



JTC IMPORT/EXPORT P/L

Personal Care Spiking Glue Mega Hold 3 oz

JTC Import Export Pty Ltd

Chemwatch: 7963-10

Version No: 2.1

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

Chemwatch Hazard Alert Code: 2

Issue Date: 30/05/2025

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S.GHS.AUS.EN

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

Product Identifier

Product name	Personal Care Spiking Glue Mega Hold 3 oz
Chemical Name	Not Applicable
Synonyms	Not Available
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Cosmetic use , Cosmetic use . Use according to manufacturer's directions. SDS are intended for use in the workplace ONLY. For domestic-use products, refer to consumer labels.
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Details of the manufacturer or importer of the safety data sheet

Registered company name	JTC Import Export Pty Ltd
Address	98-126 South Park Drive Dandenong South VIC 3175 Australia
Telephone	+61 3 9532 5100
Fax	+61 3 9532 6102
Website	https://www.jtcimportexport.com.au
Email	sales@jtcimportexport.com.au

Emergency telephone number

Association / Organisation	JTC Import Export Pty Ltd	CHEMWATCH EMERGENCY RESPONSE (24/7)
Emergency telephone number(s)	+61 3 9532 5100 (Mon-Thurs 8.30am to 5.30pm; Friday 8.30am to 3pm)	+61 1800 951 288 (ID#: 7963-10)
Other emergency telephone number(s)	Not Available	+61 3 9573 3188

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification ^[1]	Non hazardous
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	Not Applicable
Signal word	Not Applicable

Hazard statement(s)

Not Applicable

Precautionary statement(s) Prevention

Not Applicable

Precautionary statement(s) Response

Not Applicable

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

Not Applicable

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures		
CAS No	%[weight]	Name
9003-39-8	15-30	vinylpyrrolidone homopolymer
50-70-4	10-15	D-sorbitol
8009-03-8.	1-5	petrolatum
9004-62-0	0.5-1	hydroxyethylcellulose
25086-89-9	0.5-1	vinylpyrrolidone/ vinyl acetate copolymer
67762-27-0	0.5-1	cetostearyl alcohol
9003-04-7	0.1-0.5	sodium polyacrylate
8012-89-3	0.1-0.5	beeswax
9087-61-0	0.1-0.5	aluminium starch octenylsuccinate
122-99-6	0.1-0.5	ethylene glycol phenyl ether
9005-00-9	0.1-0.5	polyethylene glycol (2) stearyl ether
1310-73-2	0.05-0.1	sodium hydroxide
36653-82-4	0.05-0.1	cetyl alcohol
112-92-5	0.05-0.1	stearyl alcohol
57-55-6	0.05-0.1	propylene glycol
102972-64-5	0.01-0.05	dimethylaminoethyl methacrylate/ PVP/ N-vinylcaprolactam
6259-76-3	0.01-0.05	hexyl salicylate
55406-53-6	0.01-0.05	3-iodo-2-propynyl butyl carbamate
20298-69-5	0.01-0.05	2-tert-butylcyclohexyl acetate
140-11-4	0.01-0.05	benzyl acetate
104-67-6	0.01-0.05	gamma-undecalactone
10094-34-5	<0.01	alpha.alpha-dimethylphenethyl butyrate
1222-05-5	<0.01	galaxolide
142-19-8	<0.01	allyl heptanoate
5989-27-5	<0.01	d-limonene
99-49-0	<0.01	carvone
68039-49-6	<0.01	2,4-dimethyl-3-cyclohexene-1-carboxaldehyde
106-24-1	<0.01	geraniol
63449-41-2	<0.01	benzyl C8-18 alkyltrimethylammonium chloride
78-70-6	<0.01	linalool
128-37-0	<0.01	2,6-di-tert-butyl-4-methylphenol
91-64-5	<0.01	coumarin
Legend:		1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available

SECTION 4 First aid measures

Description of first aid measures	
Eye Contact	If this product comes in contact with the eyes: ▶ Wash out immediately with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▶ Other measures are usually unnecessary.
Ingestion	▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

for salicylate intoxication:

- Pending gastric lavage, use emetics such as syrup of Ipecac or delay gastric emptying and absorption by swallowing a slurry of activated charcoal. **Do not give ipecac after charcoal.**
- Gastric lavage with water or perhaps sodium bicarbonate solution (3%-5%). Mild alkali delays salicylate absorption from the stomach and perhaps slightly from the duodenum.
- Saline catharsis with sodium or magnesium sulfate (15-30 gm in water).
- Take an immediate blood sample for an appraisal of the patient's acid-base status. A pH determination on an anaerobic sample of arterial blood is best. An analysis of the plasma salicylate concentration should be made at the same time. Laboratory controls are almost essential for the proper management of severe salicylism.
- In the presence of an established acidosis, alkali therapy is essential, but at least in an adult, alkali should be withheld until its need is demonstrated by chemical analysis. The intensity of treatment depends on the intensity of acidosis. In the presence of vomiting, intravenous sodium bicarbonate is the most satisfactory of all alkali therapy.
- Correct dehydration and hypoglycaemia (if present) by the intravenous administration of glucose in water or in isotonic saline. The administration of glucose may also serve to remedy ketosis which is often seen in poisoned children.
- Even in patients without hypoglycaemia, infusions of glucose adequate to produce distinct hyperglycaemia are recommended to prevent glucose depletion in the brain. This recommendation is based on impressive experimental data in animals.
- Renal function should be supported by correcting dehydration and incipient shock. Overhydration is not justified. An alkaline urine should be maintained by the administration of alkali if necessary with care to prevent a severe systemic alkalosis. As long as urine remains alkaline (pH above 7.5), administration of an osmotic diuretic such as mannitol or perhaps THAM is useful, but one must be careful to avoid hypokalaemia. Supplements of potassium chloride should be included in parenteral fluids.
- Small doses of barbiturates, diazepam, paraldehyde, or perhaps other sedatives (but probably not morphine) may be required to suppress extreme restlessness and convulsions.
- For hyperpyrexia, use sponge baths.

The presence of petechiae or other signs of haemorrhagic tendency calls for a large Vitamin K dose and perhaps ascorbic acid. Minor transfusions may be necessary since bleeding in salicylism is not always due to a prothrombin effect.

Haemodialysis and haemoperfusion have proved useful in salicylate poisoning, as have peritoneal dialysis and exchange transfusions, but alkaline diuretic therapy is probably sufficient except in fulminating cases.

[GOSSELIN, *et al.*: *Clinical Toxicology of Commercial Products*]

The mechanism of the toxic effect involves metabolic acidosis, respiratory alkalosis, hypoglycaemia, and potassium depletion. Salicylate poisoning is characterised by extreme acid-base disturbances, electrolyte disturbances and decreased levels of consciousness. There are differences between acute and chronic toxicity and a varying clinical picture which is dependent on the age of the patient and their kidney function. The major feature of poisoning is metabolic acidosis due to "uncoupling of oxidative phosphorylation" which produces an increased metabolic rate, increased oxygen consumption, increased formation of carbon dioxide, increased heat production and increased utilisation of glucose. Direct stimulation of the respiratory centre leads to hyperventilation and respiratory alkalosis. This leads to compensatory increased renal excretion of bicarbonate which contributes to the metabolic acidosis which may coexist or develop subsequently. Hypoglycaemia may occur as a result of increased glucose demand, increased rates of tissue glycolysis, and impaired rate of glucose synthesis. **NOTE:** Tissue glucose levels may be lower than plasma levels. Hyperglycaemia may occur due to increased glycogenolysis. Potassium depletion occurs as a result of increased renal excretion as well as intracellular movement of potassium.

Salicylates competitively inhibit vitamin K dependent synthesis of factors II, VII, IX, X and in addition, may produce a mild dose dependent hepatitis. Salicylates are bound to albumin. The extent of protein binding is concentration dependent (and falls with higher blood levels). This, and the effects of acidosis, decreasing ionisation, means that the volume of distribution increases markedly in overdose as does CNS penetration. The extent of protein binding (50-80%) and the rate of metabolism are concentration dependent. Hepatic clearance has zero order kinetics and thus the therapeutic half-life of 2-4.5 hours but the half-life in overdose is 18-36 hours. Renal excretion is the most important route in overdose. Thus when the salicylate concentrations are in the toxic range there is increased tissue distribution and impaired clearance of the drug.

HyperTox 3.0 <https://www.ozemail.com.au/-ouad/SALI0001.HTA>

Treat symptomatically.

SECTION 5 Firefighting measures

Extinguishing media

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

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
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Advice for firefighters

Fire Fighting	▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear full body protective clothing with breathing apparatus. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ Avoid spraying water onto liquid pools. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	▶ Combustible. ▶ Slight fire hazard when exposed to heat or flame. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. ▶ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO₂) hydrogen iodide nitrogen oxides (NO_x) metal oxides other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Continued...

Minor Spills	- Remove all ignition sources. - Clean up all spills immediately. - Avoid breathing vapours and contact with skin and eyes. - Control personal contact with the substance, by using protective equipment. - Contain and absorb spill with sand, earth, inert material or vermiculite. - Wipe up. - Place in a suitable, labelled container for waste disposal.
Major Spills	Moderate hazard. - Clear area of personnel and move upwind. - Alert Fire Brigade and tell them location and nature of hazard. - Wear breathing apparatus plus protective gloves. - Prevent, by any means available, spillage from entering drains or water course. - No smoking, naked lights or ignition sources. - Increase ventilation. - Stop leak if safe to do so. - Contain spill with sand, earth or vermiculite. - Collect recoverable product into labelled containers for recycling. - Absorb remaining product with sand, earth or vermiculite. - Collect solid residues and seal in labelled drums for disposal. - Wash area and prevent runoff into drains. - If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	DO NOT allow clothing wet with material to stay in contact with skin - Avoid all personal contact, including inhalation. - Wear protective clothing when risk of exposure occurs. - Use in a well-ventilated area. - Prevent concentration in hollows and sumps. DO NOT enter confined spaces until atmosphere has been checked. - Avoid smoking, naked lights or ignition sources. - Avoid contact with incompatible materials. - When handling, DO NOT eat, drink or smoke. - Keep containers securely sealed when not in use. - Avoid physical damage to containers. - Always wash hands with soap and water after handling. - Work clothes should be laundered separately. - 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.
Other information	Store in the dark. - Store in original containers. - Keep containers securely sealed. - No smoking, naked lights or ignition sources. - Store in a cool, dry, well-ventilated area. - Store away from incompatible materials and foodstuff containers. - Protect containers against physical damage and check regularly for leaks. - Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	- Metal can or drum - Packaging as recommended by manufacturer. - Check all containers are clearly labelled and free from leaks.
Storage incompatibility	- Avoid storage with reducing agents. Cellulose and its derivatives may react vigorously with calcium oxide, bleaching powder, perchlorates, perchloric acid, sodium chlorate, fluorine, nitric acid, sodium nitrate and sodium nitrite. May be incompatible with aminacrine hydrochloride, chlorocresol, mercuric chloride, phenol, resorcinol, tannic acid and silver nitrate. - Avoid strong acids, bases. - Avoid reaction with oxidising agents, bases and strong reducing agents.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	petrolatum	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	sodium hydroxide	Sodium hydroxide	Not Available	Not Available	2 mg/m3	Not Available
Australia Exposure Standards	propylene glycol	Propane-1,2-diol total: (vapour & particulates)	150 ppm / 474 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	propylene glycol	Propane-1,2-diol: particulates only	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	2,6-di-tert-butyl-4-methylphenol	2,6-Di-tert-butyl-p-cresol	10 mg/m3	Not Available	Not Available	Not Available
Ingredient	Original IDLH		Revised IDLH			
vinylpyrrolidone homopolymer	Not Available		Not Available			

Ingredient	Original IDLH	Revised IDLH
D-sorbitol	Not Available	Not Available
petrolatum	2,500 mg/m3	Not Available
hydroxyethylcellulose	Not Available	Not Available
vinylpyrrolidone/ vinyl acetate copolymer	Not Available	Not Available
cetostearyl alcohol	Not Available	Not Available
sodium polyacrylate	Not Available	Not Available
beeswax	Not Available	Not Available
aluminium starch octenylsuccinate	Not Available	Not Available
ethylene glycol phenyl ether	Not Available	Not Available
polyethylene glycol (2) stearyl ether	Not Available	Not Available
sodium hydroxide	10 mg/m3	Not Available
cetyl alcohol	Not Available	Not Available
stearyl alcohol	Not Available	Not Available
propylene glycol	Not Available	Not Available
dimethylaminoethyl methacrylate/ PVP/ N-vinylcaprolactam	Not Available	Not Available
hexyl salicylate	Not Available	Not Available
3-iodo-2-propynyl butyl carbamate	Not Available	Not Available
2-tert-butylcyclohexyl acetate	Not Available	Not Available
benzyl acetate	Not Available	Not Available
gamma-undecalactone	Not Available	Not Available
alpha, alpha-dimethylphenethyl butyrate	Not Available	Not Available
galaxolide	Not Available	Not Available
allyl heptanoate	Not Available	Not Available
d-limonene	Not Available	Not Available
carvone	Not Available	Not Available
2,4-dimethyl-3-cyclohexene-1-carboxaldehyde	Not Available	Not Available
geraniol	Not Available	Not Available
benzyl C8-18 alkyl dimethyl ammonium chloride	Not Available	Not Available
linalool	Not Available	Not Available
2,6-di-tert-butyl-4-methylphenol	Not Available	Not Available
coumarin	Not Available	Not Available

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. An approved self contained breathing apparatus (SCBA) may be required in some situations. Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.	
	Type of Contaminant:	Air Speed:
	solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)
	Within each range the appropriate value depends on:	
	Lower end of the range	Upper end of the range
	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity

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	3: Intermittent, low production. 3: High production, heavy use 4: Large hood or large air mass in motion 4: Small hood-local control only Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
Individual protection measures, such as personal protective equipment	
Eye and face protection	▶ Safety glasses with side shields. ▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].
Skin protection	See Hand protection below
Hands/feet protection	▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Body protection	See Other protection below
Other protection	▶ Overalls. ▶ P.V.C apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the: **"Forsberg Clothing Performance Index"**.
The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:
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Material	CPI
BUTYL	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C

Respiratory protection

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

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

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

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NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE	C
PE/EVAL/PE	C
PVA	C
PVC	C
SARANEX-23	C
SARANEX-23 2-PLY	C
TEFLON	C
VITON	C
VITON/CHLOROBUTYL	C

^ - Full-face

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

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

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Off white liquid with characteristic odour; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	6.8-7.3	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 Applicable
Flash point (°C)	Not Available	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	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	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Heat of Combustion (kJ/g)	Not Available	Ignition Distance (cm)	Not Available
Flame Height (cm)	Not Available	Flame Duration (s)	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	Enclosed Space Ignition Deflagration Density (g/m3)	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

a) Acute Toxicity	Based on available data, the classification criteria are not met.
b) Skin Irritation/Corrosion	Based on available data, the classification criteria are not met.

Continued...

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c) Serious Eye Damage/Irritation	Based on available data, the classification criteria are not met.
d) Respiratory or Skin sensitisation	Based on available data, the classification criteria are not met.
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	Based on available data, the classification criteria are not met.
g) Reproductivity	Based on available data, the classification criteria are not met.
h) STOT - Single Exposure	Based on available data, the classification criteria are not met.
i) STOT - Repeated Exposure	Based on available data, the classification criteria are not met.
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Swallowing 50 grams of sorbitol has a laxative effect, with vomiting and diarrhoea. Swallowing may also cause hives. Swallowing 20 grams per day has led to severe diarrhoea and weight loss. Sorbitol may also worsen irritable bowel syndrome and similar gastrointestinal conditions. Even swallowing a small amount may result in severe abdominal pain. High oral doses of salicylates, such as aspirin, may cause a mild burning pain in the throat and stomach, causing vomiting. This is followed (within hours) by deep, rapid breathing, tiredness, nausea and further vomiting, thirst and diarrhoea. High molecular weight material; on single acute exposure would be expected to pass through gastrointestinal tract with little change / absorption. Occasionally accumulation of the solid material within the alimentary tract may result in formation of a bezoar (concretion), producing discomfort.
Skin Contact	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. There is some evidence to suggest that this material can cause inflammation of the skin on contact in some persons. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream, through, for example, cuts, abrasions or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	This material causes serious eye irritation.
Chronic	There has been some concern that this material can cause cancer or mutations but there is not enough data to make an assessment. Substance accumulation, in the human body, may occur and may cause some concern following repeated or long-term occupational exposure. There is some evidence that inhaling this product is more likely to cause a sensitisation reaction in some persons compared to the general population. Polyvinylpyrrolidone (PVP) is generally well-tolerated. It has been shown to cause sarcoma in rats but there is no evidence that it causes cancer in humans. The material may cause irritation or skin inflammation in some individuals upon prolonged contact. This material contains a substantial amount of polymer considered to be of low concern. These are classified under having MWs of between 1000 to 10000 with less than 25% of molecules with MWs under 1000 and less than 10% under 500; or having a molecular weight average of over 10000. Animal testing showed that sorbitol caused thickening of bone and excretion of citrate. Sorbitol was not shown to cause birth defects. Otherwise, sorbitol was not shown to cause toxic effects at concentrations up to 15%. Chronic exposure to salicylates produce problems with metabolism, central nervous system disturbances, or kidney damage. Those with pre-existing damage to the eye, skin or kidney are especially at risk. Although polymer with a molecular weight of more than 10000 are normally considered to be of low concern, this does not apply to water-absorbing polymers. A two year cancer study on rats, with high molecular weight polyacrylate (1 million), with no reactive functional group, showed an increase in lung tumours and scarring.

Personal Care Spiking Glue Mega Hold 3 oz	TOXICITY	IRRITATION
	Not Available	Not Available
vinylpyrrolidone homopolymer	TOXICITY	IRRITATION
	Inhalation (Rat) LC50: >5.2 mg/L4h ^[2] Oral (Rabbit) LD50; 1040 mg/kg ^[2]	Not Available
D-sorbitol	TOXICITY	IRRITATION
	Oral (Rat) LD50: 15900 mg/kg ^[2]	Not Available
petrolatum	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[1] Oral (Rat) LD50: >5000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1] Skin: no adverse effect observed (not irritating) ^[1]
hydroxyethylcellulose	TOXICITY	IRRITATION
	Not Available	Not Available
vinylpyrrolidone/ vinyl acetate copolymer	TOXICITY	IRRITATION
	Oral (Rat) LD50: >630 mg/kg ^[2]	Not Available
cetostearyl alcohol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >8000 mg/kg ^[1] Inhalation (Rat) LC50: >0.375 mg/l4h ^[1] Oral (Rat) LD50: >2000 mg/kg ^[1]	Eye (Rodent - rabbit): 25mg - Mild Eye: no adverse effect observed (not irritating) ^[1] Skin (Rodent - rabbit): 500mg - Mild

Personal Care Spiking Glue Mega Hold 3 oz

		Skin: no adverse effect observed (not irritating) ^[1]
sodium polyacrylate	TOXICITY	IRRITATION
	Oral (Mouse) LD50: >16000 mg/kg ^[2]	Eye (Rodent - rabbit): 2mg - Moderate
beeswax	TOXICITY	IRRITATION
	Not Available	Not Available
aluminium starch octenylsuccinate	TOXICITY	IRRITATION
	Not Available	Not Available
ethylene glycol phenyl ether	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (Rodent - rabbit): 250ug/24H - Severe
	Oral (Rat) LD50: 1260 mg/kg ^[2]	Eye (Rodent - rabbit): 6mg - Moderate
		Eye: adverse effect observed (irreversible damage) ^[1]
		Eye: adverse effect observed (irritating) ^[1]
		Skin (Rodent - rabbit): 500mg/24H - Mild
		Skin: adverse effect observed (irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
polyethylene glycol (2) stearyl ether	TOXICITY	IRRITATION
	Oral (Rat) LD50: 1900 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin (Human - man): 20%/2D - Moderate
sodium hydroxide		Skin: no adverse effect observed (not irritating) ^[1]
	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 1350 mg/kg ^[2]	Eye (Primate - monkey): 1%/24H - Severe
	Oral (Rabbit) LD50: 325 mg/kg ^[1]	Eye (Rodent - rabbit): 1% - Severe
		Eye (Rodent - rabbit): 100mg
		Eye (Rodent - rabbit): 1mg/24H - Severe
		Eye (Rodent - rabbit): 1mg/30S - Severe
		Eye (Rodent - rabbit): 400ug - Mild
		Eye (Rodent - rabbit): 50ug/24H - Severe
		Eye: adverse effect observed (irritating) ^[1]
		Skin (Human): 0.15%/96H
		Skin (Human): 2%/24H - Mild
		Skin (Human): 2.50%/24H
cetyl alcohol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1660 mg/kg ^[1]	Eye (Rodent - rabbit): 82mg - Mild
	Inhalation (Rat) LC50: >0.237 mg/l4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Skin (Human - man): 50mg/48H - Mild
		Skin (Human): 0.2% - Severe
		Skin (Human): 225mg/3D - Mild
		Skin (Human): 75mg/3D (intermittent) - Mild
		Skin (Rodent - guinea pig): 100% - Mild
		Skin (Rodent - guinea pig): 100mg/24H - Moderate
		Skin (Rodent - rabbit): 0.05mL/24H - Mild
		Skin (Rodent - rabbit): 100mg/24H - Severe
stearyl alcohol		Skin (Rodent - rabbit): 2600mg/24H - Mild
		Skin (Rodent - rat): 100mg/24H - Severe
		Skin: no adverse effect observed (not irritating) ^[1]
	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1660 mg/kg ^[1]	Eye (Rodent - rabbit): 100mg/24H - Mild
	Inhalation (Rat) LC50: >0.237 mg/l4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Skin (Human - man): 30%/2D - Mild
		Skin (Human): 30%
		Skin (Rodent - rabbit): 500mg/24H - Mild

		Skin: no adverse effect observed (not irritating) ^[1]
propylene glycol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 11890 mg/kg ^[2]	Eye (Rodent - rabbit): 100mg - Mild
	Inhalation (Rat) LC50: >44.9 mg/l4h ^[1]	Eye (Rodent - rabbit): 500mg/24H - Mild
	Oral (Rat) LD50: 20000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin (Human - child): 30%/96H(continuous) - Moderate
		Skin (Human - man): 10%/2D
		Skin (Human - woman): 30%/96H - Mild
		Skin (Human): 104mg/3D (intermittent) - Moderate
		Skin (Human): 20%
		Skin (Human): 500mg/7D - Mild
		Skin: no adverse effect observed (not irritating) ^[1]
dimethylaminoethyl methacrylate/ PVP/ N-vinylcaprolactam	TOXICITY	IRRITATION
	Not Available	Not Available
hexyl salicylate	TOXICITY	IRRITATION
	dermal (rat) LD50: >5000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >5000 mg/kg ^[2]	Skin: no adverse effect observed (not irritating) ^[1]
3-iodo-2-propynyl butyl carbamate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[1]	Eye: adverse effect observed (irreversible damage) ^[1]
	Inhalation (Rat) LC50: 0.63 mg/l4h ^[1]	Skin (Human): 0.3%/48H
	Oral (Rat) LD50: 1056 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
2-tert-butylcyclohexyl acetate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	Eye (Rodent - rabbit): 50% - Severe
	Oral (Rat) LD50: 4600 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin (Rodent - rabbit): 100%/4H - Moderate
		Skin: no adverse effect observed (not irritating) ^[1]
benzyl acetate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: 2490 mg/kg ^[2]	Skin (Rodent - rabbit): 100mg/24H - Moderate
		Skin: no adverse effect observed (not irritating) ^[1]
gamma-undecalactone	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: 18500 mg/kg ^[2]	Skin (Rodent - guinea pig): 100mg/24H - Moderate
		Skin (Rodent - rabbit): 100mg/24H - Severe
		Skin: no adverse effect observed (not irritating) ^[1]
alpha,alpha-dimethylphenethyl butyrate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >5000 mg/kg ^[2]	Skin (Rodent - rabbit): 500mg/24H - Moderate
		Skin: adverse effect observed (irritating) ^[1]
galaxolide	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Skin (Rodent - rabbit): 500mg/24H - Moderate
		Skin: no adverse effect observed (not irritating) ^[1]
allyl heptanoate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 810 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Guinea) LD50: 444 mg/kg ^[2]	Skin (Human): 20mg/48H - Mild
		Skin (Rodent - rabbit): 500mg/24H - Moderate
		Skin: no adverse effect observed (not irritating) ^[1]
d-limonene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]

	Oral (Rat) LD50: >2000 mg/kg ^[1]	Skin (Rodent - mouse): 700mg/7D (intermittent) - Severe
		Skin (Rodent - rabbit): 10%/24H - Mild
		Skin (Rodent - rabbit): 500mg/24H - Moderate
		Skin (Rodent - rat): 100%/1H
		Skin: no adverse effect observed (not irritating) ^[1]
carvone	TOXICITY	IRRITATION
	Oral (Guinea) LD50; 766 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin (Rodent - rabbit): 500mg/24H - Mild
		Skin: no adverse effect observed (not irritating) ^[1]
2,4-dimethyl-3-cyclohexene-1-carboxaldehyde	TOXICITY	IRRITATION
	Not Available	Not Available
geraniol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	Eye: adverse effect observed (irreversible damage) ^[1]
	Oral (Rat) LD50: 3600 mg/kg ^[2]	Skin (Human - man): 16mg/24H - Severe
		Skin (Human): 0.2mL/4H
		Skin (Human): 1%
		Skin (Human): 2%/2D
		Skin (Human): 32%/48H - Severe
		Skin (Rodent - guinea pig): 100mg/24H - Severe
		Skin (Rodent - guinea pig): 30% - Mild
		Skin (Rodent - rabbit): 0.5mL/4H - Moderate
		Skin (Rodent - rabbit): 100mg/24H - Severe
benzyl C8-18 alkyldimethylammonium chloride	TOXICITY	IRRITATION
	dermal (rat) LD50: 1420 mg/kg ^[2]	Not Available
	Oral (Mouse) LD50; 150 mg/kg ^[2]	
linalool	TOXICITY	IRRITATION
	dermal (rat) LD50: 5610 mg/kg ^[2]	Eye (Rodent - rabbit): 0.1mL/1H - Moderate
	Oral (Rat) LD50: 2790 mg/kg ^[2]	Eye (Rodent - rabbit): 100uL - Moderate
		Eye: adverse effect observed (irritating) ^[1]
		Skin (Human - man): 16mg/48H - Mild
		Skin (Human): 10%/2D
		Skin (Human): 32%/72H - Mild
		Skin (Rodent - guinea pig): 100mg/24H - Moderate
2,6-di-tert-butyl-4-methylphenol	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (Rodent - rabbit): 100mg/24H - Moderate
	Oral (Rat) LD50: 890 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin (Human): 500mg/48H - Mild
		Skin (Rodent - rabbit): 500mg/48H - Moderate
coumarin	TOXICITY	IRRITATION
	dermal (rat) LD50: 293 mg/kg ^[1]	Skin (Human - man): 5%
	Oral (Rat) LD50: ~290 mg/kg ^[1]	Skin (Human): 5%/2D

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

VINYLPYRROLIDONE HOMOPOLYMER	Chronic toxicity ** Genetic toxicity: No mutagenic effect was found in various tests with microorganisms and mammalian cell culture. The substance was not mutagenic in studies with mammals. Carcinogenicity: In long-term animal studies in which the substance was given in high doses by feed, a carcinogenic effect was not observed. Developmental toxicity/teratogenicity: No indications of a developmental toxic / teratogenic effect were seen in animal studies * ISP MSDS **BASF MSDS
PETROLATUM	Dermal (rabbit) TDLo: 100 ml/kg/30D-I Tumorigenic effects.

	"Hydrocarbon wax" describes a group of solid C20 to C36 paraffinic hydrocarbons which are not absorbed in the gastro-intestinal tract and in small quantity will pass through undigested. Refined waxes are used widely in cosmetic surgery over many years and this demonstrates their low toxicity; many guidelines exist for their safe use. However, occasionally there are reports of adverse effects with these products. Deposits under the skin, referred to as "paraffinoma" have been described, but these are not normally associated with other progressive changes. Long-term toxicity studies indicated that petroleum-derived paraffin and microcrystalline waxes are non-toxic and do not cause cancer.
VINYLPYRROLIDONE/ VINYL ACETATE COPOLYMER	28 day dietary feeding study in rats. NOEL=1000mg/kg/day (highest dose tested). 90 day dietary feeding study in rats. NOEL=1000mg/kg/day (highest dose tested).* ISP MSDS
SODIUM POLYACRYLATE	Polycarboxylates are of low toxicity by all exposure routes examined. Homopolymers(P-AA) are of low acute toxicity to the rat (LD50 > 5 g/kg bw/d) and are not irritating to the rabbit s skin and, at the most, slightly irritating to the eye. Further P-AA has no sensitising potential. The adverse effect after repeated inhalation dosing (91-d/rat) was a mild, reversible pulmonary irritation. This effect is considered as not substance related owing to the physical property of the respirable dust, which caused local and not systemic lung effects. There was neither evidence for a genotoxic potential of PAA using a variety of genetic endpoints in-vitro and in-vivo,nor for developmental toxicity or reprotoxicity in the rat. Based upon the available data, it is considered that exposure to polycarboxylates does not imply any particular hazard to humans The Cosmetic Ingredient Review (CIR) Expert Panel noted that these crosslinked alkyl acrylates are macromolecules that are not expected to pass through the stratum corneum of the skin, so significant dermal absorption is not expected. Therefore, topically applied cosmetics are not expected to result in systemic or reproductive and developmental toxicity or to have genotoxic or carcinogenic effects upon use. The Panel noted that cosmetic products containing these ingredients are reportedly used around the eyes, on the lips, and on other mucous membranes. Thus, crosslinked alkyl acrylates could be absorbed systemically through the relatively moist,n stratum cornea of the conjunctiva, lips,and other mucous membranes, and through ingestion when applied to the lips. However, the Panel noted that any absorption through healthy intact mucous membranes is likely to be not significant,primarily because of the relatively large molecular sizes. Furthermore, the chemically inert nature of the polymers precludes degradation to smaller absorbable species. Absorption of the polymers and their residual monomers in cosmetic products also would be limited after application to the lips or eye area based on the relatively small fractions of the applied products that might be inadvertently ingested or make direct contact with the conjunctiva. The Carbomers (Carbopols) are synthetic, high molecular weight, nonlinear polymers of acrylic acid, cross-linked with a polyalkenyl polyether. The Carbomer polymers are used in cosmetics and emulsifying agents at concentrations up to 50%. Acute oral animal studies showed that Carbomers-910, -934, -934P, -940, and -941 have low toxicities when ingested. Rabbits showed minimal skin irritation and zero to moderate eye irritation when tested with Carbomers-910 and -934. Subchronic feeding of rats and dogs with Carbomer-934 in the diet resulted in lower than normal body weights, but no pathological changes were observed. Dogs chronically fed Carbomer-934P manifested gastrointestinal irritation and marked pigment deposition within Kupffer cells of the liver. Clinical studies with Carbomers showed that these polymers have low potential for skin irritation and sensitization at concentrations up to 100%. Carbomer-934 demonstrated low potential for phototoxicity and photo-contact allergenicity. On the basis of the available information presented and as qualified in the report, it is concluded that the Carbomers are safe as cosmetic ingredients. Little toxicity data is available for acrylic crosspolymers; the acute dermal and oral toxicity data that were found indicated that these ingredients are not very toxic. The little genotoxicity data that were available reported negative results in Ames tests. Carcinogenicity data were not found in the published literature for the polymers, but data were available for the monomers. In an alternative method study, acrylates/vinyl neodecanoate crosspolymer was predicted to be a non-irritant. The non-human studies reported no to slight irritation with undiluted and weak sensitization with 2% aq., acrylates/C10-30 alkyl acrylate crosspolymer, no irritation with acrylates crosspolymer at 30% in olive oil, and no irritation or sensitization with sodium acrylates crosspolymer-2 (concentration not specified). Mostly, human testing with undiluted acrylates/C10-30 alkyl acrylate crosspolymer, acrylates crosspolymer, and acrylates/ethylhexyl acrylate crosspolymer, up to 2.5% aq. acrylates/vinyl isodecanoate crosspolymer, 1% aq. dilutions of formulations containing 2% acrylates/vinyl neodecanoate crosspolymer, and formulations containing up to 2.6% lauryl methacrylate/glycol dimethacrylate crosspolymers do not indicate any dermal irritation or sensitization. The only exception was a weak irritant response noted during an intensified Shelanski human repeated insult patch test (HRIPT) with undiluted acrylates/C10-30 alkyl acrylate crosspolymer. Alternative test methods for ocular irritation indicated that acrylates/vinyl isodecanoate crosspolymer and a formulation containing 1% lauryl methacrylate/glycol dimethacrylate crosspolymer are not likely ocular irritants. In studies using rabbits, undiluted acrylates/C10-30 alkyl acrylate crosspolymer produced minimal to moderate irritation, and it was considered a borderline irritant in unrnshed rabbit eyes. Acrylates crosspolymer, at 50% in olive oil,and sodium acrylates crosspolymer-2 did not appear to be ocular irritants in rabbit eyes.Two different risk assessments evaluating the carcinogenic endpoint for benzene that may be present in acrylates/ C10-30 alkyl acrylates crosspolymer resulted in different lifetime risk. One found that the risk was within the range associated with a 10exp 6 cancer risk, while the other reported a 20-fold greater risk. Final Safety Assessment: Crosslinked Alkyl Acrylates as Used in Cosmetics. Nov 2011 Cosmetic Ingredient Review (CIR) Expert Panel https://ntp.niehs.nih.gov/ntp/roc/nominations/2013/publiccomm/attachmencir_508.pdf The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
BEESWAX	Group A aliphatic monoesters (fatty acid esters) cause very little or no injury and are considered safe for use in cosmetics.
ALUMINIUM STARCH OCTENYLSUCCINATE	For aluminium compounds: Aluminium present in food and drinking water is poorly absorbed through the gastrointestinal tract. The bioavailability of aluminium is dependent on the form in which it is ingested and the presence of dietary constituents with which the metal cation can complex Ligands in food can have a marked effect on absorption of aluminium, as they can either enhance uptake by forming absorbable (usually water soluble) complexes (e.g., with carboxylic acids such as citric and lactic), or reduce it by forming insoluble compounds (e.g., with phosphate or dissolved silicate). Considering the available human and animal data it is likely that the oral absorption of aluminium can vary 10-fold based on chemical form alone. Although bioavailability appears to generally parallel water solubility, insufficient data are available to directly extrapolate from solubility in water to bioavailability. For oral intake from food, the European Food Safety Authority (EFSA) has derived a tolerable weekly intake (TWI) of 1 milligram (mg) of aluminium per kilogram of bodyweight. In its health assessment, the EFSA states a medium bioavailability of 0.1 % for all aluminium compounds which are ingested with food. This corresponds to a systemically available tolerable daily dose of 0.143 microgrammes (µg) per kilogramme (kg) of body weight. This means that for an adult weighing 60 kg, a systemically available dose of 8.6 µg per day is considered safe. Based on a neuro-developmental toxicity study of aluminium citrate administered via drinking water to rats, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) established a Provisional Tolerable Weekly Intake (PTWI) of 2 mg/kg bw (expressed as aluminium) for all aluminium compounds in food, including food additives. The Committee on Toxicity of chemicals in food, consumer products and the environment (COT) considers that the derivation of this PTWI was sound and that it should be used in assessing potential risks from dietary exposure to aluminium. The Federal Institute for Risk Assessment (BfR) of Germany has assessed the estimated aluminium absorption from antiperspirants. For this purpose, the data, derived from experimental studies, on dermal absorption of aluminium from antiperspirants for healthy and damaged skin was used as a basis. At about 10.5 µg, the calculated systemic intake values for healthy skin are above the 8.6 µg per day that are considered safe for an adult weighing 60 kg. If aluminium -containing antiperspirants are used on a daily basis, the tolerable weekly intake determined by the EFSA is therefore exceeded. The values for damaged skin, for example injuries from shaving, are many times higher. This means that in case of daily use of an aluminium-containing antiperspirant alone, the TWI may be completely exhausted. In addition, further aluminium absorption sources such as food, cooking utensils and other cosmetic products must be taken into account Systemic toxicity after repeated exposure No studies were located regarding dermal effects in animals following intermediate or chronic-duration dermal exposure to various forms of aluminium. When orally administered to rats, aluminium compounds (including aluminium nitrate, aluminium sulfate and potassium aluminium sulfate) have produced various effects, including decreased gain in body weight and mild histopathological changes in the spleen, kidney and liver of

rats (104 mg Al/kg bw/day) and dogs (88-93 mg Al/kg bw/day) during subchronic oral exposure. Effects on nerve cells, testes, bone and stomach have been reported at higher doses. Severity of effects increased with dose.

The main toxic effects of aluminium that have been observed in experimental animals are neurotoxicity and nephrotoxicity. Neurotoxicity has also been described in patients dialysed with water containing high concentrations of aluminium, but epidemiological data on possible adverse effects in humans at lower exposures are inconsistent

Reproductive and developmental toxicity:

Studies of reproductive toxicity in male mice (intraperitoneal or subcutaneous administration of aluminium nitrate or chloride) and rabbits (administration of aluminium chloride by gavage) have demonstrated the ability of aluminium to cause testicular toxicity, decreased sperm quality in mice and rabbits and reduced fertility in mice. No reproductive toxicity was seen in females given aluminium nitrate by gavage or dissolved in drinking water. Multi-generation reproductive studies in which aluminium sulfate and aluminium ammonium sulfate were administered to rats in drinking water, showed no evidence of reproductive toxicity

High doses of aluminium compounds given by gavage have induced signs of embryotoxicity in mice and rats in particular, reduced fetal body weight or pup weight at birth and delayed ossification. Developmental toxicity studies in which aluminium chloride was administered by gavage to pregnant rats showed evidence of foetotoxicity, but it was unclear whether the findings were secondary to maternal toxicity. A twelve-month neuro-development with aluminium citrate administered via the drinking water to Sprague-Dawley rats, was conducted according to Good Laboratory Practice (GLP). Aluminium citrate was selected for the study since it is the most soluble and bioavailable aluminium salt. Pregnant rats were exposed to aluminium citrate from gestational day 6 through lactation, and then the offspring were exposed post-weaning until postnatal day 364. An extensive functional observational battery of tests was performed at various times. Evidence of aluminium toxicity was demonstrated in the high (300 mg/kg bw/day of aluminium) and to a lesser extent, the mid-dose groups (100 mg/kg bw/day of aluminium). In the high-dose group, the main effect was renal damage, resulting in high mortality in the male offspring. No major neurological pathology or neurobehavioural effects were observed, other than in the neuromuscular subdomain (reduced grip strength and increased foot splay). Thus, the lowest observed adverse effect level (LOAEL) was 100 mg/kg bw/day and the no observed adverse effect level (NOAEL) was 30 mg/kg bw/day. Bioavailability of aluminium chloride, sulfate and nitrate and aluminium hydroxide was much lower than that of aluminium citrate This study was used by JECFA as key study to derive the PTWI.

Genotoxicity

Aluminium compounds were non-mutagenic in bacterial and mammalian cell systems, but some produced DNA damage and effects on chromosome integrity and segregation in vitro. Clastogenic effects were also observed in vivo when aluminium sulfate was administered at high doses by gavage or by the intraperitoneal route. Several indirect mechanisms have been proposed to explain the variety of genotoxic effects elicited by aluminium salts in experimental systems. Cross-linking of DNA with chromosomal proteins, interaction with microtubule assembly and mitotic spindle functioning, induction of oxidative damage, damage of lysosomal membranes with liberation of DNAase, have been suggested to explain the induction of structural chromosomal aberrations, sister chromatid exchanges, chromosome loss and formation of oxidized bases in experimental systems. The EFSA Panel noted that these indirect mechanisms of genotoxicity, occurring at relatively high levels of exposure, are unlikely to be of relevance for humans exposed to aluminium via the diet. Aluminium compounds do not cause gene mutations in either bacteria or mammalian cells. Exposure to aluminium compounds does result in both structural and numerical chromosome aberrations both in in-vitro and in-vivo mutagenicity tests. DNA damage is probably the result of indirect mechanisms. The DNA damage was observed only at high exposure levels.

Carcinogenicity.

The available epidemiological studies provide limited evidence that certain exposures in the aluminium production industry are carcinogenic to humans, giving rise to cancer of the lung and bladder. However, the aluminium exposure was confounded by exposure to other agents including polycyclic aromatic hydrocarbons, aromatic amines, nitro compounds and asbestos. There is no evidence of increased cancer risk in non-occupationally exposed persons.

Neurodegenerative diseases.

Following the observation that high levels of aluminium in dialysis fluid could cause a form of dementia in dialysis patients, a number of studies were carried out to determine if aluminium could cause dementia or cognitive impairment as a consequence of environmental exposure over long periods. Aluminium was identified, along with other elements, in the amyloid plaques that are one of the diagnostic lesions in the brain for Alzheimer disease, a common form of senile and pre-senile dementia. Some of the epidemiology studies suggest the possibility of an association of Alzheimer disease with aluminium in water, but other studies do not confirm this association. All studies lack information on ingestion of aluminium from food and how concentrations of aluminium in food affect the association between aluminium in water and Alzheimer disease." There are suggestions that persons with some genetic variants may absorb more aluminium than others, but there is a need for more analytical research to determine whether aluminium from various sources has a significant causal association with Alzheimer disease and other neurodegenerative diseases. Aluminium is a neurotoxicant in experimental animals. However, most of the animal studies performed have several limitations and therefore cannot be used for quantitative risk assessment.

Contact sensitivity:

It has been suggested that the body burden of aluminium may be linked to different diseases. Macrophagic myofasciitis and chronic fatigue syndrome can be caused by aluminium-containing adjuvants in vaccines. Macrophagic myofasciitis (MMF) has been described as a disease in adults presenting with ascending myalgia and severe fatigue following exposure to aluminium hydroxide-containing vaccines. The corresponding histological findings include aluminium-containing macrophages infiltrating muscle tissue at the injection site. The hypothesis is that the long-lasting granuloma triggers the development of the systemic syndrome.

Aluminium acts not only as an adjuvant, stimulating the immune system either to fend off infections or to tolerate antigens, it also acts as a sensitiser causing contact allergy and allergic contact dermatitis. In general, metal allergies are very common and aluminium is considered to be a weak allergen. A metal must be ionised to be able to act as a contact allergen, then it has to undergo haptensation to be immunogenic and to initiate an immune response. Once inside the skin, the metal ions must bind to proteins to become immunologically reactive. The most important routes of exposure and sensitisation to aluminium are through aluminium-containing vaccines. One Swedish study showed a statistically significant association between contact allergy to aluminium and persistent itching nodules in children treated with allergen-specific immunotherapy (ASIT). Nodules were overrepresented in patients with contact allergy to aluminium

Other routes of sensitisation reported in the literature are the prolonged use of aluminium-containing antiperspirants, topical medication, and tattooing of the skin with aluminium-containing pigments. Most of the patients experienced eczematous reactions whereas tattooing caused granulomas. Even though aluminium is used extensively in industry, only a low number of cases of occupational skin sensitisation to aluminium have been reported. Systemic allergic contact dermatitis in the form of flare-up reactions after re-exposure to aluminium has been documented: pruritic nodules at present and previous injection sites, eczema at the site of vaccination as well as at typically atopic localisations after vaccination with aluminium-containing vaccines and/or patch testing with aluminium, and also after use of aluminium-containing toothpaste

For aluminium starch octenylsuccinate and 7 other octenylsuccinate derivatives.

Oral doses of aluminium starch octenylsuccinate at concentrations up to 25% and oral doses of a related compound up to 30% produced no adverse effects in animal studies. Aluminium starch octenylsuccinate was not an ocular irritant. A commercial preparation of aluminium starch octenylsuccinate produced no abnormal skin reactions in guinea pigs and rabbits.

Aluminium starch octenylsuccinate was nontoxic to rats by inhalation. Clinical facial sting, chamber scarification, and closed patch studies indicated little irritation potential with aluminium starch octenylsuccinate (tested up to 3% in formulation). Aluminium starch octenylsuccinate, tested at up to 25% in formulation, was not a sensitizer in clinical RIPT. based on the available data, the CIR Expert panel concludes that aluminium starch octenylsuccinate is safe as used in cosmetic formulations provided that established limitations imposed on heavy metal concentrations are not exceeded.

Amended Safety Assessment of Aluminum Starch Octenylsuccinate As Used in Cosmetics: May 2018
Cosmetic Ingredient Review (CIR) Expert Panel

ETHYLENE GLYCOL PHENYL ETHER	Bacterial cell mutagen
POLYETHYLENE GLYCOL (2) STEARYL ETHER	Humans have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents and other cleaning products. Exposure to these chemicals can occur through swallowing, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that relatively high volumes would have to occur to produce any toxic response. No death due to poisoning with alcohol ethoxylates has ever been reported. Studies show that alcohol ethoxylates have low toxicity through swallowing and skin contact. Animal studies show these chemicals may produce gastrointestinal irritation, stomach ulcers, hair standing up, diarrhea and lethargy. Slight to severe irritation occurred when undiluted alcohol ethoxylates were applied to the skin and eyes of animals. These chemicals show no indication of genetic toxicity or potential to cause mutations and cancers. Toxicity is thought to be substantially lower than that of nonylphenol ethoxylates.

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	Some of the oxidation products of this group of substances may have sensitizing properties. As they cause less irritation, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their tendency to auto-oxidise also increases their irritation. Due to their irritating effect it is difficult to diagnose allergic contact dermatitis (ACD) by patch testing. Both laboratory and animal testing has shown that there is no evidence for alcohol ethoxylates (AEs) causing genetic damage, mutations or cancer. No adverse reproductive or developmental effects were observed. Tri-ethylene glycol ethers undergo enzymatic oxidation to toxic alkoxy acids. They may irritate the skin and the eyes. At high oral doses, they may cause depressed reflexes, flaccid muscle tone, breathing difficulty and coma. Death may result in experimental animal. However, repeated exposure may cause dose dependent damage to the kidneys as well as reproductive and developmental defects.
PROPYLENE GLYCOL	The acute oral toxicity of propylene glycol is very low; large amounts are needed to cause perceptible health damage in humans. Serious toxicity generally occurs only at blood concentrations over 1 g/L, which requires extremely high intake over a relatively short period of time; this is nearly impossible with consuming foods or supplements which contain 1g/kg of PG at most. Poisonings are usually due to injection through a vein or accidental swallowing of large amounts by children. The potential for long-term oral toxicity is also low. Prolonged contact with propylene glycol is essentially non-irritating to the skin. Undiluted propylene glycol is minimally irritating to the eye, and can produce a slight, temporary inflammation of the conjunctiva. Exposure to mists may cause irritation of both the eye and the upper airway. Inhalation of propylene glycol vapours may be irritating to some individuals. It is therefore recommended that propylene glycol not be used in applications where inhalation exposure or human eye contact with the spray mists of these materials is likely, such as fogs for theatrical productions or antifreeze solutions for emergency eye wash stations. Propylene glycol is metabolized in humans to pyruvic acid, acetic acid, lactic acid and propionaldehyde; the last of which is potentially hazardous. Propylene glycol show s no evidence of causing cancer or genetic toxicity. Research has suggested that individuals who cannot tolerate propylene glycol probably experience a special form of irritation, but they only rarely develop allergic contact dermatitis. Other investigators believe that the incidence of allergic contact dermatitis in people exposed to propylene glycol may be greater than 2% in patients with eczema. One study strongly suggests a connection between airborne concentrations of propylene glycol in houses and development of asthma and allergic reactions, such as inflammation of the nose and hives, in children. Another study suggested that the concentration of PGEs (propylene glycol and glycol ethers) in indoor air is linked to increased risk of developing numerous respiratory and immune disorders in children, including asthma, hay fever, eczema and allergies, with increased risk ranging from 50% to 180%. This concentration has been linked to use of water-based paints and water-based system cleansers. Patients with bladder inflammation and vulvodynia (chronic pain of the vulva) may be especially sensitive to propylene glycol. Women suffering with yeast infections may notice that some over the counter creams cause intense burning. Post-menopausal women who require the use of an oestrogen cream may notice that creams made with propylene glycol often cause extremely uncomfortable burning along the vulva and around the anus. Some electronic cigarette users who inhale propylene glycol vapour may experience dryness of the throat or shortness of breath. Adverse responses to administration of drugs which use propylene glycol as an incipient have been seen in a number of people especially at high doses. These include low blood pressure, slow heart rate, ECG abnormalities, heartbeat irregularities, lactic acidosis, breakdown of red cells and cardiac arrest.
HEXYL SALICYLATE	* Bedoukian Research Inc. The salicylates are well absorbed by mouth, and oral bioavailability is assumed to be total. In humans, absorption through skin is more limited. The salicylates are expected to be broken down to salicylic acid, mostly in the liver, and then conjugated with glycine or glucuronide and excreted in the urine. The expected metabolism of the salicylates do not present toxicological concerns. Animal testing shows that acute toxicity by skin contact is very low, while acute toxicity by mouth is moderate. Salicylates do not possess genetic toxicity, and generally do not have the potential to cause cancer. The reproductive and developmental toxicity data on methyl salicylate shows that high doses which are toxic to the mother may cause toxicity to the embryo and birth defects. At concentrations likely to be encountered through their use as fragrance ingredients, salicylates are considered to be non-irritating to the skin. The salicylates in general have no, or very limited, potential to sensitise skin. They do not possess light-mediated toxicity and do not cause light-mediated irritation or allergies.
3-iodo-2-propynyl butyl carbamate	Carbamate pesticides are less dangerous than organophosphorus pesticides. It requires higher dose to produce toxicity or mortality. However, its toxicity via skin contact is low to moderate. Its rapid metabolism causes acute toxic effect but subsequent rapid recovery. It may cause skin and eye irritation, increased pigmentation, sperm abnormalities, sensitization, cancer, mutations and genetic and foetal defects. It is easily absorbed through the skin, mucous membranes, airway and digestive tract. It can cause changes to the function of the blood-making system and the liver and kidney, at higher concentrations it can cause degeneration of the liver, kidney and testes. Its metabolites are detectable in urine. For 3-iodo-2-propynyl butyl carbamate (IPBC): Acute toxicity studies with IPBC show low toxicity except severe eye irritation. Animal testing showed that extended exposure may cause decreased weight gain and increased red cell and eosinophil counts. One study showed the possibility of increased breast cancer on extended contact. IPBC may cause defects in bone development at very high levels. It does not reduce fertility, but it does cause reduced body weight in infants. While it is toxic to the cell at high doses, it does not seem to cause mutations or genetic damage.
2-TERT-BUTYLCYCLOHEXYL ACETATE	There are no safety concerns regarding cyclic acetates under the present declared levels of use, for the reasons outlined below. Cyclic acetates have low acute toxicity. Cyclic acetates and cyclic alcohols also have low whole-body toxicity, after repeated application to skin. At concentrations encountered in current use, minimal, if any, skin irritation occurs. These substances have little or no sensitizing potential. Available data does not indicate that these substances cause genetic toxicity or mutations, so they are unlikely to cause cancer. They have a very wide safety margin.
BENZYL ACETATE	Neoplastic by RTECS criteria This is a member or analogue of a group of benzyl derivatives generally regarded as safe (GRAS), based partly on their self-limiting properties as flavouring substances in food. In humans and other animals, they are rapidly absorbed, broken down and excreted, with a wide safety margin. They also lack significant potential to cause genetic toxicity and mutations. The intake of benzyl derivatives as natural components of traditional foods is actually higher than the intake as intentionally added flavouring substances.
GAMMA-UNDECALACTONE	Gamma-butyrolactone may cause thymus atrophy, brain damage, severe weakness and low body weight in rats. It causes no foetal development defects but may decrease testicular weight in the male rat. There is insufficient evidence from animal testing to show that gamma-butyrolactone has cancer-causing effects. This is a member or analogue of a group of lactones generally considered as safe (GRAS). Aliphatic lactones occur naturally at high concentrations (up to 100 parts per million) in food having a high fat content such as meat, cheese, milk and coconuts.
GALAXOLIDE	Changes in liver weight, maternal effects, foetotoxicity reported. There is increasing evidence emerging that some nitromusks and polycyclic musks, including those commonly used in perfumes, may be capable (either as parent compounds or as metabolites) of interfering with hormone communication systems in fish, amphibians and mammals, and may exacerbate the effects of exposure to other toxic chemicals.
ALLYL HEPTANOATE	Oral (mouse): 630 mg/kg Skin (rabbit): 500 mg/24h - mod Somnolence, liver changes recorded.
D-LIMONENE	Tumorigenic by RTECS criteria Monomethyltin chloride, thioglycolate esters, and tall oil ester reaction product: Monomethyltin trichloride (MMTC, CAS RN: 993-16-8), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA), CAS RN: 57583-34-3), monomethyltin tris[isooctylmercaptoacetate (MMT(IOTG), CAS RN: 54849-38-6) and methyltin reverse ester tallate reaction product (TERP, CAS RNs: 201687-58-3, 201687-57-2, 68442-12-6, 151436-98-5) are considered one category of compounds for mammalian studies via the oral route. The justification for this category is based on structural similarities and the demonstrated rapid conversion of all of the esters to the MMTC when placed in simulated mammalian gastric contents [0.07M HCl] under physiological conditions. For the MMT(EHTG) >90% conversion to MMTC occurred within 0.5 hours. For TERP, 68% of the monomethyltin portion of the compound was converted to MMTC within 1 hour. Thus, MMTC is the appropriate surrogate for mammalian toxicology studies via the oral route.

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TERP is a reaction product of MMTC and dimethyltin dichloride (DMTC), Na₂S, and tall oil fatty acid [a mixture of carboxylic acids, predominantly C-18]. The reaction product is a mixture of carboxylic esters and includes short oligomers of mono/dimethyltins bridged by sulfide groups. Although the tall oil component of TERP is not structurally similar to EHTG, TERP's conversion to MMTC justifies its inclusion. While the contribution of the various ligands to the overall toxicity may vary, the contribution is expected to be small relative to that of the MMTC. Further, the EHTG ligand from MMT(EHTG) is likely to be more toxic than the oleic or linoleic acid from TERP so inclusion of TERP in the category is a rather conservative approach. The other possible degradate of tall oil and EHTG is 2-mercaptoethanol (2-ME), and it is common to both ligands.

Data for MMT(EHTG) and MMT(IOTG) are used interchangeably because they are isomers differing only slightly in the structure of the C-8 alcohol of the mercaptoester ligand. In addition, the breakdown products of MMT(EHTG) and MMT(IOTG) are the thioglycolate esters (EHTG and IOTG), which have the common degradates, thioglycolic acid and C-8 alcohols (either 2-ethylhexanol or isooctanol). EHTG and IOTG also have similar physicochemical and toxicological properties.

The chemistry of the alkyl organotin has been well studied. For organotin, like MMT(EHTG), the alkyl groups are strongly bound to tin and remain bound to tin under most reaction conditions. However, other ligands, such as carboxylates or sulfur based ligands (EHTG), are more labile and are readily replaced under mild reaction conditions. To assess the reactivity of MMT(EHTG) under physiological conditions simulating the mammalian stomach, an in-vitro hydrolysis test was performed. This in vitro test provides chemical information that strongly suggests both the probable in vivo metabolic pathway and the toxicokinetics of the MMT(EHTG) substance. This result verifies that under physiological conditions MMT(EHTG) is rapidly and essentially completely converted to the corresponding monomethyltin chloride, MMTC.

Acute toxicity:

The majority of toxicology studies were conducted with commercial mixtures having high monoalkyltin to dialkyltin ratios.

Gastric hydrolysis studies were conducted with TERP and MMT(EHTG) in which simulated gastric fluid [0.07M HCl under physiological conditions] converted these substances to methyltin chloride and the respective organic acids. Based on data for DMTC and DMT esters the dermal penetration of MMTC and its esters is expected to be low.

Oral:

Acute oral LD50 values for MMTC, MMT(EHTG), MMT(IOTG), and TERP indicated low to moderate toxicity; the most reliable data place the LD50s in the range of 1000 mg/kg.

The acute oral LD50 of MMT(2-EHMA) was 880 mg/kg in rats. Clinical observations included depression, comatose, piloerection, eye squinting, hunched posture, laboured breathing, ataxia, faecal/urine stains, and masticatory movement. No gross pathological changes were reported in surviving animals.

Dermal

Acute dermal LD50 values were =1000 mg/kg bw, and inhalation LC50 was >200 mg/L. MMTC was corrosive to skin and assumed corrosive to eyes.

The acute dermal LD50 of MMT(2-EHMA) in rabbits was 1000 (460 to 2020) mg/kg for females and 2150 (1000 to 4620) mg/kg for males.

There were no deaths at 215 and 464 mg/kg, 0/2 males and 1/2 females died at 1000 mg/kg and 1/2 males and 2/2 females died at 2150 mg/kg. All animals died at 4640 and 10 000 mg/kg. A variety of clinical abnormalities were observed and disappeared in surviving animals by the end of the exposure period. Clinical signs included death, uncoordinated movements, shaking, and hypersensitivity to external stimuli. Gross necropsy results for animals that died during the study included irritated intestines; blanched stomach; reddened lungs; pale or congested kidneys; and oral, ocular and/or nasal discharges

Inhalation:

The acute inhalation LC50 of MMT(2-EHMA) was 240 mg/L.

The study reported an acute inhalation LC50 of 240 (212 to 271) mg/L in a 1-hr aerosol exposure to male and female rats. The mortality rate was 2/10, 6/10, 9/10 and 10/10 animals at dose levels of 200, 250, 300 and 250 mg/L/hr, respectively. Gross findings included blood in lungs, dark spleen, pale kidneys, fluid in the chest cavity, and heart failure. The slope of the dose-response curve was 1.22 (1.04 to 1.43).

Irritation:

MMT(IOTG)/(EHTG) are irritating to skin, but not to eyes.

Sensitisation:

No data on sensitization are available on MMT(EHTG)/(IOTG), but the hydrolysis products EHTG or IOTG are sensitizers. No primary irritation data were available for TERP, but it was a sensitizer in the mouse Local Lymph Node Assay.

Topical application with 5, 25 and 50 % v/v MMT(2-EHMA) elicited a stimulation index (SI) of 2.13, 7.25 and 9.05, respectively in a local lymph node assay (OECD 429), thus the material is a sensitizer.

Repeat dose toxicity:

There are no repeated-dose studies for the category members via the dermal or inhalation routes.

In a 90-day repeated dose oral study of MMTC, treatment-related changes were limited to the high dose group (750 ppm in diet; 50 mg/kg bw/d with some gender-related variation). Organ weight changes (adrenal, kidney, thymus, spleen, brain, epididymides), haematology, clinical chemistry, and urinalysis changes were noted, but histopathology only confirmed effects in the thymus and brain. The critical toxic effects were neurotoxicity and thymic atrophy. Both sexes had decreased cortex/medulla ratios in the thymus. In the brain there was loss of perikarya of neuronal cells in the pyramidal layer of the Hippocampus CA1/2 in both sexes, and in males there was loss of perikarya in the piriform cortex. The NOAEL was 150 ppm (10 mg/kg bw/d). Another 90-day dietary study using MMTC showed increased relative kidney weights and slight to moderate epithelial hyperplasia of the bladder in females at the lowest dose (NOAEL <20 ppm in diet [$<1\text{--}3.6\text{ mg/kg bw/d}$]) and additional effects including increased relative thymus weights in females and urinalysis results in both sexes at higher doses.

A 90-day dietary study with dose levels of 30, 100, 300, and 1000 ppm TERP in the diet resulted in slightly decreased food intake, body and organ weight changes, and decreased specific gravity of the urine at the highest dose. The NOAEL was 300 ppm in diet (equivalent to 15 mg/kg bw/d). A 28-day gavage study using TERP showed changes in clinical chemistry and slight differences in haematology at 150 mg/kg bw/d and higher. The NOAEL was 50 mg/kg bw/d.

The effects of MMT(IOTG) were evaluated in a 90-day dietary study using doses of 100, 500, and 1500 ppm (decreased from 2500 ppm) in the diet. Based on clinical chemistry effects at 500 ppm and other effects at higher doses, the NOAEL was 100 ppm in diet (approximately 6-21 mg/kg bw/d).

Neurotoxicity:

In a guideline 90-day subchronic dietary study conducted in Wistar rats, effects occurred at the high dose of 750 ppm MMT(2-EHMA), (equivalent to 49.7 mg/kg bw/day in males and 53.6 mg/kg bw/day in females), which consisted of changes in neurobehavioral parameters and associated brain histopathology. The NOAEL was the next lower dose of 150 ppm (equivalent to 9.8 mg/kg bw/day in males and 10.2 mg/kg bw/day in females).

Immunotoxicity:

Immune function was assessed in male Sprague-Dawley rats exposed to the mixture of organotin used in PVC pipe production.

Adult male rats were given drinking water for 28 d containing a mixture of dibutyltin dichloride (DBTC), dimethyltin dichloride (DMTC), monobutyltin trichloride (MBT), and monomethyltin trichloride (MMT) in a 2:2:1:1 ratio, respectively, at 3 different concentrations (5:5:2.5:2.5, 10:10:5:5, or 20:20:10:10 mg organotin/L). Rats were also exposed to MMT alone (20 or 40 mg MMT/L) or plain water as a control. Delayed-type hypersensitivity, antibody synthesis, and natural killer cell cytotoxicity were evaluated in separate endpoint groups immediately after exposure ended.

The evaluated immune functions were not affected by the mixture or by MMT alone. The data suggest that immunotoxicity is unlikely to result from the concentration of organotin present in drinking water delivered via PVC pipes, as the concentrations used were several orders of magnitude higher than those expected to leach from PVC pipes

Genotoxicity:

In a guideline 90-day subchronic dietary study in rats, with MMT(2-EHMA), based on the changes in neurobehavioral parameters and associated brain histopathology that occurred at the high dose of 750 ppm (equivalent to 49.7 mg/kg bw/day in males and 53.6 mg/kg bw/day in females), as well as changes in haematology, clinical chemistry, urinalysis, organ weights, and pathology of the thymus at the same dose, the NOAEL was the next lower dose of 150 ppm (equivalent to 9.8 mg/kg bw/day in males and 10.2 mg/kg bw/day in females). The monomethyltin compounds as a class are not mutagenic in the Ames test. TERP was positive in a human lymphocyte assay. MMTC was equivocal for induction of micronucleated polychromatic erythrocytes (MPEs) in an in vivo rat micronucleus test (OECD 474). In this study a statistically significant increase in MPE was observed only at 24 h and not at 48 h after treatment and there was no dose-response. Based on these observations the overall conclusion is that MMTC does not have genotoxic potential.

From the results obtained in a micronucleus test with MMT(2-EHMA), it was demonstrated that the substance was weakly genotoxic to bone marrow cells of rats and that the substance has the potential to induce damage to the mitotic spindle apparatus of the bone marrow target cells.

Carcinogenicity:

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	In a limited carcinogenicity study, MMT(EHTG) produced no compound-related macroscopic or microscopic changes in rats fed 100 ppm in the diet for two years. Toxicity to reproduction: In the reproductive satellite portion of the 90-day study using MMT (with dose levels of 30, 150, and 750 ppm in the diet), post-implantation loss, decreased litter size and increased neonatal mortality occurred at 750 ppm (26-46 mg/kg bw/d for females). Maternal gestational body weights were transiently suppressed and other maternal toxicity was inferred from the repeated dose results at this dose. There were no malformations observed at any dose. The NOAEL for maternal toxicity, and reproductive, and fetotoxic effects was 150 ppm in the diet (6-12 mg/kg bw/d). SIDS Initial Assessment Profile (SIAM 23 2006) ECHA Registration Dossier for MMT(2-EHMA) (ethylhexyl 10-ethyl-4-[[2-[(2-ethylhexyl)oxy]-2-oxoethyl]thio]-4-methyl-7-oxo-8-oxa-3,5-dithia-4-stannatetradecanoate) d-Limonene is readily absorbed by inhalation and swallowing. Absorption through the skin is reported to be lower than by inhalation. It is rapidly distributed to different tissues in the body, readily metabolized and eliminated, primarily through the urine. Limonene shows low acute toxicity by all three routes in animals. Limonene is a skin irritant in both experimental animals and humans. Limited data is available on the potential to cause eye and airway irritation. Autooxidised products of d-limonene have the potential to sensitise the skin. Limited data is available on the potential to cause respiratory sensitization in humans. Limonene will automatically oxidize in the presence of light in air, forming a variety of oxygenated monocyclic terpenes. When contact with these oxidation products occurs, the risk of skin sensitization is high. Limonene does not cause genetic toxicity of birth defects, and it is not toxic to the reproductive system.
CARVONE	- for (+/-)-carvone for (R)-(-)-carvone [RTECS No.: OS 8650000] Carvone substances have been reported to occur naturally in foods, including fruits, spices, and berries. Currently there are no safety concerns for any of the carvones based on current levels of intake. Following short term intake, increases in serum cholesterol and lipid concentrations have been reported in rats, as well as decreases in food consumption and body weight. Repeated intake may also cause shrinking of the testes. They appear to be protective against liver, lung, stomach and intestine tumours in rats. A member or analogue of a group of aliphatic and alicyclic terpenoid tertiary alcohols and structurally related substances generally regarded as safe. Most alicyclic substances used as flavour ingredients are mono- and bicyclic terpenes which occur naturally in a wide variety of foods. With the exception of pulegone, alicyclic substances show very low oral acute toxicity. In most subchronic studies performed on animals, no adverse effects were observed at any dose level.
GERANIOL	Allergic reactions involving the respiratory tract are usually due to interactions between IgE antibodies and allergens and occur rapidly. Allergic potential of the allergen and period of exposure often determine the severity of symptoms. Some people may be genetically more prone than others, and exposure to other irritants may aggravate symptoms. Allergy causing activity is due to interactions with proteins. Attention should be paid to atopic diathesis, characterised by increased susceptibility to nasal inflammation, asthma and eczema. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure. Citronellol, geraniol, nerol, and geranyl acetate are currently generally regarded as safe by the US FDA for their intended use as flavouring substances. They are ubiquitous in the plant kingdom. Terpenoid alcohol, formed in the gastrointestinal tract, as a result of hydrolysis, is rapidly absorbed, metabolised and excreted via the urine. It has no repeat dose effect, no genetic and cancer causing effect but may harm the unborn child of a pregnant woman. Geraniol does have sensitising properties, but the response it exhibits tends to be weak and variable. Animal testing revealed an oral semi-lethal dose of more than 3.6 g/kg in rats and an acute semi-lethal dose via skin absorption of over 5.0 g/kg.
BENZYL C8-18 ALKYLDIMETHYLAMMONIUM CHLORIDE	General depressed activity, impaired liver function tests, increased urine volume, changes in bone marrow, chronic pulmonary oedema, gastrointestinal changes recorded. For similar compound benzyl C12-18 alkyl dimethyl ammonium chloride CAS RN 68391-01-5: For acid mists, aerosols, vapours Test results suggest that eukaryotic cells are susceptible to genetic damage when the pH falls to about 6.5. Cells from the respiratory tract have not been examined in this respect. Mucous secretion may protect the cells of the airway from direct exposure to inhaled acidic mists (which also protects the stomach lining from the hydrochloric acid secreted there). The material may produce respiratory tract irritation, and result in damage to the lung including reduced lung function. Alkyldimethylammonium chlorides are in the list of dangerous substances of council directive, classified as "harmful in contact with skin and on ingestion", and "corrosive and very toxic to aquatic organisms". It can cause dose dependent skin and eye irritation with possible deterioration of vision, possible sensitisation in those with pre-existing eczema. It does not cause cancer, genetic defect, foetal or developmental abnormality.
LINALOOL	Inhalational exposure of mice and man to linalool caused slight sedative effects but a dose dependent response characteristic could not be determined. It may irritate the digestive tract, skin, nose and the eyes but is not considered to be a sensitiser. It is equally shown to cause kidneys and liver damage but no genetic or reproductive defect was observed. Opinion holds that there are no safety concerns for linalool and the linalyl esters, as fragrance ingredients, under the present declared levels of use and exposure for the following reasons: - Linalool and the linalyl esters have a low order of acute toxicity. - No significant toxicity was observed in subchronic tests; it is concluded that these materials have dermal and oral NOAELs of 50 mg/kg/day or greater. - Based on a critical review of all available mutagenicity and genotoxicity studies, it has been determined that these materials are negative in short-term tests and therefore would have no significant potential to produce genotoxic effects. - The metabolic fate of linalool and the linalyl esters is either known or assumed from analogies with structurally related substances that indicate no production of toxic or persistent metabolites and the structural analogies indicate no concern. - Human dermatological studies show that these materials are not irritating, phototoxic or sensitizing. - These materials are used at low levels of exposure relative to doses that elicit adverse effects. The estimate for maximum systemic exposure by humans using cosmetic products is 0.3 mg/kg/ day for linalool and linalyl acetate and 0.1 mg/ kg/day or lower for the other linalyl esters. Using the NOAELs (50 mg/kg/day or greater) and the maximum exposure estimates and assuming 100% absorption, a margin of safety for the exposure of humans to linalool and the linalyl esters may conservatively be calculated as 167 times the maximum daily exposure for linalool and linalyl acetate (50 mg/kg/day 0.3 mg/kg/day for linalool or linalyl acetate=167) and 500 times the maximum daily exposure for the other individual linalyl esters (50 mg/kg/day / 0.1 mg/kg/day for the other individual linalyl esters=500). In general, linalool esters are hydrolyzed to their corresponding alcohol (linalool) and carboxylic acid. Hydrolysis is catalyzed by carboxylesterases or esterases. Tertiary alcohols such as linalool are metabolized primarily through conjugation with glucuronic acid and are excreted in the urine and to a lesser extent faeces. Alkyl or alkenyl substituents may undergo oxidation to form polar metabolites that may also be excreted free or in the conjugated form. Oxidation is mediated by cytochrome P-450 dependant mono-oxygenases. The carboxylic acids formed by hydrolysis of the linalyl esters included in this summary are all known to be easily and rapidly metabolized. The linear saturated carboxylic acids are metabolized normally as fatty acids that undergo beta-oxidation. The branched-chain carboxylic acids from linalyl isovalerate and isobutyrate are similarly oxidized, but the end product is acetone. The carboxylic acids from linalyl benzoate and phenylacetate are conjugated and excreted. The cinnamic acid from linalyl cinnamate is conjugated and excreted, or metabolized to benzoic acid. No sensitization was observed with linalool in guinea pig sensitization studies at concentrations up to 20%. With linalyl acetate at a concentration of 10%, weak to moderate sensitization effects were observed in guinea pig sensitization studies. Linalyl acetate was non-sensitizing when tested at 5% in these same guinea pig sensitization studies. No sensitization reactions were observed with linalyl isobutyrate and linalyl propionate (data were not available for the other linalyl esters) when tested at 8% in open epicutaneous tests in guinea pigs The Research Institute for Fragrance Materials (RIFM) Expert Panel
2,6-DI-TERT-BUTYL-4-METHYLPHENOL	* Degussa SDS Effects such as behavioral changes, reduction in body weight gain, and decrement in body weight have been observed after long-term administration of BHT to mice and rats. Toxic effects may be attributed more to BHT metabolites than to their parent compound, only a few studies have focused on their carcinogenicity and toxicity, and not only on that of BHT. The metabolite BHT-QM (syn: 2,6-di-tert-butyl-1,4-methylene-2,5-cyclohexadien-1-one, CAS RN: 2607-52-5) is a very reactive compound which is considered to play a significant

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role in hepatotoxicity, pneumotoxicity, and skin tumor promotion in mice. In addition, it was reported that another quinone derivative, BHT-OH(t)QM (syn 2-tert-butyl-6-(2-hydroxy-tert-butyl-4-methylene-2,5-cyclohexadien-1-one, CAS RN: 124755-19-7), is chemically more reactive than BHT-QM, and it has been recognized as the principal metabolite responsible for lung tumor promotion activity of BHT in mice. BHT has been reported to exert prooxidant effects under certain conditions. Thus, when BHT was added in excess to a wheat seedling medium in aerobic conditions, an enhancement of the generation rate of superoxide anion was observed. This is a reactive particle that may damage cellular structures at high concentrations. In addition, an increase in hepatic microsomal lipid peroxidation was observed in rats fed with diets containing 0.2% of BHT for 30 days. Due to this ability of BHT to exert prooxidant effects at high concentrations, it has been used to induce experimental models of oxidative stress in several animals and fungi in order to study the protective effects of other compounds. Quinone methide derivatives form adducts with several proteins, including enzymes that protect cells from oxidative stress; this prooxidant state can also lead to cell oxidative damage. It must be noted that relationships between chronic oxidative stress and tumor promotion are well known. Some authors have reported that at high aeration rate, BHT can react with molecular oxygen rather than with the reactive oxygen species present, yielding BHT-phenoxyl radical and superoxide anion. In addition, the phenolic radical itself may undergo redox recycling which can be a critical factor depending on the reductant involved. However, it has to be noted that BHT-phenoxyl radical has been reported to be relatively stable. Furthermore, the potential reactivity of BHT-derived metabolites should be taken into account; some studies reported that not only BHT but also its metabolites, such as BHT-Q and BHT-QM, can act as prooxidant. As BHT undergoes several reactions during biotransformation, a large number of intermediate metabolites have been identified. However, their nature and concentration depend on the environmental conditions and on the animal species. Although the changes undergone by BHT during *in vivo* digestion processes have not been studied, after submission of a fluid deep-frying fat containing BHT and BHT-QM to an *in vitro* gastrointestinal digestion model, both these were detected in the digested samples. These results indicate that BHT and its toxic metabolite could remain bioaccessible for intestinal absorption. Studies concerning BHT metabolism have shown that, unlike other synthetic antioxidants, BHT is a potent inducer of the microsomal monooxygenase system and its major route of degradation is oxidation catalyzed by cytochrome P450. Studies have reported potential toxicity derived from the ingestion or administration of BHT. As for acute oral toxicity, although this is considered low in animals, it must be noted that 2 clinical cases were reported in patients who suffered acute neurotoxicity and gastritis after ingesting a high dose of BHT (4 and 80 g without medical prescription) to cure recurrent genital herpes. Regarding short-term subchronic toxicity studies, it has been reported that BHT causes dose-related increase in the incidence and severity of toxic nephrosis in mice, nephrotoxicity and pneumotoxicity in rats, and in chicken a marked congestion of the liver and kidney, as well as diffuse enlargement of the liver with rounded borders and rupture with hemorrhaging. It has to be noted that the EFSA Panel (2012) pointed out certain inconsistencies in the findings obtained from the short-term and subchronic toxicity studies. Several genotoxicity studies on BHT concluded that BHT does not represent a genotoxic risk, because most of the studies carried out to that date had shown BHT was not able to induce mutations or to damage deoxyribonucleic acid (DNA). Nevertheless, it must be mentioned that other studies reported contrary results. The effect of BHT and 7 of its metabolites on *in vitro* DNA cleavage was studied and the metabolites BHT-Q (syn: 2,6-di-tert-butyl-2,5-cyclohexadiene-1,4-dione, CAS RN: 719-22-2), BHT-CHO (syn: 3,5-di-tert-butyl-4-hydroxybenzaldehyde, CAS RN: 1620-98-0 and BHT-OOH (syn: 2,6-di-tert-butyl-4-methyl-4-hydroperoxy-2,5-cyclohexadien-1-one, CAS RN: 6485-57-0) were able to cleave DNA. The Panel on Food Additives and Nutrient Sources Added to Food of the European Food Safety Authority (EFSA) recognized that these positive genotoxicity results may be due to the prooxidative chemistry of BHT, which gives rise to reactive metabolites. Some studies addressed the carcinogenicity and chronic toxicity of BHT and its metabolites in rodents with contradictory results. Thus, mice-fed dietary BHT for a year developed marked hyperplasia of the hepatic bile ducts with an associated subacute cholangitis. Moreover, after 104 wk of administration of BHT, the formation of hepatocellular tumors in male mice was observed. After 10 months of feeding mice with a diet containing different amounts of BHT, an increased incidence of liver tumors in male, but not female, animals was also reported. However, in a similar study no evidence of the carcinogenicity of BHT administered to mice was observed. Studies performed in rats also reported dose-related increases in hepatocellular adenomas and carcinomas; nevertheless, other studies carried out with rats showed no consistent carcinogenic effects. Several studies have demonstrated the potential of BHT to act either as a tumor promoter or as a tumor suppressor, modulating the carcinogenicity of some well-known carcinogens. Barbara Nieva-Echevarria et al: Comprehensive reviews in Food Science and Food Safety, Vol 14, Dec 2014 <https://onlinelibrary.wiley.com/doi/10.1111/1541-4337.12121/pdf> for bridged alkyl phenols:

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

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

Reproductive toxicity: Evaluation of effects on reproduction for the bridged alkyl phenols is supplemented by histopathological data on male and female reproductive organs in repeated dose studies. The data on the effects of bridged alkyl phenols on reproduction and reproductive organs span the range of structures and molecular weights. While not all of the data for reproductive effects are from reproduction studies, microscopic evaluations of reproductive organs along with other short-term tests for reproductive effects provide adequate data to evaluate the effects of these bridged alkyl phenols on reproduction. It can be concluded that reproductive toxicity is low. Typically a two-year chronic feeding study provides data for 4,4'-thiobis-6-(t-butyl-m-cresol) (96-69-5). No adverse effects were noted on reproductive organs

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

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

The Bridged Alkyl Phenols Category consists of a group of chemicals in which two molecules of mono or di-substituted alkyl (C1, C4, and/or C9) phenols are "bridged" or linked by a single atom (carbon or sulfur). The carbon atom linking the alkyl phenol groups contains hydrogen, propyl, or methyl substitutions. CAS No. 128-37-0 (BHT) is included in this category for data purposes because it is an alkyl phenol with a single carbon group such as the ones that link the phenol groups

ferroptosis inhibitors are currently being treated systemically rather than specifically, which may have multiple side effects. For example, Desferoxamin (DFO), an iron chelating agent, is known to have a short half-life, need long-term subcutaneous infusions, and provoke ototoxicity and neurotoxicity. Deferasirox (DFX), an iron chelator, is associated with gastrointestinal and renal toxicity.

Data show that acute toxicity following oral and topical use of hindered phenols is low. They are not proven to cause mutations. However, long term use may affect the liver, thyroid, kidney and lymph nodes. Liver tumours have been reported.

COUMARIN

umarin is moderately toxic to the liver and kidneys of rodents, with a median lethal dose (LD50) of 293 mg/kg in the rat, a low toxicity compared to related compounds. Coumarin is hepatotoxic in rats, but less so in mice. Rodents metabolize it mostly to 3,4-coumarin epoxide, a toxic, unstable compound that on further differential metabolism may cause liver cancer in rats and lung tumors in mice. Humans metabolize it mainly to 7-hydroxycoumarin, a compound of lower toxicity, and no adverse effect has been directly measured in humans. The German Federal Institute for Risk Assessment has established a tolerable daily intake (TDI) of 0.1 mg coumarin per kg body weight, but also advises that higher intake for a short time is not dangerous. The Occupational Safety and Health Administration (OSHA) of the United States does not classify coumarin as a carcinogen for humans. European health agencies have warned against consuming high amounts of cassia bark, one of the four main species of cinnamon, because of its coumarin content. According to the German Federal Institute for Risk Assessment (BfR), 1 kg of (cassia) cinnamon powder contains about 2.1 to 4.4 g of coumarin. Powdered cassia cinnamon weighs 0.56 g/cm³, [33] so a kilogram of cassia cinnamon powder equals 362.29 teaspoons. One teaspoon of cassia cinnamon powder therefore contains 5.8 to 12.1 mg of coumarin, which may be above the tolerable daily intake value for smaller individuals. [32] However, the BfR only cautions against high daily intake of foods containing coumarin. Its report specifically states that Ceylon cinnamon (Cinnamomum verum) contains "hardly any" coumarin. The European Regulation (EC) No 1334/2008 describes the following maximum limits for coumarin: 50 mg/kg in traditional and/or seasonal bakery wares containing a reference to cinnamon in the labeling, 20 mg/kg in breakfast cereals including muesli, 15 mg/kg in fine bakery ware, with the exception of traditional and/or seasonal bakery ware containing a reference to cinnamon in the labeling, and 5 mg/kg in desserts. An investigation from the Danish Veterinary and Food Administration in 2013 shows that bakery goods characterized as fine bakery ware exceeds the European limit (15 mg/kg) in almost 50% of the cases. [34] The paper also mentions tea as an additional important contributor to the overall coumarin intake, especially for children with a sweet habit. Coumarin was banned as a food

	additive in the United States in 1954, largely because of the hepatotoxicity results in rodent] Coumarin is currently listed by the Food and Drug Administration (FDA) of the United States among "Substances Generally Prohibited From Direct Addition or Use as Human Food," according to 21 CFR 189.130,[36][37] but some natural additives containing coumarin, such as the flavorant sweet woodruff are allowed "in alcoholic beverages only" under 21 CFR 172.510. In Europe, popular examples of such beverages are Maiwein, white wine with woodruff, and Zubrówka, vodka flavoured with bison grass. umarin is subject to restrictions on its use in perfumery,[39] as some people may become sensitized to it, however the evidence that coumarin can cause an allergic reaction in humans is disputed nor neurological dysfunction was found in children exposed to the anticoagulants acenocoumarol or phenprocoumon during pregnancy. A group of 306 children were tested at ages 7–15 years to determine subtle neurological effects from anticoagulant exposure. Results showed a dose–response relationship between anticoagulant exposure and minor neurological dysfunction. Overall, a 1.9 (90%) increase in minor neurological dysfunction was observed for children exposed to these anticoagulants, which are collectively referred to as "coumarins." In conclusion, researchers stated, "The results suggest that coumarins have an influence on the development of the brain which can lead to mild neurologic dysfunctions in children of school age. Alcoholic beverages sold in the European Union are limited to a maximum of 10 mg/L coumarin by law. Cinnamon flavor is generally cassia bark steam-distilled to concentrate the cinnamaldehyde, for example, to about 93%. Clear cinnamon-flavored alcoholic beverages generally test negative for coumarin, but if whole cassia bark is used to make mulled wine, then coumarin shows up at significant levels
VINYLPYRROLIDONE HOMOPOLYMER & BENZYL ACETATE & D-LIMONENE & 2,6-DI-TERT-BUTYL-4- METHYLPHENOL & COUMARIN	The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
HYDROXYETHYLCELLULOSE & CETOSTEARYL ALCOHOL & BEESWAX & ALUMINIUM STARCH OCTENYLSUCCINATE & DIMETHYLAMINOETHYL METHACRYLATE/ PVP/ N- VINYLCAPROLACTAM & 2,4- DIMETHYL-3-CYCLOHEXENE- 1-CARBOXALDEHYDE	No significant acute toxicological data identified in literature search.
CETOSTEARYL ALCOHOL & SODIUM HYDROXIDE & CETYL ALCOHOL & STEARYL ALCOHOL & HEXYL SALICYLATE & 2- TERT-BUTYLCYCLOHEXYL ACETATE & BENZYL ACETATE & GAMMA- UNDECALACTONE & GERANIOL & BENZYL C8-18 ALKYLDIMETHYLAMMONIUM CHLORIDE & 2,6-DI-TERT- BUTYL-4-METHYLPHENOL & COUMARIN	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.
CETOSTEARYL ALCOHOL & CETYL ALCOHOL & STEARYL ALCOHOL & BENZYL C8-18 ALKYLDIMETHYLAMMONIUM CHLORIDE	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
CETOSTEARYL ALCOHOL & SODIUM POLYACRYLATE & SODIUM HYDROXIDE & CETYL ALCOHOL & GAMMA- UNDECALACTONE & GERANIOL & BENZYL C8-18 ALKYLDIMETHYLAMMONIUM CHLORIDE & LINALOOL	The material may cause severe skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin. Repeated exposures may produce severe ulceration.
CETOSTEARYL ALCOHOL & CETYL ALCOHOL & LINALOOL	Alkyl alcohols of chain length C6-13 are absorbed from skin, when inhaled or swallowed but show evidence of little harm. They are broken down and rapidly excreted by the body.
ETHYLENE GLYCOL PHENYL ETHER & SODIUM HYDROXIDE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
ETHYLENE GLYCOL PHENYL ETHER & STEARYL ALCOHOL & PROPYLENE GLYCOL & BENZYL ACETATE & GALAXOLIDE & ALLYL HEPTANOATE & 2,6-DI-TERT- BUTYL-4-METHYLPHENOL	The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.
ETHYLENE GLYCOL PHENYL ETHER & BENZYL ACETATE & ALPHA,ALPHA- DIMETHYLPHENETHYL BUTYRATE	The aryl alkyl alcohol (AAA) fragrance ingredients have diverse chemical structures, with similar metabolic and toxicity profiles. The AAA fragrances demonstrate low acute and subchronic toxicity by skin contact and swallowing. At concentrations likely to be encountered by consumers, AAA fragrance ingredients are non-irritating to the skin. The potential for eye irritation is minimal. With the exception of benzyl alcohol, phenethyl and 2-phenoxyethyl AAA alcohols, testing in humans indicate that AAA fragrance ingredients generally have no or low sensitization potential. Available data indicate that the potential for photosensitization is low. Testing suggests that at current human exposure levels, this group of chemicals does not cause maternal or developmental toxicity. Animal testing shows no cancer-causing evidence, with little or no genetic toxicity. It has been concluded that these materials would not present a safety concern at current levels of use, as fragrance ingredients.
HEXYL SALICYLATE & BENZYL ACETATE	For certain benzyl derivatives: The members of this group are rapidly absorbed through the gastrointestinal tract, metabolised primarily in the liver, and excreted primarily in the urine either unchanged or as conjugates of benzoic acid derivatives. At high dose levels, gut micro-organisms may act to produce minor amounts of breakdown products. However, no adverse effects have been reported even at repeated high doses. Similarly, no effects were observed on reproduction, foetal development and tumour potential.
3-iodo-2-propynyl butyl carbamate & alpha,alpha-	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

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DIMETHYLPHENETHYL BUTYRATE & GALAXOLIDE & D-LIMONENE & CARVONE & 2,4-DIMETHYL-3-CYCLOHEXENE-1-CARBOXALDEHYDE & GERANIOL & LINALOOL & 2,6-DI-TERT-BUTYL-4-METHYLPHENOL & COUMARIN	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.
BENZYL ACETATE & ALPHA,ALPHA-DIMETHYLPHENETHYL BUTYRATE	Aryl alkyl alcohol simple acid ester derivatives (AAASAE) have a low level of acute toxicity. Repeat-dose toxicity tests did not show significant toxicity. Testing did not show any evidence of AAASAE to have potential to cause cancer, mutations or genetic toxicity. At expected exposure levels, there is no evidence that AAASAE causes adverse effects on reproduction or development. In general there are currently no safety concerns regarding AAASAE at current levels of use and exposure.
GAMMA-UNDECALACTONE & ALPHA,ALPHA-DIMETHYLPHENETHYL BUTYRATE & GALAXOLIDE & D-LIMONENE & CARVONE & 2,4-DIMETHYL-3-CYCLOHEXENE-1-CARBOXALDEHYDE & GERANIOL & LINALOOL & COUMARIN	Adverse reactions to fragrances in perfumes and fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, sensitivity to light, immediate contact reactions, and pigmented contact dermatitis. Airborne and connubial contact dermatitis occurs. Contact allergy is a lifelong condition, so symptoms may occur on re-exposure. Allergic contact dermatitis can be severe and widespread, with significant impairment of quality of life and potential consequences for fitness for work. If the perfume contains a sensitizing component, intolerance to perfumes by inhalation may occur. Symptoms may include general unwellness, coughing, phlegm, wheezing, chest tightness, headache, shortness of breath with exertion, acute respiratory illness, hayfever, asthma and other respiratory diseases. Perfumes can induce excess reactivity of the airway without producing allergy or airway obstruction. Breathing through a carbon filter mask had no protective effect. Occupational asthma caused 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. Prevention of contact sensitization to fragrances is an important objective of public health risk management. Hands: Contact sensitization may be the primary cause of hand eczema or a complication of irritant or atopic hand eczema. However hand eczema is a disease involving many factors, and the clinical significance of fragrance contact allergy in severe, chronic hand eczema may not be clear. Underarm: Skin inflammation of the armpits 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 skin specialist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy. Face: An important manifestation of fragrance allergy from the use of cosmetic products is eczema of the face. In men, after-shave products can cause eczema around the beard area and the adjacent part of the neck. Men using wet shaving as opposed to dry have been shown to have an increased risk of allergic to fragrances. Irritant reactions: Some individual fragrance ingredients, such as citral, are known to be irritant. Fragrances may cause a dose-related contact urticaria (hives) which is not allergic; cinnamal, cinnamic alcohol and Myroxylon pereirae are known to cause hives, but others, including menthol, vanillin and benzaldehyde have also been reported. Pigmentary anomalies: Type IV allergy is responsible for "pigmented cosmetic dermatitis", referring to increased pigmentation on the face and neck. Testing showed a number of fragrance ingredients were associated, including jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol and geranium oil. Light reactions: Musk ambrette produced a number of allergic reactions mediated by light and was later banned from use in Europe. Furocoumarins (psoralens) in some plant-derived fragrances have caused phototoxic reactions, with redness. There are now limits for the amount of furocoumarins in fragrances. 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 the nose / airway. 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. A significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients and hand eczema.
GAMMA-UNDECALACTONE & ALPHA,ALPHA-DIMETHYLPHENETHYL BUTYRATE & GALAXOLIDE & COUMARIN	Fragrance allergens act as haptens, low molecular weight chemicals that cause an immune response only when attached to a carrier protein. However, not all sensitizing fragrance chemicals are directly reactive, but require previous activation. A prehapten is a chemical that itself causes little or no sensitization, but 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 a prohaptens , or both. Prohaptens: Compounds that are bioactivated in the skin and thereby form haptens are referred to prohaptens. The possibility of a prohaptens being activated cannot be avoided by outside measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Various enzymes play roles in both activating and deactivating prohaptens. Skin-sensitizing prohaptens can be recognized and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or studies of sensitization. QSAR prediction: Prediction of sensitization activity of these substances is complex, especially for those substances that can act both as pre- and prohaptens.
ALPHA,ALPHA-DIMETHYLPHENETHYL BUTYRATE & LINALOOL	A member or analogue of a group of aliphatic and alicyclic terpenoid tertiary alcohols and structurally related substances generally regarded as safe. Animal testing suggests that the acute toxicity of tertiary alcohols and related esters is extremely low. Genetic toxicity: Tests on bacterial and animal cells showed no evidence of genetic toxicity or potential to cause mutations. For terpenoid tertiary alcohols and their related esters: These substances are metabolised in the liver and excreted primarily in the urine and faeces. A portion is also excreted unchanged. They have low short term toxicity when ingested or applied on the skin. However, repeated and long term use may cause dose dependent harm to both the foetus and mother.
D-LIMONENE & GERANIOL	Epoxidation of double bonds is a common bioactivation pathway for alkenes. The allylic epoxides formed were found to be sensitizing. Research has shown that conjugated dienes in or in conjunction with a six-membered ring are prohaptens, while related dienes containing isolated double bonds or an acrylic conjugated diene were weak or non-sensitising.
D-LIMONENE & CARVONE & 2,4-DIMETHYL-3-CYCLOHEXENE-1-CARBOXALDEHYDE & GERANIOL & LINALOOL	Fragrance allergens act as haptens, which are small molecules that cause an immune reaction only when attached to a carrier protein. However, not all sensitizing fragrance chemicals are directly reactive, but some require previous activation. A prehapten is a chemical that itself causes little or no sensitization, but it is transformed into a hapten outside the skin by a chemical reaction (oxidation in air or reaction with light) without the requirement of an enzyme. For prohaptens, it is possible to prevent activation outside the body to a certain extent by different measures, for example, 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 sensitizers. Prohaptens: Most terpenes with oxidisable allylic positions can be expected to self-oxidise on air exposure. Depending on the stability of the oxidation products that are formed, the oxidized products will have differing levels of sensitization potential. Tests shows that air exposure of lavender oil increased the potential for sensitization. Prohaptens: Compounds that are bioactivated in the skin and thereby form haptens are referred to prohaptens. The possibility of a prohaptens being activated cannot be avoided by outside measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Various enzymes play roles in both activating and deactivating prohaptens. Skin-sensitizing prohaptens can be recognized and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or studies of sensitization. QSAR prediction: Prediction of sensitization activity of these substances is complex, especially for those substances that can act both as pre- and prohaptens.
GERANIOL & LINALOOL	The terpenoid hydrocarbons are found in needle trees and deciduous plants. This category of chemicals shows very low acute toxicity. They are excreted in the urine. They are unlikely to cause genetic damage, but animal testing shows that they do cause increased rates of kidney cancer. They have low potential to cause reproductive and developmental toxicity.

	Current opinion holds that there are no safety concerns regarding the branched chain unsaturated non-cyclic alcohols, as fragrance ingredients, at current declared levels of use and exposure; however, use of these materials at higher maximum levels of skin or whole-body exposure requires re-evaluation. At current declared levels of use, there was no evidence or only minimal evidence of skin irritation in humans. Sensitising hydroperoxides may be formed by contact with air. It should be ensured that oxidation reactions are prevented in the end product. The use of these materials under the declared levels of use and exposure will not induce sensitization. These compounds generally have low acute toxicity. The branched chain, unsaturated alcohols tested had low whole-body toxicity after repeated application. In animals, repeated exposure at high doses caused liver changes and kidney damage. There was little or no evidence of adverse effects on fertility or development. Data on cancer-causing potential is not available, but they are not of primary concern. With few exceptions* (see below), there are no safety concerns regarding certain cyclic and non-cyclic terpene alcohols **, as fragrance ingredients, under present declared levels of use and exposure, because - They have low acute toxicity - No significant toxicity was observed in repeat dose toxicity tests - They were not found to cause mutations or genetic toxicity - Substances in this group are processed similarly in the body - There is no indication of persistent breakdown products causing severe toxicity - They practically do not irritate the skin - They have a generally low potential for sensitization - The margin of safety is more than 100 times the maximum daily exposure. *Safety concerns exist for the following substances for the following reasons: - 6,7-dihydrogeraniol, hydroabietyl alcohol and 2-isopropyl-2-decahydronaphthalenol are potent skin sensitizers. - Farnesol is a weak sensitizer. - Scaleryl and linalool may contain impurities and/or oxidation products that are strong sensitizers. - No sensitization test results were available for 2(10)-pinen-3-ol, 2,6-dimethyloct-3,5-dien-2-ol, and 3,7-dimethyl-4,6-octadien-3-ol. These materials should be regarded as potential sensitizers until tested. ** The common characteristic structural element of acyclic -noncyclic- and cyclic terpene alcohols is the typically branched isoprene unit 2-methyl-1,3-butadiene
2,6-DI-TERT-BUTYL-4-METHYLPHENOL & COUMARIN	Laboratory (in vitro) and animal studies show, exposure to the material may result in a possible risk of irreversible effects, with the possibility of producing mutation.

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

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

SECTION 12 Ecological information

Toxicity					
Personal Care Spiking Glue Mega Hold 3 oz	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
vinylpyrrolidone homopolymer	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
D-sorbitol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC10(ECx)	168h	Algae or other aquatic plants	8408.53mg/L	4
petrolatum	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
hydroxyethylcellulose	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
vinylpyrrolidone/ vinyl acetate copolymer	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
cetostearyl alcohol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	1666mg/l	1
	EC50	72h	Algae or other aquatic plants	0.33mg/l	2
	LC50	96h	Fish	0.012mg/l	2
	EC50	96h	Algae or other aquatic plants	235mg/l	1
	NOEC(ECx)	504h	Crustacea	0.98mg/l	1
sodium polyacrylate	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

Continued...

beeswax	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
aluminium starch octenylsuccinate	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
ethylene glycol phenyl ether	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	460mg/l	2
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	NOEC(ECx)	24h	Fish	5mg/l	2
	LC50	96h	Fish	154mg/l	2
polyethylene glycol (2) stearyl ether	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>=0.044<=14mg/l	2
	NOEC(ECx)	504h	Crustacea	>=0.001<=0.107mg/L	2
	LC50	96h	Fish	>=0.423<=8.211mg/l	2
sodium hydroxide	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	34.59-47.13mg/l	4
	EC50(ECx)	48h	Crustacea	34.59-47.13mg/l	4
	LC50	96h	Fish	144-267mg/l	4
cetyl alcohol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	>0.01mg/l	2
	EC50	72h	Algae or other aquatic plants	0.02mg/l	2
	EC50	96h	Algae or other aquatic plants	>0.047mg/L	2
	NOEC(ECx)	1440h	Fish	>=0.001mg/l	2
	LC50	96h	Fish	>0.01mg/l	2
stearyl alcohol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	1666mg/l	1
	EC50	72h	Algae or other aquatic plants	0.02mg/l	2
	EC50	96h	Algae or other aquatic plants	235mg/l	1
	NOEC(ECx)	504h	Crustacea	0.98mg/l	1
	LC50	96h	Fish	>0.01mg/l	2
propylene glycol	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	710mg/L	4
	EC50	48h	Crustacea	>114.4mg/L	4
	EC50	72h	Algae or other aquatic plants	19300mg/l	2
	NOEC(ECx)	336h	Algae or other aquatic plants	<5300mg/l	1
	EC50	96h	Algae or other aquatic plants	19000mg/l	2
dimethylaminoethyl methacrylate/ PVP/ N-vinylcaprolactam	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
hexyl salicylate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	0.357mg/l	2
	EC50	72h	Algae or other aquatic plants	0.28mg/l	2
	LC50	96h	Fish	1.34mg/l	2
	NOEC(ECx)	24h	Crustacea	0.14mg/l	2
3-iodo-2-propynyl butyl carbamate	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	0.05-0.089mg/L	4
	EC50	48h	Crustacea	0.04mg/L	5
	EC50	72h	Algae or other aquatic plants	0.022mg/L	2
	NOEC(ECx)	0.5h	Fish	<0.001mg/L	4
2-tert-butylcyclohexyl acetate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	17mg/l	2
	EC50	72h	Algae or other aquatic plants	4.2mg/l	2

	NOEC(ECx)	72h	Algae or other aquatic plants	0.57mg/l	2
	LC50	96h	Fish	5.6mg/l	2
benzyl acetate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	17mg/l	2
	EC50	72h	Algae or other aquatic plants	92mg/l	2
	NOEC(ECx)	672h	Fish	0.92mg/l	2
	LC50	96h	Fish	3.48-4.6mg/l	4
gamma-undecalactone	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	4mg/l	2
	EC50	72h	Algae or other aquatic plants	7.218mg/l	2
	NOEC(ECx)	504h	Crustacea	0.138mg/l	2
	EC50	96h	Algae or other aquatic plants	5mg/l	2
alpha,alpha-dimethylphenethyl butyrate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	96h	Algae or other aquatic plants	~4.766mg/l	2
	EC50	96h	Algae or other aquatic plants	~4.766mg/l	2
	LC50	96h	Fish	~8.901mg/l	2
	EC50	48h	Crustacea	~15.4mg/l	2
galaxolide	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	0.194mg/l	2
	EC50	72h	Algae or other aquatic plants	0.678-0.778mg/l	4
	NOEC(ECx)	3h	Crustacea	<=0.001mg/L	4
	LC50	96h	Fish	0.464-0.512mg/L	4
allyl heptanoate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	0.89mg/l	2
	EC50	72h	Algae or other aquatic plants	1.37mg/l	2
	EC10(ECx)	672h	Fish	0.056mg/L	2
	LC50	96h	Fish	0.051mg/L	2
d-limonene	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	0.46mg/l	2
	EC50	48h	Crustacea	0.307mg/l	2
	EC50	72h	Algae or other aquatic plants	0.214mg/l	2
	NOEC(ECx)	0h	Algae or other aquatic plants	<0.05-1.5mg/L	4
carvone	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	249.516mg/l	2
	EC50	72h	Algae or other aquatic plants	154.67mg/l	2
	EC50	96h	Algae or other aquatic plants	110mg/l	2
	EC50(ECx)	96h	Algae or other aquatic plants	110mg/l	2
	LC50	96h	Fish	50mg/l	2
	EC50	48h	Crustacea	>9.59mg/l	2
	EC50	72h	Algae or other aquatic plants	19mg/l	2
	LC50	96h	Fish	1.1mg/l	2
	EC50	96h	Algae or other aquatic plants	26mg/l	2
2,4-dimethyl-3-cyclohexene-1-carboxaldehyde	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
geraniol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	10.8mg/l	2
	EC50	72h	Algae or other aquatic plants	13.1mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	1mg/l	2
	LC50	96h	Fish	2.3-3mg/l	4

benzyl C8-18 alkyldimethylammonium chloride	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	0.31mg/L	4
	NOEC(ECx)	72h	Fish	9.6mg/L	4
linalool	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	<19.9mg/l	1
	EC50	48h	Crustacea	20mg/l	1
	NOEC(ECx)	96h	Fish	<3.5mg/l	1
2,6-di-tert-butyl-4- methylphenol	EC50	96h	Algae or other aquatic plants	88.3mg/l	1
	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1344h	Fish	220-2800	7
	EC50	48h	Crustacea	>0.17mg/l	2
	EC50	72h	Algae or other aquatic plants	>0.42mg/l	1
	ErC50	72h	Algae or other aquatic plants	>0.42mg/l	1
	EC50	96h	Algae or other aquatic plants	0.758mg/l	2
coumarin	EC0(ECx)	48h	Crustacea	>=0.31mg/l	1
	LC50	96h	Fish	0.199mg/l	2
	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	8.012mg/l	2
	EC50	96h	Algae or other aquatic plants	1.452mg/l	2
Legend:	NOEC(ECx)	1440h	Fish	0.119mg/l	2
	LC50	96h	Fish	1.324mg/l	2
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					

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

Non-ionic polymers with MWs > 1,000 that do not contain reactive functional groups and are comprised of minimal low MW oligomers are estimated to display no effects at saturation (NES). These polymers display NES because the amount dissolved in water is not anticipated to reach a concentration at which adverse effects may be expressed. Guidance for the assessment of aquatic toxicity hazard results in a Low hazard designation for those materials that display NES.

For high molecular weight synthetic polymers: (according to the Sustainable Futures (SF) program (U.S. EPA 2005b; U.S. EPA 2012c) polymer assessment guidance.)

High MW polymers are expected:

- to have low vapour pressure and are not expected to undergo volatilization .
- to adsorb strongly to soil and sediment
- to be non-biodegradable (not anticipated to be assimilated by microorganisms.- therefore, biodegradation is not expected to be an important removal process. However many exceptions exist

High MW polymers are not expected to undergo removal by other degradative processes under environmental conditions

Cellulosic products, including cellulose ethers, generally have a low biodegradation rate and are generally of low toxicity to fish.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
vinylpyrrolidone homopolymer	LOW	LOW
D-sorbitol	LOW	LOW
hydroxyethylcellulose	LOW	LOW
ethylene glycol phenyl ether	LOW	LOW
polyethylene glycol (2) stearyl ether	HIGH	HIGH
sodium hydroxide	LOW	LOW
cetyl alcohol	LOW	LOW
stearyl alcohol	LOW	LOW
propylene glycol	LOW	LOW
hexyl salicylate	LOW	LOW
3-iodo-2-propynyl butyl carbamate	HIGH	HIGH
2-tert-butylcyclohexyl acetate	HIGH	HIGH
benzyl acetate	LOW	LOW
gamma-undecalactone	LOW	LOW
alpha, alpha-dimethylphenethyl butyrate	HIGH	HIGH
galaxolide	HIGH	HIGH
allyl heptanoate	LOW	LOW
d-limonene	HIGH	HIGH
carvone	HIGH	HIGH
2,4-dimethyl-3-cyclohexene-1-carboxaldehyde	LOW	LOW

Ingredient	Persistence: Water/Soil	Persistence: Air
geraniol	LOW	LOW
linalool	HIGH	HIGH
2,6-di-tert-butyl-4-methylphenol	HIGH	HIGH
coumarin	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
vinylpyrrolidone homopolymer	LOW (LogKOW = 0.29)
D-sorbitol	LOW (LogKOW = -3.0108)
petrolatum	LOW (LogKOW = 12.05)
hydroxyethylcellulose	LOW (LogKOW = -8.995)
cetostearyl alcohol	MEDIUM (BCF = 1300)
ethylene glycol phenyl ether	LOW (LogKOW = 1.16)
polyethylene glycol (2) stearyl ether	HIGH (LogKOW = 6.34)
sodium hydroxide	LOW (LogKOW = -3.88)
cetyl alcohol	HIGH (LogKOW = 6.73)
stearyl alcohol	LOW (LogKOW = 7.72)
propylene glycol	LOW (BCF = 1)
hexyl salicylate	MEDIUM (LogKOW = 3.8035)
3-iodo-2-propynyl butyl carbamate	LOW (LogKOW = 2.4542)
2-tert-butylcyclohexyl acetate	MEDIUM (LogKOW = 4.42)
benzyl acetate	LOW (LogKOW = 1.96)
gamma-undecalactone	LOW (LogKOW = 3.06)
alpha,alpha-dimethylphenethyl butyrate	MEDIUM (LogKOW = 4.43)
galaxolide	HIGH (LogKOW = 5.9)
allyl heptanoate	LOW (LogKOW = 3.67)
d-limonene	HIGH (LogKOW = 4.8275)
carvone	LOW (LogKOW = 2.71)
2,4-dimethyl-3-cyclohexene-1-carboxaldehyde	LOW (LogKOW = 2.85)
geraniol	LOW (LogKOW = 3.47)
linalool	LOW (LogKOW = 2.97)
2,6-di-tert-butyl-4-methylphenol	HIGH (BCF = 2500)
coumarin	LOW (LogKOW = 1.39)

Mobility in soil

Ingredient	Mobility
vinylpyrrolidone homopolymer	LOW (Log KOC = 40.46)
D-sorbitol	LOW (Log KOC = 10)
hydroxyethylcellulose	LOW (Log KOC = 10)
ethylene glycol phenyl ether	LOW (Log KOC = 12.12)
polyethylene glycol (2) stearyl ether	LOW (Log KOC = 10000000000)
sodium hydroxide	LOW (Log KOC = 14.3)
cetyl alcohol	LOW (Log KOC = 3786)
stearyl alcohol	LOW (Log KOC = 12880)
propylene glycol	HIGH (Log KOC = 1)
hexyl salicylate	LOW (Log KOC = 2736)
3-iodo-2-propynyl butyl carbamate	LOW (Log KOC = 365.3)
2-tert-butylcyclohexyl acetate	LOW (Log KOC = 528.1)
benzyl acetate	LOW (Log KOC = 133.7)
gamma-undecalactone	LOW (Log KOC = 476.5)
alpha,alpha-dimethylphenethyl butyrate	LOW (Log KOC = 2089)
galaxolide	LOW (Log KOC = 10380)
allyl heptanoate	LOW (Log KOC = 252.8)
d-limonene	LOW (Log KOC = 1324)
carvone	LOW (Log KOC = 123.7)
2,4-dimethyl-3-cyclohexene-1-carboxaldehyde	LOW (Log KOC = 87.49)
geraniol	LOW (Log KOC = 70.79)

Ingredient	Mobility
linalool	LOW (Log KOC = 56.32)
2,6-di-tert-butyl-4-methylphenol	LOW (Log KOC = 23030)
coumarin	LOW (Log KOC = 146.1)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▶ 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.
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SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

14.7. Maritime transport in bulk according to IMO instruments

14.7.1. Transport in bulk according to Annex II of MARPOL and the IBC code
Not Applicable

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

Product name	Group
vinylpyrrolidone homopolymer	Not Available
D-sorbitol	Not Available
petrolatum	Not Available
hydroxyethylcellulose	Not Available
vinylpyrrolidone/ vinyl acetate copolymer	Not Available
cetostearyl alcohol	Not Available
sodium polyacrylate	Not Available
beeswax	Not Available
aluminium starch octenylsuccinate	Not Available
ethylene glycol phenyl ether	Not Available
polyethylene glycol (2) stearyl ether	Not Available
sodium hydroxide	Not Available
cetyl alcohol	Not Available
stearyl alcohol	Not Available
propylene glycol	Not Available
dimethylaminoethyl methacrylate/ PVP/ N-vinylcaprolactam	Not Available
hexyl salicylate	Not Available
3-iodo-2-propynyl butyl carbamate	Not Available
2-tert-butylcyclohexyl acetate	Not Available
benzyl acetate	Not Available

Product name	Group
gamma-undecalactone	Not Available
alpha,alpha-dimethylphenethyl butyrate	Not Available
galaxolide	Not Available
allyl heptanoate	Not Available
d-limonene	Not Available
carvone	Not Available
2,4-dimethyl-3-cyclohexene-1-carboxaldehyde	Not Available
geraniol	Not Available
benzyl C8-18 alkyl(dimethylammonium chloride	Not Available
linalool	Not Available
2,6-di-tert-butyl-4-methylphenol	Not Available
coumarin	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
vinylpyrrolidone homopolymer	Not Available
D-sorbitol	Not Available
petrolatum	Not Available
hydroxyethylcellulose	Not Available
vinylpyrrolidone/ vinyl acetate copolymer	Not Available
cetostearyl alcohol	Not Available
sodium polyacrylate	Not Available
beeswax	Not Available
aluminium starch octenylsuccinate	Not Available
ethylene glycol phenyl ether	Not Available
polyethylene glycol (2) stearyl ether	Not Available
sodium hydroxide	Not Available
cetyl alcohol	Not Available
stearyl alcohol	Not Available
propylene glycol	Not Available
dimethylaminoethyl methacrylate/ PVP/ N-vinylcaprolactam	Not Available
hexyl salicylate	Not Available
3-iodo-2-propynyl butyl carbamate	Not Available
2-tert-butylcyclohexyl acetate	Not Available
benzyl acetate	Not Available
gamma-undecalactone	Not Available
alpha,alpha-dimethylphenethyl butyrate	Not Available
galaxolide	Not Available
allyl heptanoate	Not Available
d-limonene	Not Available
carvone	Not Available
2,4-dimethyl-3-cyclohexene-1-carboxaldehyde	Not Available
geraniol	Not Available
benzyl C8-18 alkyl(dimethylammonium chloride	Not Available
linalool	Not Available
2,6-di-tert-butyl-4-methylphenol	Not Available
coumarin	Not Available

SECTION 15 Regulatory information

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

vinylpyrrolidone homopolymer is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

D-sorbitol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

petrolatum is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

hydroxyethylcellulose is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

vinylpyrrolidone/ vinyl acetate copolymer is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

cetostearyl alcohol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

sodium polyacrylate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

beeswax is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

aluminium starch octenylsuccinate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

ethylene glycol phenyl ether is found on the following regulatory lists

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

polyethylene glycol (2) stearyl ether is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

sodium hydroxide is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australian Inventory of Industrial Chemicals (AIIC)

cetyl alcohol is found on the following regulatory lists

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

stearyl alcohol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

propylene glycol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

dimethylaminoethyl methacrylate/ PVP/ N-vinylcaprolactam is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

hexyl salicylate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

3-iodo-2-propynyl butyl carbamate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

2-tert-butylcyclohexyl acetate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

benzyl acetate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

gamma-undecalactone is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

alpha,alpha-dimethylphenethyl butyrate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

galaxolide is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

allyl heptanoate is found on the following regulatory lists

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

d-limonene is found on the following regulatory lists

- Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
- Australian Inventory of Industrial Chemicals (AIIC)
- International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

carvone is found on the following regulatory lists

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

2,4-dimethyl-3-cyclohexene-1-carboxaldehyde is found on the following regulatory lists

- Australian Inventory of Industrial Chemicals (AIIC)

geraniol is found on the following regulatory lists

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

benzyl C8-18 alkyl dimethylammonium chloride is found on the following regulatory lists

- Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
- Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
- Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
- Australian Inventory of Industrial Chemicals (AIIC)

linalool is found on the following regulatory lists

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

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

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

coumarin is found on the following regulatory lists

- Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
- Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
- Australian Inventory of Industrial Chemicals (AIIC)
- FEI Equine Prohibited Substances List - Banned Substances
- FEI Equine Prohibited Substances List (EPSL)
- International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (vinylpyrrolidone homopolymer; D-sorbitol; petrolatum; hydroxyethylcellulose; vinylpyrrolidone/ vinyl acetate copolymer; cetostearyl alcohol; sodium polyacrylate; beeswax; aluminium starch octenylsuccinate; ethylene glycol phenyl ether; polyethylene glycol (2) stearyl ether; sodium hydroxide; cetyl alcohol; stearyl alcohol; propylene glycol; dimethylaminoethyl methacrylate/ PVP/ N-vinylcaprolactam; hexyl salicylate; 3-iodo-2-propynyl butyl carbamate; 2-tert-butylcyclohexyl acetate; benzyl acetate; gamma-undecalactone; alpha, alpha-dimethylphenethyl butyrate; galaxolide; allyl heptanoate; d-limonene; carvone; 2,4-dimethyl-3-cyclohexene-1-carboxaldehyde; geraniol; benzyl C8-18 alkyl dimethylammonium chloride; linalool; coumarin)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (vinylpyrrolidone homopolymer; hydroxyethylcellulose; vinylpyrrolidone/ vinyl acetate copolymer; sodium polyacrylate; aluminium starch octenylsuccinate; dimethylaminoethyl methacrylate/ PVP/ N-vinylcaprolactam)
Japan - ENCS	No (dimethylaminoethyl methacrylate/ PVP/ N-vinylcaprolactam; benzyl C8-18 alkyl dimethylammonium chloride)
Korea - KECI	No (aluminium starch octenylsuccinate)
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	No (cetostearyl alcohol; beeswax; aluminium starch octenylsuccinate; hexyl salicylate)
Vietnam - NCI	Yes
Russia - FBEPH	No (aluminium starch octenylsuccinate; dimethylaminoethyl methacrylate/ PVP/ N-vinylcaprolactam)
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	30/05/2025
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Personal Care Spiking Glue Mega Hold 3 oz

Initial Date	30/05/2025
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Other information

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

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

Definitions and abbreviations

- PC – TWA: Permissible Concentration-Time Weighted Average
- PC – STEL: Permissible Concentration-Short Term Exposure Limit
- IARC: International Agency for Research on Cancer
- ACGIH: American Conference of Governmental Industrial Hygienists
- STEL: Short Term Exposure Limit
- TEEL: Temporary Emergency Exposure Limit.
- IDLH: Immediately Dangerous to Life or Health Concentrations
- ES: Exposure Standard
- OSF: Odour Safety Factor
- NOAEL: No Observed Adverse Effect Level
- LOAEL: Lowest Observed Adverse Effect Level
- TLV: Threshold Limit Value
- LOD: Limit Of Detection
- OTV: Odour Threshold Value
- BCF: BioConcentration Factors
- BEI: Biological Exposure Index
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration
- MARPOL: International Convention for the Prevention of Pollution from Ships
- IMSBC: International Maritime Solid Bulk Cargoes Code
- IGC: International Gas Carrier Code
- IBC: International Bulk Chemical Code

- 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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