:** The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptens) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptens. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation
diurethane dimethacrylate	* Possible carcinogen; possible sensitizer; possible irreversible effects * Polysciences MSDS The skin sensitising potential of the test substance was investigated in a Local Lymph Node Assay (LLNA) in mice according to OECD Guideline 429 and in compliance with GLP (Vogel, 2009). The highest technically achievable test substance concentration was 50% (w/w) in dimethylformamide. To determine the highest non-irritant test concentration, a pre-test was performed in two animals. Two mice were treated with concentrations of 25 and 50% each on three consecutive days. No signs of irritation or systemic toxicity were observed at the tested concentrations. In the main study, four female CBA/CaOlaHsd mice per test group were treated with the test substance at concentrations of 10, 25 and 50% (w/w) in dimethylformamide or with vehicle alone for three consecutive days by open application on the ears (25 µL/ear). Three days after the last exposure, all animals were injected with 3H-methyl thymidine and approximately after five hours the draining (auricular) lymph nodes were excised and pooled for each test group. After precipitating the DNA of the lymph node cells, radioactivity measurements were performed. Treatment with test substance concentrations of 10, 25 and 50% (w/w) in dimethylformamide resulted in DPM values per lymph node of 1266.3, 1363.5 and 3562.1, respectively. The SI values calculated for the substance concentrations 10, 25 and 50% were 1.58, 1.70 and 4.44, respectively. The EC3 value was calculated to be 36.9%. Based on the results, the test substance was regarded as a skin sensitizer under the conditions of the test. Repeat Dose Toxicity: NOAEL = 100 mg/kg bw/day for males NOAEL = 300 mg/kg bw/day for females The lowest observed adverse effect level (LOAEL) in male animals is 300 mg/kg bw/day. According to Annex I of Regulation (EC) No 1272/2008 classification as STOT RE Category 2 is applicable, when significant toxic effects observed in a 90-day repeated-dose study conducted in experimental animals are seen to occur within the guidance value ranges of 10 < C = 100 mg/kg bw/day. These guidance values can be used as a basis to extrapolate equivalent guidance values for toxicity studies of greater or lesser duration, using dose/exposure time extrapolation similar to Habers rule for inhalation, which states essentially that the effective dose is directly proportional to the exposure concentration and the duration of exposure. The assessment shall be done on a case-by- case basis; for a 28-day study the guidance value is increased by a factor of three. The available repeated dose toxicity study was conducted in combination with the reproductive/developmental toxicity screening test. Male animals were exposed to the test substance for 56 days. Thus, the guidance value is increased by a factor of 1.6 leading to a guidance value range of 16 < C = 160 mg/kg bw/day for a classification as STOT RE Category 2. The LOAEL of 300 mg/kg/bw/day in the present study is above the guidance value for a classification with regard to repeated exposure. Thus, the available data on oral repeated dose toxicity do not meet the criteria for classification according to Regulation (EC) No 1272/2008, and is therefore conclusive but not sufficient for classification. Genetic toxicity: The available data on genetic toxicity are not sufficient for classification according to Regulation (EC) No 1272/2008. Gene mutation in bacteria A bacterial gene mutation assay with the test substance was performed in accordance with OECD Guideline 471 and in compliance with GLP (Paulus, 2009). In two independent experiments, the Salmonella typhimurium strains TA 97a, TA 98, TA 100, TA 102 and TA 1535 were exposed to the test

	substance dissolved in DMSO using either the preincubation or the plate incorporation method. Test substance concentrations of 50, 150, 500, 1501 and 5004 µg/plate were selected for the plate incorporation test with and without metabolic activation. In the second experiment, 312, 624, 1247, 2493 and 4986 µg/plate were selected for the preincubation method with and without metabolic activation. No signs of cytotoxicity were observed up to and including the limit concentration. Up to 5000 µg/plate, the test substance did not induce an increase in the mutation frequency of the tester strains in the presence and absence of a metabolic activation system. The determined vehicle values for the spontaneous revertants of the controls and all positive control values were within the range of historical data. Under the conditions of this experiment, the test substance did not show mutagenicity in the selected S. typhimurium strains in the presence and absence of metabolic activation. In vitro cytogenicity An in vitro micronucleus assay was performed with the test substance (Schweikl, 2001). In two independent experiments, Chinese hamster lung fibroblasts were exposed to the test substance dissolved in DMSO at concentrations of 11.75, 23.5, 35.25 µg/mL for 24 h in the absence of metabolic activation. Cytotoxicity of the test substance was observed and the TC50 value was assessed to be 24 µg/mL. At cytotoxic concentration levels of the test substance (= 24 µg/mL) the numbers of micronuclei were slightly increased in the absence of metabolic activation. Ethyl methanesulphonate was used as positive control and produced a distinct increase in micronuclei frequency indicating that the test conditions were adequate. Under the conditions of this experiment, the potential of the test substance to induce micronuclei is equivocal. In vitro mutagenicity in mammalian cells An in vitro HPRT assay was performed with the test substance (Schweikl, 1998). In three replicate cultures Chinese hamster lung fibroblasts were exposed to the test substance dissolved in DMSO at concentrations of 11.75, 23.5, 35.25 µg/mL for 24 h in the absence of metabolic activation. Cytotoxicity of the test substance was observed at concentrations = 23.5 µg/mL. No mutagenic activity of UDMA was detected. Ethyl methanesulphonate was used as positive control and produced a distinct increase in mutant frequency indicating that the test conditions were adequate. Thus, under the conditions of this experiment, the test substance did not show mutagenicity in V79 cells without metabolic activation. Due to the positive result in the in vitro micronucleus test without metabolic activation at cytotoxic concentrations a micronucleus test in vivo should be conducted to conclude on genotoxic potential of the test substance. Reproductive toxicity: The available data on toxicity to reproduction do not meet the criteria for classification according to Regulation (EC) 1272/2008, and are therefore conclusive but not sufficient for classification. reproductive toxicity: NOAEL >= 1000 mg/kg bw/day for males and females of the parental generation systemic toxicity: NOAEL = 100 mg/kg bw/day for males and 300 mg/kg bw/day for females of the parental generation A reliable sub-acute study regarding reproductive/developmental toxicity is available for the test substance. The potential reproductive or developmental toxicity of the test substance was assessed in a sub-acute combined repeated dose toxicity study with the reproductive/developmental toxicity screening test in Hsd.Han: Wistar rats performed according to OECD Guideline 422 and in compliance with GLP. Three groups of 12 male and 12 female rats received the test substance in polyethylene glycol as vehicle at doses of 100, 300 or 600 mg/kg bw/day orally via gavage at concentrations of 0, 25, 75 and 150 mg/mL corresponding to a 4 mL/kg bw dosing volume. A control group of 12 animals/sex received the vehicle only. In addition, 5 animals/sex were added to the control and high dose group to assess the reversibility of any effects observed at the high dose level (recovery group). All animals of the parental generation were dosed prior to mating (14 days) and throughout mating. In addition, males received the test item or vehicle after mating up to the day before necropsy (altogether for 56 days). Females were additionally exposed through the gestation period and up to lactation days 13 - 21, i.e. up to the day before necropsy (altogether for 56, 57 or 64 days). Observations included mortality, clinical signs, body weight, food consumption, mating, pregnancy and delivery process, lactation as well as development of offspring. The dams were allowed to litter, and rear their offspring up to day 13 post-partum. Litters were weighed and offspring were observed for possible abnormalities and were euthanized on post-natal day 13 or shortly thereafter. Blood samples were collected for determination of serum levels of thyroid hormones (T4) from all pups per litter at termination on post-natal day 13. No adverse effect on mortality, clinical signs, body weight or necropsy findings were detected in the offspring terminated as scheduled. Thyroid homone levels (T4) in pups on post-natal day 13 were not affected. The anogenital distance (male and female) or nipple retention (male) was not affected due to treatment with the test substance. For the parental animals pale livers and histopathological changes in the liver (hepatic lipidosis) were observed at 300 mg/kg bw/day for males and 1000 mg/kg bw/day for females. Thus, under the conditions of this study, the NOAEL of the test substance for systemic toxicity of the parental generation following oral administration via gavage for 56 days is 100 mg/kg bw/day in male Wistar rats. The corresponding NOAEL in female Wistar rats following oral administration via gavage for 56, 57 or 64 days is 300 mg/kg bw/day. The corresponding NOAEL for the offspring is 1000 mg/kg bw/day. * REACh Dossier UV (ultraviolet)/ EB (electron beam) acrylates are generally of low toxicity UV/EB acrylates are divided into two groups; "stenomeric" and "eurymeric" acrylates. The first group consists of well-defined acrylates which can be described by a simple idealised chemical;they are low molecular weight species with a very narrow weight distribution profile. The eurymeric acrylates cannot be described by an idealised structure and may differ fundamentally between various suppliers; they are of relatively high molecular weigh and possess a wide weight distribution. Stenomeric acrylates are usually more hazardous than the eurymeric substances. Stenomeric acrylates are also well defined which allows comparison and exchange of toxicity data - this allows more accurate classification. The stenomerics cannot be classified as a group; they exhibit substantial variation. 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 (CH2=CHCOO or CH2=C(CH3)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. 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 Monalkyl or monoarylesters of acrylic acids should be classified as R36/37/38 and R51/53 Monoalkyl or monoaryl esters of methacrylic acid should be classified as R36/37/38
PEPPERMINT OIL	Oral (rat) TDLo: 9000 mg/kg/90D-I *[Givaudan] Ataxia, respiratory depression and convulsions recorded. Bacterial mutagen. The toxicity studies of the plant have received controversial results. Some authors reported that the plant may induce hepatic diseases (liver disease), while others found that it is of protective functions against the liver damages which are caused by heavy metal inductions. In addition to that, the toxicities of the plant seem to vary from one cultivar to anotherand are dose dependent. This is probably attributed from the content level of pulegone. Some of the toxic components may come from herbicides The mechanisms by which pulegone and its proximate hepatotoxicant, menthofuran exert toxic effects have been studied

extensively both in vitro and in vivo, presumably because of the use and abuse of pennyroyal oil. Pulegone has been shown to be the active constituent of pennyroyal oil, and menthofuran produced the same toxic effects as pulegone after intraperitoneal injection to mice. These effects are similar to those reported in humans after ingestion of pennyroyal oil.
In rats given R(+)-pulegone by intraperitoneal injection at a dose of 300 mg/kg bw, the livers showed dilatation of the central veins and distension of sinusoidal spaces 6 h after treatment and centrilobular necrosis beginning at 12 h. Electron microscopic examination after 24 h revealed degeneration of the endoplasmic reticulum, swelling of mitochondria, and nuclear changes. It has been suggested that pulegone metabolites specifically deactivate cytochrome P450 isozymes by modifying the prosthetic haem- or apo- protein of the enzyme. In human liver microsomes, menthofuran specifically inhibited CYP2A6, and adducts with this enzyme have been isolated.
In a screening test for toxicity, menthofuran was added to the diet of rats at a concentration resulting in an average daily intake of 23 mg/kg bw for 14 days. No effects on body-weight gain, food consumption, liver or kidney weights, or gross histological appearance of the liver and kidney were seen
While there is good evidence that 8-pulegone aldehyde is the ultimate toxicant, there is also evidence that this metabolite of menthofuran accounts for only some of the toxicity of pulegone.
Assays for genotoxicity have been performed with pulegone and menthofuran.. Pulegone did not induce reverse mutation in Salmonella typhimurium strain TA1537, TA1535, TA100, TA98, or TA97, with or without metabolic activation, at concentrations up to 800 ug/plate. Neither substance was mutagenic in S. typhimurium strains TA100 and TA98 at concentrations up to 1000 ug/plate, with or without metabolic activation. In a study of the insecticidal properties of mint oils, concentrations of pulegone in excess of the LD50 value for Drosophila larvae (0.17 uL) induced a slight increase in the frequency of wing mutations (mosaic spots) over that induced by control solutions
d-Limonene is readily absorbed by inhalation and ingestion. Dermal absorption is reported to be lower than by the inhalation route. d-Limonene is rapidly distributed to different tissues in the body, readily metabolised and eliminated primarily through the urine.
Limonene exhibits low acute toxicity by all three routes in animals. Limonene is a skin irritant in both experimental animals and humans. Limited data are available on the potential to cause eye and respiratory irritation. Autooxidised products of d-limonene have the potential to be skin sensitisers. Limited data are available in humans on the potential to cause respiratory sensitisation. Autooxidation of limonene occurs readily in the presence of light and air forming a variety of oxygenated monocyclic terpenes. Risk of skin sensitisation is high in situations where contact with oxidation products of limonene occurs.
Renal tumours induced by limonene in male rats is though to be sex and species specific and are not considered relevant to humans. Repeated exposure affects the amount and activity of liver enzymes, liver weight, blood cholesterol levels and bile flow in animals. Increase in liver weight is considered a physiological adaption as no toxic effects on the liver have been reported. From available data it is not possible to identify an NOAEL for these effects. Limonene is neither genotoxic or teratogenic nor toxic to the reproductive system.
Cross-reactivity is also expected between ester derivatives and their parent alcohols, as the esters will be hydrolysed by esterases in the skin. Esters of important contact allergens that can be activated by hydrolysis in the skin are isoeugenol acetate, eugenyl acetate and geranyl acetate all of which are known to be used as fragrance ingredients.
Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A **prehapten** is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems.
In the case of prehaptens, it is possible to prevent activation outside the body to a certain extent by different measures, e.g. prevention of air exposure during handling and storage of the ingredients and the final product, and by the addition of suitable antioxidants. When antioxidants are used, care should be taken that they will not be activated themselves and thereby form new sensitisers.

Prehaptens

Most terpenes with oxidisable allylic positions can be expected to autoxidise on air exposure due to their inherent properties. Depending on the stability of the oxidation products that are formed, a difference in the sensitisation potency of the oxidised terpenes can be seen
Autoxidation is a free radical chain reaction in which hydrogen atom abstraction in combination with addition of oxygen forms peroxyl radicals. The reaction shows selectivity for positions where stable radicals can be formed. So far, all fragrance substances that have been investigated with regard to the influence of autoxidation on the allergenic potential, including identification of formed oxidation products, have oxidisable allylic positions that are able to form hydroperoxides and/or hydrogen peroxide as primary oxidation products upon air exposure. Once the hydroperoxides have been formed outside the skin they form specific antigens and act as skin sensitisers. Secondary oxidation products such as aldehydes and epoxides can also be allergenic, thus further increasing the sensitisation potency of the autoxidation mixture. The process of photoactivation may also play a role, but further research is required to establish whether this activation route is currently underestimated in importance due to insufficient knowledge of the true haptens in this context.
It should be noted that activation of substances via air oxidation results in various haptens that might be the same or cross-reacting with other haptens (allergens). The main allergens after air oxidation of linalool and linalyl acetate are the hydroperoxides. If linalyl acetate is chemically hydrolysed outside the skin it can thereafter be oxidised to the same haptens as seen for linalool. A corresponding example is citronellol and citronellyl acetate. In clincal studies, concomitant reactions to oxidised linalool and oxidised linalyl acetate have been observed. Whether these reactions depend on cross-reactivity or are due to exposure to both fragrance substances cannot be elucidated as both have an allergenic effect themselves. Linalool and linalyl acetate are the main components of lavender oil. They autoxidise on air exposure also when present in the essential oil, and form the same oxidation products found in previous studies of the pure synthetic terpenes. Experimental sensitisation studies showed that air exposure of lavender oil increased the sensitisation potency. Patch test results in dermatitis patients showed a connection between positive reactions to oxidised linalool, linalyl acetate and lavender oil.

Prohaptens

Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohaptens.
In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal.

	The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin . These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohaptens can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity. **QSAR prediction:** The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptens) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptens. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation.
DuoTEMP & diurethane dimethacrylate & PEPPERMINT OIL	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. The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
DuoTEMP & PEPPERMINT OIL	Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and connubial contact dermatitis occur. Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes. Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits. Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water. Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis. Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a suffcient degree of fragrance contact allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the allergen. As a consequence, symptoms, i.e. allergic contact dermatitis, may occur upon re-exposure to the fragrance allergen(s) in question. Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or hands. It may affect fitness for work and the quality of life of the individual. Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease Fragrance contact

	allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure. **Hands:** Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema may often be complicated by sensitisation .Fragrance allergy may be a relevant problem in patients with hand eczema; perfumes are present in consumer products to which their hands are exposed. A significant relationship between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe) chronic hand eczema may not be clear. **Axillae Bilateral axillary** (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy. **Face** Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of of being fragrance allergic. **Irritant reactions (including contact urticaria):** Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this, Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported . The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested , but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen. **Pigmentary anomalies:** The term “pigmented cosmetic dermatitis” was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified.. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil. **Photo-reactions** Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon . Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. Phototoxic reactions still occur but are rare. **General/respiratory:** Fragrances are volatile and therefore, in addition to skin exposure, a perfume also exposes the eyes and naso-respiratory tract. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma . Asthma-like symptoms can be provoked by sensory mechanisms. In an epidemiological investigation, a significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.
diurethane dimethacrylate	Combined repeated dose toxicity study with the reproduction/developmental toxicity screening test, oral (OECD 422), rat:
ZINC OXIDE & PEPPERMINT OIL	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.
ZINC SULFATE MONOHYDRATE & PEPPERMINT OIL	No significant acute toxicological data identified in literature search.

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

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

11.2 Information on other hazards

11.2.1. Endocrine Disruption Properties

Not Available

11.2.2. Other Information

See Section 11.1

SECTION 12 Ecological information

12.1. Toxicity

DuoTEMP

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

diurethane dimethacrylate

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	0.21mg/l	2
EC50	72h	Algae or other aquatic plants	>0.68mg/l	2
LC50	96h	Fish	10.1mg/l	Not Available
EC50	48h	Crustacea	>1.2mg/l	2

zinc oxide

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1344h	Fish	19-110	7
LC50	96h	Fish	0.112mg/l	2
EC50	72h	Algae or other aquatic plants	0.036-0.049mg/l	4
EC50	48h	Crustacea	0.105mg/l	2
EC10(ECx)	168h	Algae or other aquatic plants	0.0025mg/l	2
EC50	96h	Algae or other aquatic plants	0.3mg/l	2

zinc sulfate monohydrate

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1344h	Fish	59-112	7
EC20(ECx)	72h	Algae or other aquatic plants	0.001-0.075mg/l	4
EC50	96h	Algae or other aquatic plants	0.0101mg/l	4
EC50	72h	Algae or other aquatic plants	0.01-0.122mg/l	4
LC50	96h	Fish	0.000017-0.000034mg/l	4
EC50	48h	Crustacea	0.06mg/l	4

peppermint oil

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	96h	Algae or other aquatic plants	2.61mg/l	2
EC50	96h	Algae or other aquatic plants	2.61mg/l	2
LC50	96h	Fish	3.4mg/l	2
EC50	48h	Crustacea	2.7mg/l	2
EC50(ECx)	48h	Crustacea	2.43mg/l	2
EC50	96h	Algae or other aquatic plants	2.63mg/l	2
EC50	48h	Crustacea	2.43mg/l	2
LC50	96h	Fish	3.01mg/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*

Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
for inorganic sulfates:
Environmental fate:
Data from tap water studies with human volunteers indicate that sulfates produce a laxative effect at concentrations of 1000 - 1200 mg/litre, but no increase in diarrhoea, dehydration or weight loss. The presence of sulfate in drinking-water can also result in a noticeable taste; the lowest taste threshold concentration for sulfate is approximately 250 mg/litre as the sodium salt. Sulfate may also contribute to the corrosion of distribution systems. No health-based guideline value for sulfate in drinking water is proposed. However, there is an increasing likelihood of complaints arising from a noticeable taste as concentrations in water increase above 500 mg/litre.
Sulfates are removed from the air by both dry and wet deposition processes. Wet deposition processes including rain-out (a process that occurs within the clouds) and washout (removal by precipitation below the clouds) contribute to the removal of sulfate from the atmosphere.
In soil, the inorganic sulfates can adsorb to soil particles or leach into surface water and groundwater. Sulfates can be taken up by plants and be incorporated into the parenchyma of the plant.
Sulfate in water can also be reduced by sulfate bacteria (*Thiobacilli*) which use them as a source of energy.

In anaerobic environments sulfate is biologically reduced to (hydrogen) sulfide by sulfate reducing bacteria, or incorporated into living organisms as source of sulfur, and thereby included in the sulfur cycle. Sodium sulfate is not reactive in aqueous solution at room temperature. Sodium sulfate will completely dissolve, ionise and distribute across the entire planetary "aquasphere". Some sulfates may eventually be deposited, the majority of sulfates participate in the sulfur cycle in which natural and industrial sodium sulfate are not distinguishable

The BCF of sodium sulfate is very low and therefore significant bioconcentration is not expected. Sodium and sulfate ions are essential to all living organisms and their intracellular and extracellular concentrations are actively regulated. However some plants (e.g. corn and *Kochia Scoparia*), are capable of accumulating sulfate to concentrations that are potentially toxic to ruminants.

Ecotoxicity:

For sulfate in general:

Fish LC50: toxic from 7000 mg/l

Bacteria: toxic from 2500 mg/l

Algae were shown to be the most sensitive to sodium sulfate; EC50 120 h = 1,900 mg/l. For invertebrates *(Daphnia magna)* the EC50 48 h = 4,580 mg/l and fish appeared to be the least sensitive with a LC50 96h = 7,960 mg/l for *Pimephales promelas*. Activated sludge showed a very low sensitivity to sodium sulfate. There was no effect up to 8 g/l. Sodium sulfate is not very toxic to terrestrial plants. *Picea banksiana* was the most sensitive species, an effect was seen at 1.4 g/l. Sediment dwelling organisms were not very sensitive either, with an LC50 96h = 660 mg/l for *Trycorythus sp*. Overall it can be concluded that sodium sulfate has no acute adverse effect on aquatic and sediment dwelling organisms. Toxicity to terrestrial plants is also low.

No data were found for long term toxicity. The acute studies all show a toxicity of sodium sulfate higher than 100 mg/l, no bioaccumulation is expected,

For zinc and its compounds:

Environmental fate:

Zinc is capable of forming complexes with a variety of organic and inorganic groups (ligands). Biological activity can affect the mobility of zinc in the aquatic environment, although the biota contains relatively little zinc compared to the sediments. Zinc bioconcentrates moderately in aquatic organisms; bioconcentration is higher in crustaceans and bivalve species than in fish. Zinc does not concentrate appreciably in plants, and it does not biomagnify significantly through terrestrial food chains.

However biomagnification may be of concern if concentration of zinc exceeds 1632 ppm in the top 12 inches of soil.

Zinc can persist in water indefinitely and can be toxic to aquatic life. The threshold concentration for fish is 0.1 ppm. Zinc may be concentrated in the aquatic food chain; it is concentrated over 200,000 times in oysters. Copper is synergistic but calcium is antagonistic to zinc toxicity in fish. Zinc can accumulate in freshwater animals at 5 -1,130 times the concentration present in the water. Furthermore, although zinc actively bioaccumulates in aquatic systems, biota appears to represent a relatively minor sink compared to sediments. Steady-state zinc bioconcentration factors (BCFs) for 12 aquatic species range from 4 to 24,000. Crustaceans and fish can accumulate zinc from both water and food. A BCF of 1,000 was reported for both aquatic plants and fish, and a value of 10,000 was reported for aquatic invertebrates. The order of enrichment of zinc in different aquatic organisms was as follows (zinc concentrations in µg/g dry weight appear in parentheses): fish (25), shrimp (50), mussel (60), periphyton (260), zooplankton (330), and oyster (3,300). The high enrichment in oysters may be due to their ingestion of particulate matter containing higher concentrations of zinc than ambient water. Other investigators have also indicated that organisms associated with sediments have higher zinc concentrations than organisms living in the aqueous layer. With respect to bioconcentration from soil by terrestrial plants, invertebrates, and mammals, BCFs of 0.4, 8, and 0.6, respectively, have been reported. The concentration of zinc in plants depends on the plant species, soil pH, and the composition of the soil.

Plant species do not concentrate zinc above the levels present in soil.

In some fish, it has been observed that the level of zinc found in their bodies did not directly relate to the exposure concentrations. Bioaccumulation of zinc in fish is inversely related to the aqueous exposure. This evidence suggests that fish placed in environments with lower zinc concentrations can sequester zinc in their bodies.

The concentration of zinc in drinking water may increase as a result of the distribution system and household plumbing. Common piping materials used in distribution systems often contain zinc, as well as other metals and alloys. Trace metals may enter the water through corrosion products or simply by the dissolution of small amounts of metals with which the water comes in contact. Reactions with materials of the distribution system, particularly in soft low-pH waters, very often have produced concentrations of zinc in tap water much greater than those in the raw or treated waters at the plant of origin. Zinc gives water a metallic taste at low levels. Overexposures to zinc also have been associated with toxic effects. Ingestion of zinc or zinc-containing compounds has resulted in a variety of systemic effects in the gastrointestinal and hematological systems and alterations in the blood lipid profile in humans and animals. In addition, lesions have been observed in the liver, pancreas, and kidneys of animals.

Environmental toxicity of zinc in water is dependent upon the concentration of other minerals and the pH of the solution, which affect the ligands that associate with zinc.

Zinc occurs in the environment mainly in the +2 oxidation state. Sorption is the dominant reaction, resulting in the enrichment of zinc in suspended and bed sediments. Zinc in aerobic waters is partitioned into sediments through sorption onto hydrous iron and manganese oxides, clay minerals, and organic material. The efficiency of these materials in removing zinc from solution varies according to their concentrations, pH, redox potential (Eh), salinity, nature and concentrations of complexing ligands, cation exchange capacity, and the concentration of zinc. Precipitation of soluble zinc compounds appears to be significant only under reducing conditions in highly polluted water. Generally, at lower pH values, zinc remains as the free ion. The free ion (Zn+2) tends to be adsorbed and transported by suspended solids in unpolluted waters.

Zinc is an essential nutrient that is present in all organisms. Although biota appears to be a minor reservoir of zinc relative to soils and sediments, microbial decomposition of biota in water can produce ligands, such as humic acids, that can affect the mobility of zinc in the aquatic environment through zinc precipitation and adsorption.

The relative mobility of zinc in soil is determined by the same factors that affect its transport in aquatic systems (i.e., solubility of the compound, pH, and salinity) The redox status of the soil may shift zinc partitioning. Reductive dissolution of iron and manganese (hydr)oxides under suboxic conditions release zinc into the aqueous phase; the persistence of suboxic conditions may then lead to a repartitioning of zinc into sulfide and carbonate solids. The mobility of zinc in soil depends on the solubility of the speciated forms of the element and on soil properties such as cation exchange capacity, pH, redox potential, and chemical species present in soil; under anaerobic conditions, zinc sulfide is the controlling species.

Since zinc sulfide is insoluble, the mobility of zinc in anaerobic soil is low. In a study of the effect of pH on zinc solubility: When the pH is <7, an inverse relationship exists between the pH and the amount of zinc in solution. As negative charges on soil surfaces increase with increasing pH, additional sites for zinc adsorption are activated and the amount of zinc in solution decreases. The active zinc species in the adsorbed state is the singly charged zinc hydroxide species (i.e., Zn[OH]+). Other investigators have also shown that the mobility of zinc in soil increases at lower soil pH under oxidizing conditions and at a lower cation exchange capacity of soil. On the other hand, the amount of zinc in solution generally increases when the pH is >7 in soils high in organic matter. This is a result of the release of organically complexed zinc, reduced zinc adsorption at higher pH, or an increase in the concentration of chelating agents in soil. For calcareous soils, the relationship between zinc solubility and pH is nonlinear. At a high pH, zinc in solution is precipitated as Zn(OH)2, zinc carbonate (ZnCO3), or calcium

zincate. Clay and metal oxides are capable of sorbing zinc and tend to retard its mobility in soil. Zinc was more mobile at pH 4 than at pH 6.5 as a consequence of sorption
Zinc concentrations in the air are relatively low, except near industrial sources such as smelters. No estimate for the atmospheric lifetime of zinc is available at this time, but the fact that zinc is transported long distances in air indicates that its lifetime in air is at least on the order of days. There are few data regarding the speciation of zinc released to the atmosphere. Zinc is removed from the air by dry and wet deposition, but zinc particles with small diameters and low densities suspended in the atmosphere travel long distances from emission sources.
DO NOT discharge into sewer or waterways.

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
zinc sulfate monohydrate	HIGH	HIGH

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
zinc oxide	LOW (BCF = 217)
zinc sulfate monohydrate	LOW (BCF = 112)

12.4. Mobility in soil

Ingredient	Mobility
zinc sulfate monohydrate	LOW (KOC = 6.124)

12.5. Results of PBT and vPvB assessment

	P	B	T
Relevant available data	Not Available	Not Available	Not Available
PBT	✗	✗	✗
vPvB	✗	✗	✗

PBT Criteria fulfilled?	No
vPvB	No

12.6. Endocrine Disruption Properties

Not Available

12.7. Other adverse effects

One or more ingredients within this SDS has the potential of causing ozone depletion and/or photochemical ozone creation.

SECTION 13 Disposal considerations

13.1. Waste treatment methods

Product / Packaging disposal	Dispose of waste according to applicable legislation. Special country-specific regulations may apply. Can be disposed together with household waste in compliance with official regulations in contact with approved waste disposal companies and with authorities in charge. (Only dispose of completely emptied packages.) ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.
Waste treatment options	Not Available
Sewage disposal options	Not Available

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	2Z

Land transport (ADR-RID)

14.1. **UN number**	3077
14.2. **UN proper shipping name**	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains zinc oxide)
14.3. **Transport hazard class(es)**	Class: 9 Subrisk: Not Applicable
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Hazard identification (Kemler): 90 Classification code: M7 Hazard Label: 9 Special provisions: 274 335 375 601 Limited quantity: 5 kg Tunnel Restriction Code: 3 (-)

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	3077
14.2. **UN proper shipping name**	Environmentally hazardous substance, solid, n.o.s. (contains zinc oxide)
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 9 ICAO / IATA Subrisk: Not Applicable ERG Code: 9L
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: A97 A158 A179 A197 A215 Cargo Only Packing Instructions: 956 Cargo Only Maximum Qty / Pack: 400 kg Passenger and Cargo Packing Instructions: 956 Passenger and Cargo Maximum Qty / Pack: 400 kg Passenger and Cargo Limited Quantity Packing Instructions: Y956 Passenger and Cargo Limited Maximum Qty / Pack: 30 kg G

Sea transport (IMDG-Code / GGVSee)

14.1. **UN number**	3077
14.2. **UN proper shipping name**	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains zinc oxide)
14.3. **Transport hazard class(es)**	IMDG Class: 9 IMDG Subrisk: Not Applicable
14.4. **Packing group**	III

14.5. **Environmental hazard**	Marine Pollutant
14.6. **Special precautions for user**	EMS Number: F-A, S-F Special provisions: 274 335 966 967 969 Limited Quantities: 5 kg

Inland waterways transport (ADN)

14.1. **UN number**	3077
14.2. **UN proper shipping name**	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains zinc oxide)
14.3. **Transport hazard class(es)**	9 Not Applicable
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Classification code: M7 Special provisions: 274; 335; 375; 601 Limited quantity: 5 kg Equipment required: PP, A*** Fire cones number: 0

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

Not Applicable

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

Product name	Group
diurethane dimethacrylate	Not Available
zinc oxide	Not Available
zinc sulfate monohydrate	Not Available
peppermint oil	Not Available

14.9. Transport in bulk in accordance with the ICG Code

Product name	Ship Type
diurethane dimethacrylate	Not Available
zinc oxide	Not Available
zinc sulfate monohydrate	Not Available
peppermint oil	Not Available

SECTION 15 Regulatory information

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

diurethane dimethacrylate is found on the following regulatory lists

Not Applicable

zinc oxide is found on the following regulatory lists

Great Britain GB Biocidal Active Substances
Great Britain GB mandatory classification and labelling list (GB MCL)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
UK REACH grandfathered registrations notified substances list

zinc sulfate monohydrate is found on the following regulatory lists

Great Britain GB mandatory classification and labelling list (GB MCL)
UK REACH grandfathered registrations notified substances list

peppermint oil is found on the following regulatory lists

Great Britain GB Biocidal Active Substances
UK REACH grandfathered registrations notified substances list

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : Directives 98/24/EC, - 92/85/EEC, - 94/33/EC,

- 2008/98/EC, - 2010/75/EU; Commission Regulation (EU) 2020/878; Regulation (EC) No 1272/2008 as updated through ATPs.

Information according to 2012/18/EU (Seveso III):

Seveso Category	E1

15.2. Chemical safety assessment

No Chemical Safety Assessment has been carried out for this substance/mixture by the supplier.

ECHA SUMMARY

Ingredient	CAS number	Index No	ECHA Dossier
diurethane dimethacrylate	72869-86-4*	Not Available	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Skin Sens. 1	Wng	H317
2	Skin Sens. 1B; Aquatic Chronic 2; Skin Irrit. 2; Eye Irrit. 2; STOT SE 3	GHS07; GHS09; Wng	H317; H411; H315; H319; H335

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
zinc oxide	1314-13-2	030-013-00-7	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Aquatic Acute 1; Aquatic Chronic 1	GHS09; Wng	H410
2	Aquatic Acute 1; Aquatic Chronic 1; Repr. 1A; STOT SE 3; STOT SE 1; STOT RE 1; Acute Tox. 2; Acute Tox. 2; Skin Sens. 1; Eye Dam. 1; Muta. 2; Carc. 1A; Skin Corr. 1B	GHS09; GHS08; Dgr; GHS06; GHS05	H410; H360; H400; H335; H370; H372; H300; H330; H317; H318; H341; H350; H314
1	Acute Tox. 4; Eye Dam. 1; Acute Tox. 4; Carc. 1A; Repr. 1A; Lact.; STOT RE 1; Aquatic Acute 1; Aquatic Chronic 1	GHS08; GHS09; GHS05; Dgr	H302; H332; H315; H318; H350; H360; H373; H410
2	Acute Tox. 4; Eye Dam. 1; Acute Tox. 4; Carc. 1A; Repr. 1A; Lact.; STOT RE 1; Aquatic Acute 1; Aquatic Chronic 1	GHS08; GHS09; GHS05; Dgr	H302; H332; H315; H318; H350; H360; H373; H410
1	Not Classified	Not Available	Not Available
2	Not Classified	Not Available	Not Available

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
zinc sulfate monohydrate	7446-19-7	030-006-00-9	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Acute Tox. 4; Eye Dam. 1; Aquatic Acute 1; Aquatic Chronic 1	GHS09; GHS05; Dgr	H302; H318; H410
2	Acute Tox. 4; Eye Dam. 1; Aquatic Acute 1; Aquatic Chronic 1; Skin Irrit. 2	GHS09; GHS05; Dgr	H302; H318; H410; H400; H314
1	Acute Tox. 4; Eye Dam. 1; Aquatic Acute 1; Aquatic Chronic 1	GHS05; GHS09; Dgr	H302; H318; H410
2	Acute Tox. 4; Eye Dam. 1; Aquatic Acute 1; Aquatic Chronic 1; Acute Tox. 4; Skin Sens. 1; Carc. 1A; STOT RE 2; Acute Tox. 4; Skin Irrit. 2; STOT SE 3	GHS05; GHS09; Dgr; GHS08	H302; H318; H410; H400; H312; H317; H350; H372; H315; H335

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
peppermint oil	8006-90-4	Not Available	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Skin Irrit. 2; Skin Sens. 1; Aquatic Chronic 2	GHS07; GHS09; Wng	H315; H317; H411
2	Skin Irrit. 2; Skin Sens. 1A; Aquatic Chronic 2; Acute Tox. 4; Eye Irrit. 2; Asp. Tox. 1	GHS09; GHS08; Dgr	H315; H317; H411; H319; H304; H301; H310; H400

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Skin Irrit. 2; Aquatic Chronic 3	GHS07; Wng	H315; H412
2	Skin Irrit. 2; Skin Sens. 1B; Eye Irrit. 2; Aquatic Chronic 2; Asp. Tox. 1; Flam. Liq. 3; Acute Tox. 4; Resp. Sens. 1; STOT SE 3	GHS09; GHS08; Dgr; GHS02	H315; H317; H411; H304; H318; H226; H401; H302; H334; H335; H312; H332
1	Skin Irrit. 2; Skin Sens. 1; Aquatic Chronic 2	GHS07; GHS09; Wng	H315; H317; H411
2	Acute Tox. 4; Skin Irrit. 2; Skin Sens. 1A; Eye Irrit. 2; Asp. Tox. 1; Flam. Liq. 3; Aquatic Acute 1; Aquatic Chronic 1	GHS09; Dgr; GHS08; GHS02	H302; H315; H317; H319; H304; H226; H400; H410
1	Skin Irrit. 2; Skin Sens. 1; Aquatic Chronic 2	GHS07; GHS09; Wng	H315; H317; H411
2	Skin Irrit. 2; Skin Sens. 1; Aquatic Chronic 2; Eye Irrit. 2	GHS07; GHS09; Wng	H315; H317; H411; H319

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (diurethane dimethacrylate)
Canada - NDSL	No (zinc sulfate monohydrate; peppermint oil)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (diurethane dimethacrylate; peppermint oil)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (diurethane dimethacrylate)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (diurethane dimethacrylate)
Vietnam - NCI	Yes
Russia - FBEPH	No (diurethane dimethacrylate)
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	21/04/2022
Initial Date	14/02/2022

Full text Risk and Hazard codes

H226	Flammable liquid and vapour.
H300	Fatal if swallowed.
H301	Toxic if swallowed.
H302	Harmful if swallowed.
H304	May be fatal if swallowed and enters airways.
H310	Fatal in contact with skin.
H312	Harmful in contact with skin.
H314	Causes severe skin burns and eye damage.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H330	Fatal if inhaled.
H332	Harmful if inhaled.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H335	May cause respiratory irritation.

H341	Suspected of causing genetic defects.
H350	May cause cancer.
H360	May damage fertility or the unborn child.
H370	Causes damage to organs.
H372	Causes damage to organs through prolonged or repeated exposure.
H373	May cause damage to organs through prolonged or repeated exposure.
H400	Very toxic to aquatic life.
H401	Toxic to aquatic life.
H411	Toxic to aquatic life with long lasting effects.
H412	Harmful to aquatic life with long lasting effects.

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.

For detailed advice on Personal Protective Equipment, refer to the following EU CEN Standards:
EN 166 Personal eye-protection
EN 340 Protective clothing
EN 374 Protective gloves against chemicals and micro-organisms
EN 13832 Footwear protecting against chemicals
EN 133 Respiratory protective devices

Definitions and abbreviations

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

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