BUGALUGS Baby Fresh Cologne

Adeval Group Pty Ltd

Part Number: **BCLBF200, BCLBF5L**
Version No: **1.2**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

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

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

Product Identifier

Product name	BUGALUGS Baby Fresh Cologne
Synonyms	Not Available
Proper shipping name	ETHANOL (ETHYL ALCOHOL) or ETHANOL SOLUTION (ETHYL ALCOHOL SOLUTION)
Other means of identification	BCLBF200, BCLBF5L

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

Relevant identified uses	Dog perfuming spray

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**Adeval Group Pty Ltd**
Address	276 Proximity Drive Sunshine West Victoria 3020 Australia
Telephone	03 8566 7660
Fax	Not Available
Website	www.garth.com.au
Email	info@garth.com.au

Emergency telephone number

Association / Organisation	**Poisons Information Centre**
Emergency telephone numbers	**13 11 26**
Other emergency telephone numbers	**000**

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Poisons Schedule	S5
Classification [1]	Serious Eye Damage/Eye Irritation Category 2A, Flammable Liquids Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
Legend:	*1. Classification by vendor; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

Hazard pictogram(s)	

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Signal word	**Danger**

Hazard statement(s)

H319	Causes serious eye irritation.
H225	Highly flammable liquid and vapour.
H412	Harmful to aquatic life with long lasting effects.

Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P233	Keep container tightly closed.
P240	Ground and bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use non-sparking tools.
P243	Take action to prevent static discharges.
P273	Avoid release to the environment.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].

Precautionary statement(s) Storage

P403+P235	Store in a well-ventilated place. Keep cool.

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
64-17-5	50-70	ethanol
127-51-5	0.1-0.25	isomethyl-alpha-ionone
106-22-9	0.1-0.25	beta-citronellol
91-64-5	0.1-0.25	coumarin
2050-08-0	0.1-0.25	amyl salicylate
34902-57-3	0.1-0.25	12-musk decenone
81-13-0	0.1-0.25	d-panthenol
77-92-9	0.1-0.25	citric acid
85507-69-3	0.1-0.25	Aloes, extract

Legend:	*1. Classification by vendor; 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

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Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Wash out immediately with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ Immediately give a glass of water. ▸ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

Indication of any immediate medical attention and special treatment needed

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 http://www.ozemail.com.au/-ouad/SALI0001.HTA

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citing from :
MARTINDALE: The Extra Pharmacopoeia, 27th Ed.
For acute or short term repeated exposures to ethanol:

- Acute ingestion in non-tolerant patients usually responds to supportive care with special attention to prevention of aspiration, replacement of fluid and correction of nutritional deficiencies (magnesium, thiamine pyridoxine, Vitamins C and K).
- Give 50% dextrose (50-100 ml) IV to obtunded patients following blood draw for glucose determination.
- Comatose patients should be treated with initial attention to airway, breathing, circulation and drugs of immediate importance (glucose, thiamine).
- Decontamination is probably unnecessary more than 1 hour after a single observed ingestion. Cathartics and charcoal may be given but are probably not effective in single ingestions.
- Fructose administration is contra-indicated due to side effects.

SECTION 5 Firefighting measures

Extinguishing media

- Alcohol stable foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.
- Water spray or fog - Large fires only.

Special hazards arising from the substrate or mixture

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

Advice for firefighters

Fire Fighting	
Fire/Explosion Hazard	‣ Liquid and vapour are highly flammable. ‣ Severe fire hazard when exposed to heat, flame and/or oxidisers. ‣ Vapour may travel a considerable distance to source of ignition. ‣ Heating may cause expansion or decomposition leading to violent rupture of containers. ‣ On combustion, may emit toxic fumes of carbon monoxide (CO). Combustion products include: carbon dioxide (CO_2) other pyrolysis products typical of burning organic material.
HAZCHEM	•2Y

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	‣ Remove all ignition sources. ‣ Clean up all spills immediately. ‣ Avoid breathing vapours and contact with skin and eyes. ‣ Control personal contact with the substance, by using protective equipment. ‣ Contain and absorb small quantities with vermiculite or other absorbent material. ‣ Wipe up. ‣ Collect residues in a flammable waste container.
Major Spills	‣ Clear area of personnel and move upwind. ‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear full body protective clothing with breathing apparatus. ‣ Prevent, by all means available, spillage from entering drains or water courses. ‣ Consider evacuation (or protect in place). ‣ No smoking, naked lights or ignition sources. ‣ Increase ventilation. ‣ Stop leak if safe to do so. ‣ Water spray or fog may be used to disperse / absorb vapour. ‣ Contain or absorb spill with sand, earth or vermiculite. ‣ Collect recoverable product into labelled containers for recycling.

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	▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▸ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ Containers, even those that have been emptied, may contain explosive vapours. ▸ Do NOT cut, drill, grind, weld or perform similar operations on or near containers. ▸ 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, heat or ignition sources. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Vapour may ignite on pumping or pouring due to static electricity. ▸ **DO NOT** use plastic buckets. ▸ Earth and secure metal containers when dispensing or pouring product. ▸ Use spark-free tools when handling. ▸ Avoid contact with incompatible materials. ▸ Keep containers securely sealed. ▸ 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. ▸ **DO NOT** allow clothing wet with material to stay in contact with skin
Other information	▸ Store in original containers in approved flame-proof area. ▸ No smoking, naked lights, heat or ignition sources. ▸ **DO NOT** store in pits, depression, basement or areas where vapours may be trapped. ▸ Keep containers securely sealed. ▸ Store away from incompatible materials in a cool, dry well ventilated area. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this MSDS. ▸ Tank storage: Tanks must be specifically designed for use with this product. Bulk storage tanks should be diked (bunded). Locate tanks away from heat and other sources of ignition. Cleaning, inspection and maintenance of storage tanks is a specialist operation, which requires the implementation of strict procedures and precautions. ▸ Keep in a cool place. Electrostatic charges will be generated during pumping. Electrostatic discharge may cause fire. Ensure electrical continuity by bonding and grounding (earthing) all equipment to reduce the risk. The vapours in the head space of the storage vessel may lie in the flammable/explosive range and hence may be flammable. ▸ For containers, or container linings use mild steel, stainless steel. Examples of suitable materials are: high density polyethylene (HDPE), polypropylene (PP), and Viton (FMK), which have been specifically tested for compatibility with this product. ▸ For container linings, use amine-adduct cured epoxy paint. ▸ For seals and gaskets use: graphite, PTFE, Viton A, Viton B. ▸ Unsuitable material: Some synthetic materials may be unsuitable for containers or container linings depending on the material specification and intended use. Examples of materials to avoid are: natural rubber (NR), nitrile rubber (NBR), ethylene propylene rubber (EPDM), polymethyl methacrylate (PMMA), polystyrene, polyvinyl chloride (PVC), polyisobutylene. However, some may be suitable for glove materials. ▸ Do not cut, drill, grind, weld or perform similar operations on or near containers. Containers, even those that have been emptied, can contain explosive vapours.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Packing as supplied by manufacturer. ▸ Plastic containers may only be used if approved for flammable liquid. ▸ Check that containers are clearly labelled and free from leaks. ▸ For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : Where a can is to be used as an inner package, the can must have a screwed enclosure. ▸ For materials with a viscosity of at least 2680 cSt. (23 deg. C) ▸ For manufactured product having a viscosity of at least 250 cSt. (23 deg. C) ▸ Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used.

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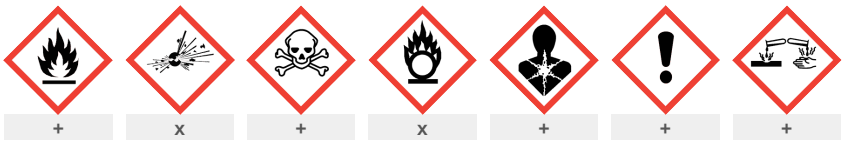
	▸ Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages ▸ In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	▸ Avoid oxidising agents, acids, acid chlorides, acid anhydrides, chloroformates. ▸ Avoid strong bases. *



+	x	+	x	+	+	+

X — *Must not be stored together*
0 — *May be stored together with specific preventions*
+ — *May be stored together*

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

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	ethanol	Ethyl alcohol	1000 ppm / 1880 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
ethanol	Not Available	Not Available	15000* ppm
coumarin	0.88 mg/m3	9.7 mg/m3	58 mg/m3

Ingredient	Original IDLH	Revised IDLH
ethanol	3,300 ppm	Not Available
isomethyl-alpha-ionone	Not Available	Not Available
beta-citronellol	Not Available	Not Available
coumarin	Not Available	Not Available
amyl salicylate	Not Available	Not Available
12-musk decenone	Not Available	Not Available
d-panthenol	Not Available	Not Available
citric acid	Not Available	Not Available
Aloes, extract	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
isomethyl-alpha-ionone	D	> 0.1 to ≤ 1 ppm
beta-citronellol	E	≤ 0.1 ppm
coumarin	E	≤ 0.01 mg/m³
amyl salicylate	E	≤ 0.1 ppm
citric acid	E	≤ 0.01 mg/m³
Aloes, extract	C	> 0.1 to ≤ milligrams per cubic meter of air (mg/m³)
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

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

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

Fragrance substance with is an established contact allergen in humans.
Scientific Committee on Consumer Safety SCCS OPINION on Fragrance allergens in cosmetic products 2012
For ethanol:
Odour Threshold Value: 49-716 ppm (detection), 101 ppm (recognition)
Eye and respiratory tract irritation do not appear to occur at exposure levels of less than 5000 ppm and the TLV-TWA is thought to provide an adequate margin of safety against such effects. Experiments in man show that inhalation of 1000 ppm caused slight symptoms of poisoning and 5000 ppm caused strong stupor and morbid sleepiness. Subjects exposed to 5000 ppm to 10000 ppm experienced smarting of the eyes and nose and coughing. Symptoms disappeared within minutes. Inhalation also causes local irritating effects to the eyes and upper respiratory tract, headaches, sensation of heat intraocular tension, stupor, fatigue and a need to sleep. At 15000 ppm there was continuous lachrymation and coughing.

These exposure guidelines have been derived from a screening level of risk assessment and should not be construed as unequivocally safe limits. ORGS represent an 8-hour time-weighted average unless specified otherwise.
CR = Cancer Risk/10000; UF = Uncertainty factor:
TLV believed to be adequate to protect reproductive health:
LOD: Limit of detection
Toxic endpoints have also been identified as:
D = Developmental; R = Reproductive; TC = Transplacental carcinogen
Jankovic J., Drake F.: A Screening Method for Occupational Reproductive
American Industrial Hygiene Association Journal 57: 641-649 (1996)

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

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

The Odour Safety Factor (OSF) is defined as:

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

Classification into classes follows:

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

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. ▸ Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area. ▸ Work should be undertaken in an isolated system such as a "glove-box" . Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system. ▸ Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within. ▸ Open-vessel systems are prohibited.

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	‣ Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation. ‣ Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system. ‣ For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ‣ Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas). ‣ Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air. ‣ Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed.
Individual protection measures, such as personal protective equipment	
Eye and face protection	‣ Safety glasses with side shields. ‣ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ‣ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].
Skin protection	See Hand protection below
Hands/feet protection	‣ Wear chemical protective gloves, e.g. PVC. ‣ Wear safety footwear or safety gumboots, e.g. Rubber **NOTE:** ‣ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ‣ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · 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.

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	· 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	‣ Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent] ‣ Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent] ‣ Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely. ‣ Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. ‣ Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ‣ Overalls. ‣ PVC Apron. ‣ PVC protective suit may be required if exposure severe. ‣ Eyewash unit. ‣ Ensure there is ready access to a safety shower. ‣ Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity. ‣ For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets). ‣ Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.

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 5 x ES	Air-line*	A-2 P2	A-PAPR-2 P2 ^
up to 10 x ES	-	A-3 P2	-
10+ x ES	-	Air-line**	-

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

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Colourless

Physical state	Liquid	**Relative density (Water = 1)**	Not Available

Continued...

Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	5.25-5.75	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	80-100	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	23	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Flammable.	**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

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. The most common signs of inhalation overexposure to ethanol, in animals, include ataxia, incoordination and drowsiness for those surviving narcosis. The narcotic dose for rats, after 2 hours of exposure, is 19260 ppm. The material has **NOT** been classified by EC Directives or other classification systems as "harmful by inhalation". This is because of the lack of corroborating animal or human evidence. In the absence of such evidence, care should be taken nevertheless to ensure exposure is kept to a minimum and that suitable control measures be used, in an occupational setting to control vapours, fumes and aerosols. Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.

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Ingestion

Ingestion of ethanol (ethyl alcohol, "alcohol") may produce nausea, vomiting, bleeding from the digestive tract, abdominal pain, and diarrhoea. Effects on the body:

Blood concentration	Effects
<1.5 g/L	Mild: impaired vision, co-ordination and reaction time; emotional instability
1.5-3.0 g/L	Moderate: Slurred speech, confusion, inco-ordination, emotional instability, disturbances in perception and senses, possible blackouts, and impaired objective performance in standardized tests. Possible double vision, flushing, fast heart rate, sweating and incontinence. Slow breathing may occur rarely and fast breathing may develop in cases of metabolic acidosis, low blood sugar and low blood potassium. Central nervous system depression may progress to coma.
3-5 g/L	Severe: cold clammy skin, low body temperature and low blood pressure. Atrial fibrillation and heart block have been reported. Depression of breathing may occur, respiratory failure may follow serious poisoning, choking on vomit may result in lung inflammation and swelling. Convulsions due to severe low blood sugar may also occur. Acute liver inflammation may develop.

Large oral doses of salicylates may cause mild burning pain in the throat, stomach and usually prompt vomiting. Several hours may elapse before the development of deep and rapid breathing, lassitude, anorexia, nausea, vomiting, thirst and occasional diarrhoea. Common derivatives of salicylic acid produce substantially the same toxic syndrome, ("salicylism"). Major signs and symptoms arise from stimulation and terminal depression of the central nervous system. Stimulation produces vomiting, hyperpnea (abnormal increase in rate and depth of respiration), headache, tinnitus (ringing in the ears) confusion, bizarre behaviour or mania, generalised convulsions. Death is due to respiratory failure or cardiovascular collapse. Severe sensory disturbances such as deafness and dimness of vision are common. Less common features include sweating, skin eruptions, gastrointestinal and other hemorrhages, renal failure and pancreatitis. A tendency to bleed may be manifest by blood in the vomitus (haematemesis), bloody stools (melena) or purplish-red spots (petechiae) on the skin. Many of the toxic effects detailed here are due to or aggravated by severe disturbance of acid-base balance with the chief cause being prolonged hyperventilation from central stimulation. An assessment of acute salicylate intoxication based on dose suggests; 500 mg/kg: Potentially lethal
The material has **NOT** been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health).
Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.
Accidental ingestion of the material may be damaging to the health of the individual.

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.
Open cuts, abraded or irritated skin should not be exposed to this material
Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
The material may produce moderate skin irritation; limited evidence or practical experience suggests, that the material either:

- produces moderate inflammation of the skin in a substantial number of individuals following direct contact and/or
- produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period.

Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.

Eye

Direct contact of the eye with ethanol may cause immediate stinging and burning with reflex closure of the lid and tearing, transient injury of the corneal epithelium and hyperaemia of the conjunctiva. Foreign-body type discomfort may persist for up to 2 days but healing is usually spontaneous and complete.
Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may

Continued...

	cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. On the basis of epidemiological data, the material is regarded as carcinogenic to humans. There is sufficient data to establish a causal association between human exposure to the material and the development of cancer. Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. There is sufficient evidence to establish a causal relationship between human exposure to the material and impaired fertility Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Long-term exposure to ethanol may result in progressive liver damage with fibrosis or may exacerbate liver injury caused by other agents. Repeated ingestion of ethanol by pregnant women may adversely affect the central nervous system of the developing foetus, producing effects collectively described as foetal alcohol syndrome. These include mental and physical retardation, learning disturbances, motor and language deficiency, behavioural disorders and reduced head size. Consumption of ethanol (in alcoholic beverages) may be linked to the development of Type I hypersensitivities in a small number of individuals. Symptoms, which may appear immediately after consumption, include conjunctivitis, angioedema, dyspnoea, and urticarial rashes. The causative agent may be acetic acid, a metabolite (1). (1) Boehncke W.H., & H.Gall, Clinical & Experimental Allergy, 26, 1089-1091, 1996 Mild chronic salicylate intoxication, or "salicylism", may occur after repeated exposures to large doses. Symptoms include dizziness, tinnitus, deafness, sweating, nausea and vomiting, headache and mental confusion. Symptoms of more severe intoxication include hyperventilation, fever, restlessness, ketosis, and respiratory alkalosis and metabolic acidosis. Depression of the central nervous system may lead to coma, cardiovascular collapse and respiratory failure. Chronic exposure to the salicylates (o-hydroxybenzoates) may produce metabolic and central system disturbances or damage to the kidneys. Persons with pre-existing skin disorders, eye problems or impaired kidney function may be more susceptible to the effects of these substances. Certain individuals (atopics), notably asthmatics, exhibit significant hyper- sensitivity to salicylic acid derivatives. Reactions include urticaria and other skin eruptions, rhinitis and severe (even fatal) bronchospasm and dyspnea. Chronic exposure to the p-hydroxybenzoates (parabens) is associated with hypersensitivity reactions following application of these to the skin. Hypersensitivity reactions have also been reported following parenteral or oral administration. Cross-sensitivity occurs between the p-hydroxybenzoates Hypersensitivity reactions may include by acute bronchospasm, hives (urticaria), deep dermal wheals (angioneurotic oedema), running nose (rhinitis) and blurred vision. Anaphylactic shock and skin rash (non-thrombocytopenic purpura) may also occur. Any individual may be predisposed to such anti-body mediated reaction if other chemical agents have caused prior sensitisation (cross-sensitivity).

BUGALUGS Baby Fresh Cologne	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

ethanol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 17100 mg/kg[1]	Eye (rabbit): 500 mg SEVERE
	Inhalation(Rat) LC50: 64000 ppm4h[2]	Eye (rabbit):100mg/24hr-moderate
	Oral (Rat) LD50: 7060 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
		Skin (rabbit):20 mg/24hr-moderate

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		Skin (rabbit):400 mg (open)-mild
		Skin: no adverse effect observed (not irritating)[1]
isomethyl-alpha-ionone	**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: no adverse effect observed (not irritating)[1]
beta-citronellol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 2650 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
	Oral (Rat) LD50: 3450 mg/kg[2]	Skin (guin.pig): 100mg/24h-SEVERE
		Skin (man): 16 mg/48h - mod
		Skin (rabbit): 100 mg/24h-SEVERE
		Skin: adverse effect observed (irritating)[1]
coumarin	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: 293 mg/kg[1]	Not Available
	Oral (Rat) LD50: ~290 mg/kg[1]	
amyl salicylate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: ~2000 mg/kg[1]	Skin (rabbit): 100 mg/24h SEVERE
		Skin: no adverse effect observed (not irritating)[1]
12-musk decenone	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[2]	Eye (rabbit): slight *
	Oral (Rat) LD50: >2000 mg/kg[2]	Skin (rabbit): slight *
d-panthenol	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit): 0.5 mg - mild
	Oral (Mouse) LD50; 15000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin (rabbit): 500 mg/4h - mild
		Skin: no adverse effect observed (not irritating)[1]
citric acid	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit): 0.75 mg/24h-SEVERE
	Oral (Rat) LD50: 3000 mg/kg[2]	Skin (rabbit): 500 mg/24h - mild
Aloes, extract	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
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*	

ETHANOL	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
ISOMETHYL-ALPHA-IONONE	For ionone, damascones and other other structurally related compounds: **Human Health:** The only hazards identified are slight irritation to the eyes and changes in the liver, kidneys and thyroid after repeated oral exposure, which were either of minor severity or were considered to be a species-specific effect in male rats beta-Ionone has only low acute toxicity after oral ingestion. From animal experiments it can be concluded that beta-ionone is absorbed after oral exposure. Metabolism takes place mainly in the liver. Metabolites, which were identified in the urine of exposed rabbits, are 3-oxo-beta-ionone, 3-oxo-beta-ionol, dihydro-3-oxo-beta-ionol and 3-hydroxy-beta-ionol. beta-Ionone was found to be an inducer of CYP 1A and 2B isozymes in the liver of rodents. A gavage study conducted with a mixture of 60 % alpha-Ionone and 40 % beta-ionone revealed a LD50 of 4590 mg/kg bw. Clinical signs of toxicity were depression and tremors. In studies conducted according to OECD test guidelines and under GLP conditions, beta-ionone was not irritating to the skin of rabbits after semiocclusive application for 4 hours and only slightly irritating to the eyes. After a 24-hours exposure under occlusive conditions, a slight irritation of the skin was observed in rabbits. A limited human patch test did not reveal a potential for skin irritation when a not further specified mixture of alpha-- and

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beta-ionone was applied undiluted to the skin of volunteers. A limited Guinea pig maximization test found no evidence that beta-ionone is a dermal sensitiser. According to secondary sources, ionone (a not further specified mixture of alpha-- and beta-ionone) was negative in an open epicutaneous test with Guinea pigs as well as in a human maximisation test with a product containing 97.5 % alpha-ionone and 2.5 % beta-ionone.
The administration of beta-ionone over a period of 90 days according to OECD TG 408 at dietary concentrations of 100, 1000 and 10 000 ppm (7 and 8 mg/kg bw/day, 72 and 83 mg/kg bw/day or 720 and 801 mg/kg bw/day for males and females) to rats led to signs of general systemic toxicity at the high and mid dose. Target organs were liver, kidneys and thyroid glands. The liver findings in both sexes and the increased kidney weights in high dose females were indicative of adaptive and most likely reversible processes with the aim to increase the metabolising and/or excretory capacity of these organs. The findings in males with respect to kidneys as well as kidney relevant parameters should be seen in the light of high amounts of alpha2u-globulin in these animals. The occurrence of alpha2u-globulin was confirmed by immunohistochemical examination. The accumulation of this protein appears to be a unique feature of male rats and is not known to occur in other species, including man. No signs of neurotoxicity were observed during functional observational battery as well as measurement of motor activity performed towards the end of the administration period.
Thus, the no-observed-effect-level (NOEL) under the conditions of the present study was 100 ppm for both sexes (about 7 and 8 mg/kg bw/day for males and females) based on adaptive liver effects in both sexes and minor urine findings in males at 1000 ppm which correspond to a dosage of 72 and 83 mg/kg bw/day for males and females (no-observed-adverse-effect-level, NOAEL). The lowest-observed-adverse-effect-level (LOAEL) was found at 10 000 ppm (720 and 801 mg/kg bw/day for males and females) due to liver, kidney and thyroid findings in both sexes. beta-Ionone gave no indication of a mutagenic effect in bacteria or a clastogenic potential in an *in vivo* mouse micronucleus test. Therefore, there is no indication of a genotoxic potential *in vivo*. No studies that would be considered adequate for the evaluation of carcinogenic potential were available. A short-term screening experiment investigating a tumor-promoting potential on mouse skin did not indicate such an effect at a low test concentration. In a well-conducted 90 days study in rats according to OECD TG 408 with administration of the test substance in the diet, beta-ionone did not have the potential to damage the reproductive organs at least up to the highest tested concentration of 10,000 ppm (720 and 801 mg/kg bw/day for males and females) beta-Ionone gave no indication of a mutagenic effect in bacteria or a clastogenic potential in an *in vivo* mouse micronucleus test. Therefore, there is no indication of a genotoxic potential *in vivo*. No studies that would be considered adequate for the evaluation of carcinogenic potential were available. A short-term screening experiment investigating a tumor-promoting potential on mouse skin did not indicate such an effect at a low test concentration. In a well-conducted 90 days study in rats according to OECD TG 408 with administration of the test substance in the diet, beta-ionone did not have the potential to damage the reproductive organs at least up to the highest tested concentration of 10,000 ppm (720 and 801 mg/kg bw/day for males and females).
OECD SIDS Initial Assessment Report for SIAM 20
For ionones and rose ketones, when used as fragrance ingredients:

- Ionones have low to moderate oral toxicity (LD50 values of 1.5 g to >5 g/kg body weight). In acute dermal toxicity studies, LD50 values are greater than 2 or 5 g/kg body weight (the limit doses commonly used in LD50 assays).
- No systemic toxicity was observed in uncomplicated subchronic oral or dermal 90-day toxicity studies in rats. It is concluded that these materials administered by the dermal route have a systemic NOAEL value of 50 mg/kg/day. They have an oral NOAEL value of 10 mg/kg body weight.
- Under intended conditions of use as fragrance ingredients, they do not have significant genotoxic, reproductive or developmental potential.
- The ionones at concentrations likely to be encountered by humans through their use as fragrance ingredients are non-irritating, and the rose ketones have limited irritation potential in sensitive subjects.
- The ionones are considered to be without significant skin sensitization potential, while the rose ketones are sensitizers when present at concentrations in excess of 0.2% (based on human data). IFRA (2007) has established Standards on the methyl ionones and the rose ketones using a Quantitative Risk Assessment (QRA) for dermal sensitization
- Use of the ionones and rose ketones in fragrances produces low levels of exposure relative to doses that elicit adverse dermal or systemic effects in laboratory animals exposed via dermal or oral routes. The estimate for maximum systemic exposure of humans using cosmetic products containing ionones or rose ketones ranges from 0.0002 to 0.331 mg/kg/day. If the estimate of 100% absorption is used and using the NOAEL of 10 mg/kg body weight/day, a margin of safety for systemic exposure of humans to the individual ionones in cosmetic products can be calculated to range from 30 to 50,000 times the maximum daily exposure.
- The limited metabolic data on ionones demonstrate biotransformation pathways involving combinations of hydroxylation/oxygenation, reduction, oxidation, and conjugation. Metabolism of the majority of this class of compounds is not likely to increase the toxicity of parent compounds. Those that could undergo epoxidation have not been subjected to subchronic testing and are considered to be inadequately characterized for the purposes of human health safety assessment

The Research Institute for Fragrance Materials (RIFM) Expert Panel
A member or analogue of a group of alicyclic substance generally regarded as safe (GRAS) .
The majority of alicyclic substances used as flavour ingredients are mono- and bicyclic terpenes which occur naturally in a wide variety of foods. Alicyclic compounds have one or more all-carbon rings which may be either saturated or unsaturated, but do not have aromatic character; alicyclic compounds may have one or more aliphatic side chains attached.
With the exception of pulegone, alicyclic substances exhibit very low oral acute toxicity (i.e. LD50 > 1000 mg/kg). Rodent LD50 values in the range from 1000 to more than 5000 mg/kg have been reported for 83 of the 1199 alicyclic- substances in this group The majority of these LD50 values are greater than 2000 mg/kg.
In most of the reported subchronic studies, no adverse effects were observed at any dose level. In studies that showed adverse effects (e.g. studies for alpha- and beta ionone and iso-bornyl acetate), NOAELs were in the range from 15 mg/kg/day to 500 mg/kg/day. The dose levels that resulted in no adverse effects for a parent or representative substance was at least 1000 times the total daily per capita intake, as flavour ingredients, for all members of this group..
The metabolic options available to alicyclic substances increase with an increase in the number and types of functional groups and ring substituents in the molecule. If a primary alcohol, aldehyde or carboxylic acid function is present on an alkyl side-chain, the substance may undergo beta-oxidation and cleavage. If the number of carbons in the side-chain is even,beta

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	oxidation may lead to cleavage of the alicyclic ring. If the number of carbons in the side-chain is even, beta-oxidation may lead to cleavage of the alicyclic ring. Alicyclic terpenoid primary alcohols which contain alkyl ring substituents generally oxidize to the corresponding carboxylic acid, conjugate with glucuronic acid, and are excreted. Terpenoid aldehydes also undergo oxidation to the corresponding carboxylic acid or, to a lesser extent, reduction to the corresponding alcohol with subsequent conjugation and excretion.If the substance has an endocyclic alkene function and is excreted into the bile, intestinal microflora may promote hydrogenation of the double bond. Excretion metabolites, therefore, may include conjugates of the reduced form of the alcohol or acid. As with acyclic substances, simple, unsubstituted,.alicyclic secondary alcohols and ketones are readily interconverted by oxidation-reduction reactions. For low molecular weight, polar alicyclic substances the ketone is stereoselectively reduced by cytosolic carbonyl reductases to yield the secondary alcohol which is conjugated primarily with glucuronic acid. The resuling conjugate may be excreted in the faeces or, more importantly, enter enterohepatic circulation and be excreted in the urine. For higher molecular weight, more lipophilic substances or those with sterically hindered functional groups, oxidation of a ring position by non-specific cytochrome P-450 mixed-function oxidases may compete with reduction of the ketone function or oxidation of the alcohol function. If the alicyclic alcohol or ketone contains an endocyclic double bond, oxidation or hydrogenation of the alkene may lead to additional metabolites. If a secondary alcohol or ketone function is located on a ring containing alkyl substituents, as in simple terpenoid derivatives, oxidation of the alkyl substituents competes with oxidation-reduction reactions of the alcohol or ketone function. If the substance contains allylic or tertiary hydrogens, the rate of oxidation increases often leading to polyoxygenated metabolites. Substances exhibiting greater lipophilicity may undergo oxidation of the secondary alcohol function to the corresponding ketone in addition to oxidation of alkyl substituents If the functional group is on an alkyl side-chain, as in the ionone derivatives, the ketone may be reduced to the corresponding alcohol. In addition, oxidation of activated ring positions may also occur. Tertiary alcohol functions are relatively stable in vivo and eventually are excreted as the glucuronic acid conjugates. Ring alkyl substituents of tertiary alcohols are generally oxidized to diols and hydroxyacids, similar to that of secondary alcohols and ketones. Tertiary alcohols with ring unsaturation would yield products of hydrogenation or oxidation of the alkene. Flavor and Extract Manufacturers' Association (FEMA)
BETA-CITRONELLOL	With few exceptions * (see below) there are no safety concerns regarding certain cyclic and non-cyclic terpene alcohols **, as fragrance ingredients, under the present declared levels of use and exposure for the following reasons · The non-cyclic and cyclic terpene alcohols have a low order of acute toxicity · No significant toxicity was observed in repeated dose toxicity tests; it is concluded that these materials have dermal and oral NOAELs of 50 mg/kg body weight/day or greater. · These materials were inactive in mutagenicity and genotoxicity tests. · Based on data on metabolism it is concluded that members of this category exhibit similar chemical and biochemical fate. · Although there is some indication for the production of reactive metabolites by some materials, these metabolites appear to be efficiently detoxicated and not expected to result in overt toxicity. There is no indication for the production of persistent metabolites. · The results from materials studied to date are indicative of the group and there are no grounds for environmental concern with respect to cyclic and non-cyclic terpene alcohol compounds as currently used in fragrance compounds. · Human dermatological studies show that, at current use levels, these materials are practically non-irritating. · The sensitization potential is generally low. · The margin of safety is generally greater than 100 times the maximum daily exposure. Sufficient data are available from farnesol, linalool, menthol and a-terpineol, i.e., compounds that contain all key structural elements and potential sites of metabolism of all other members in the group, to demonstrate that the non-cyclic and cyclic terpenes share common metabolic pathways. In most cases, metabolism yields innocuous metabolites. Some materials, however, may generate alpha, b-unsaturated compounds or be oxidized to hydroperoxides. Such compounds have the capacity to participate in a range of nucleophilic and electrophilic addition reactions with biological material. * Safety concerns exist for:the following substances for the following reasons. · 6,7-Dihydrogeraniol, hydroabietyl alcohol and 6-isopropyl-2-decahydro-naphthalenol are potent skin sensitizers. These materials are prohibited for use in fragrance materials by IFRA Standards. · Farnesol is a weak sensitizer. Its use in fragrance materials is therefore restricted by IFRA Standards. · Sclareol and linalool may contain impurities and/or oxidation products that are strong sensitizers. For use in fragrance materials,these compounds must comply with the purity criteria stated in their IFRA Standards. · 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 The Research Institute for Fragrance Materials (RIFM) Expert Panel For terpenoid primary alcohols and related esters This family includes includes three terpenoid acyclic aliphatic primary alcohols, citronellol, geraniol, and nerol. The category also includes a mixture of terpenoid esters and alcohols called acetylated myrcene. Geranyl acetate and neryl acetate are the principal products formed when myrcene is acetylated. Thus, the mixture is commonly recognised as acetylated myrcene. The four substances are grouped together because of their close structural relationships and the resulting similarities of their physiochemical and toxicological properties. Citronellol, geraniol, nerol, and geranyl acetate are currently recognized by the U.S. Food and Drug Administration (FDA) as GRAS (“generally regarded as safe”) for their intended use as flavouring substances In nature, terpenes are produced by the isoprene pathway that is an integral part of normal plant and animal biosynthesis. Oxygenated terpene substances {e.g., geraniol, nerol, citronellol, citral (a mixture of aldehydes, geranial and neral), and geranyl acetate} are therefore, ubiquitous in the plant kingdom Acetylated myrcene (geranyl and neryl acetate), being mainly a mixture of esters, is expected to be somewhat less polar and therefore less water soluble than the three terpenoid alcohols. It is however, expected to be rapidly hydrolysed *in vivo* to yield nerol, geraniol, and acetic acid . Similar hydrolysis also occurs in the environment albeit at a somewhat slower rate. Terpenoid

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alcohols formed in the gastrointestinal tract, as a result of hydrolysis are rapidly absorbed.
Following hydrolysis, geraniol, nerol, and citronellol undergo a complex pattern of alcohol oxidation, *omega*-oxidation, hydration, selective hydrogenation and subsequent conjugation to form oxygenated polar metabolites, which are rapidly excreted primarily in the urine of animals. Alternately, the corresponding carboxylic acids formed by oxidation of the alcohol function may enter the *beta*-oxidation pathway and eventually undergo cleavage to yield shorter chain carboxylic acids that are completely metabolised to carbon dioxide. Geraniol, related terpenoid alcohols (citronellol and nerol), and the related terpene aldehydes (geranial and neral) exhibit similar pathways of metabolic detoxication in animals.
In rats and mice, a mixture of geranial and neral, commonly recognised as citral, undergoes rapid absorption from the gastrointestinal tract and distribution throughout the body.
Genotoxicity: *In vitro* genotoxicity assays available for citronellol, geraniol, citral (geranial and neral mixture) and acetylated myrcene (geranyl acetate and neryl acetate mixture) demonstrate that these substances have a low genotoxic potential. No evidence of mutagenicity was reported in an Ames assay with citronellol metabolites. In two chromosomal aberration assays with geraniol and a geranial/neral mixture, there was no evidence of increased incidence of chromosomal aberrations when Chinese hamster lung fibroblasts were incubated with 125 ug/plate of geraniol or 30 ug/plate of the geranial/neral mixture, respectively. Nerol, being a geometrical isomer of geraniol would also be expected to be negative. The acetates of nerol and geraniol, the principal constituents of acetylated myrcene, which will hydrolyse to nerol and geraniol, have also been tested and found to be negative in Ames assays at concentrations up to 20,000 ug/plate.
In vivo: Tests on citronellol and acetylated myrcene (geranyl acetate) confirm the lack of genotoxic potential. A mixture of geranyl acetate (79%) and citronellyl acetate (21%) showed no evidence of increased micronuclei in a standardized mouse (B6C3F1 strain) micronucleus assay at dose levels up to and including 1800 mg/kg bw and there was no evidence of unscheduled DNA synthesis when the geranyl acetate/citronellyl acetate mixture was given orally to Fisher F344 rats. Since these esters hydrolyse to geraniol and citronellol in rodents, these results apply directly to geraniol and citronellol.
Repeat dose toxicity:
Short term: Citronellol, as an equal mixture with the structurally similar material linalool, administered to rats at 100 mg/kg/day for 12 weeks, resulted in no adverse effects. Geraniol, in combination with a structural isomer, was administered to groups of rats (5/sex/group) in the diet at concentrations of 10,000 ppm for 16 weeks or 1000 ppm for 27 weeks. No adverse effects were reported in either study. Likewise, no adverse effects were observed when rats were maintained on a diet calculated to provide an estimated average daily intake of greater than 200 mg/kg bw/day of citral, a mixture of geranial and neral, for 91 days.
Long-term studies: Citronellol, geraniol and nerol and the principal hydrolysis products of acetylated myrcene (geranyl acetate) were all included as structural similar acyclic terpenes in a QSAR study by molecular orbital calculations for prediction of their potential toxicity/carcinogenicity. None of the substances in this group were predicted to have significant toxicity and/or carcinogenicity potential. This conclusion is supported by the results of a 2 year bioassay on a mixture of acetate esters of geraniol and citronellol that showed no toxic or carcinogenic effects at dose levels up to 2000 mg/kg bw/day in rats and 1000 mg/kg bw/day in mice.
Reproductive toxicity: A mixture of the aldehydes, geranial and neral, has been subjected to an oral 2-generation reproductive study in rats. There were no reproductive effects at the maternal NOAEL of 50 mg/kg/day and a foetal/pup NOAEL of 160 mg/kg bw/day. At a maternally toxic level of 500 mg/kg bw/day, the only effect reported was a slightly decreased pup weight. Given that other studies show the mixture of aldehydes exhibits a higher level of toxicity than the corresponding alcohols geraniol and nerol , data on reproductive and developmental toxicity for the aldehydes may be used to conservatively estimate reproductive toxicity for the corresponding alcohols.
Developmental toxicity: In a developmental/reproduction screening study, rats were administered the acetal formed from citral (geranial and neral mixture) and ethanol. The acetal will readily hydrolyse to citral. The NOAELs for maternal toxicity and developmental toxicity were reported to be 125 and 250 mg/kg bw/day, respectively.
A geranial/neral mixture has been subjected to an oral foetotoxicity study in rats an NOAEL for maternal and developmental toxicities were reported to be 60 mg/kg bw/day
In an inhalation developmental study in rats using a geranial/ neral mixture A NOAEL for maternal toxicity was reported to be 35 ppm. There were some slight foetotoxic effects at the maternally toxic level of 85 ppm (as a vapor/aerosol)
Current opinion holds that there are no safety concerns regarding the unsaturated branched chain non-cyclic alcohols, as fragrance ingredients, under the present declared levels of use and exposure; use of these materials at higher maximum dermal levels or higher systemic exposure levels requires re-evaluation. This opinion was based on the following reasons:

- No evidence or only minimal evidence of skin irritation in humans was associated with current levels of use at 2–30% for individual compounds considered.
- Sensitizing 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.
- The compounds have a low order of acute toxicity.
- The branched chain, unsaturated alcohols tested were of low systemic toxicity after repeated application. Changes indicative of enzyme induction in the liver (liver enlargement) and a2u nephropathy in male rats have been observed at doses from >=200 mg/kg body weight/day.
- There was little or no indication of specific adverse effects in relation to fertility and developmental toxicity.
- Apart from the double bonds, especially those in conjugation with primary and secondary alcohol groups, the substances of this group evaluation do not posses further reactive structures that may give rise to genotoxic potential.
- Valid data on carcinogenicity of the compounds or for closely structurally related substances are not available, but in view of the negative mutagenicity tests so far obtained, they are not of primary concern

The dermal LD50 values in rats, rabbits and guinea pigs are greater than 2000 mg/kg body weight and even greater than 5000 mg/kg body weight in some cases, indicating that these compounds are of low acute toxicity or are practically non-toxic via the dermal route.
The oral LD50 values in rats and mice are is generally greater than 2000 mg/kg body weight.
The most reported clinical sign was lethargy after oral or dermal application, diarrhea and gastrointestinal tract irritation after oral application, and irritation of the skin after dermal application.

The common characteristic structural elements, of this group, are one hydroxyl group per molecule, a C4 to C16 carbon chain with one or several methyl or ethyl side chains and up to four non-conjugated double bonds.

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	Due to their structural similarity, these alcohols also share common metabolic pathways. As metabolism is crucial for toxicokinetics and toxicity, these alcohols are expected to have the same target organs (liver and kidney) as was shown for selected compounds. . As the data base for these alcohols is limited, additional data on pharmacokinetics, metabolism, genotoxicity and systemic toxicity of the structurally related non-cyclic unsaturated branched alcohols, citronellol, dehydrolinalool, 6,7-dihydrolinalool, farnesol, geraniol, linalool, nerol, and nerolidol (cis and isomer unspecified), from an evaluation of terpene alcohols. In most cases, metabolism yields innocuous metabolites. Some materials, however, may generate alpha, beta-unsaturated compounds, e.g. aldehydes formed from primary allylic alcohols, or undergo oxidation to hydroperoxides. Such compounds can take part in a range of nucleophilic and electrophilic addition reactions with biological material. The presence of a double bond may give rise to the metabolic formation of reactive and genotoxic epoxides although Ames tests did not indicate mutagenic activity, which would be expected if epoxides were formed in appreciable amounts. The Research Institute for Fragrance Materials (RIFM) Expert Panel For alkyl alcohols C6-13: This group of products are very similar in terms of physicochemical and toxicological properties. Interpolation of data can be used to assess the alkyl alcohols for which data is not available. **Acute toxicity:** All of these alcohols have a low order of toxicity in rats via the oral route. The LD50 for C6-branched and linear alcohols were >3700 mg/kg; LD50s for the C6-8, C7-9, C8-10, C9-11 and C11-14 branched alkyl alcohols were all >2000 mg/kg. These alcohols have a low order of toxicity via the dermal route. Dermal LD50s were greater than 2600 mg/kg. **Subchronic toxicity**: Repeat dose studies indicate these alcohols have a low order of subchronic toxicity by both the oral and dermal route. Further they demonstrate that these alcohols display a consistent degree of subchronic toxicity by these routes **Developmental toxicity:** Studies demonstrate that the alcohols are not selective developmental toxicants by either the oral or inhalation route of exposure. Inhalation of alkyl alcohols C6-13 is a primary concern during industrial use, particularly for lower molecular weight alcohols. Collectively the weight of evidence demonstrates that these alcohols have a low order of maternal toxicity and do not induce signs of developmental toxicity until maternal toxicity is observed. The NOAELs for inhalation reflect the maximum achievable vapour concentration. **Reproductive toxicity:** Developmental toxicity studies for several of these alcohols, conducted by the oral route, produce consistent results and demonstrate that these substances do not affect reproductive parameters. Although a slight increase in resorptions was observed in several studies, this occurred only in the highest dose group and in the presence of overt maternal toxicity. **Genotoxicity:** The weight of evidence from existing data supports the conclusion that these materials are not genotoxic. Further data to support this assessment comes from a series of alkyl acetates C6-13. Alkly acetates arre produced from alkyl alcohols and undergo metabolism by esterases to produce acetic acid and the corresponding alkyl alcohol. There is no evidence for genotoxicity with these compounds in a variety of strains of S. typhimurium in the presence or absence of metabolic activation. C6, C6-8, C7-9 and C11-14 alkyl acetates produced negative results in the Ames test. Based on data for structurally similar substances these alcohols are not expected to be clastogenic. Alkyl acetates can also be used to predict clastogenic potential of alkyl alcohols. Although there is evidence of cytotoxicity at extremely high doses, no clastogenic activity was seen in a homologous family of alkyl acetates. **Metabolism:**:Alkyl alcohols are broken down, in the body, by mitochondrial beta-oxidation or by cytochrome P450 omega and and omega-minus oxidation. The alcohol undergoes various oxidative steps to yield other alcohols, ketones, aldehydes, carboxylic acids and carbon dioxide, Data for monohydric, aliphatic alcohols show a systematic variation according to molecular weight in a manner similar to other homologous series. The body handles aliphatioc hydrocarbons in a similar manner via oxidative conversion to alcohols, ketones, and eventual elimination as carbon dioxide and carboxylic acids. The undegraded alcohols can be conjugated either directly or as a metabolite with glucuronic acid, sulfuric acid or glycine and are reapidly excreted. Intermediate aldehydes may be reactive and bind with DNA and/ or proteins.
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.
AMYL SALICYLATE	*Vigon SDS ** REACh Dossier For certain benzyl derivatives: All members of this group (benzyl, benzoate and 2-hydroxybenzoate (salicylate) esters) contain a benzene ring bonded directly to an oxygenated functional group (aldehyde or ester) that is hydrolysed and/or oxidised to a benzoic acid derivative. As a stable animal metabolite, benzoic acid derivatives are efficiently excreted primarily in the urine. These reaction pathways have been reported in both aquatic and terrestrial species. The similarity of their toxicologic properties is a reflection their participation in these common metabolic pathways. In general, members of this group are rapidly absorbed through the gastrointestinal tract, metabolised primarily in the liver, and excreted in the urine either unchanged or as conjugates of benzoic acid derivatives At high doses, conjugation pathways (*e.g.,* glycine) may be saturated; in which case, free benzoic acid is excreted unchanged. Absorption, distribution and excretion studies have been conducted several members of this group and structural relatives. These substances exhibit remarkably similar patterns of pharmacokinetics and metabolism. The benzyl, benzoate, and 2-hydroxybenzoate (salicylate) esters which comprise this category are hydrolysed to the corresponding alcohols and carboxylic acids. The benzyl alcohol and benzaldehyde derivatives are oxidised to the corresponding benzoic acid derivatives that are subsequently excreted unchanged or as glycine or glucuronic acid conjugates. If methoxy or phenolic functional groups are present on the benzene ring, additional minor metabolic options become available. *O*-demethylation yields the corresponding phenol that is subsequently excreted as the glucuronic acid or sulfate conjugates. At high dose levels, gut microflora may act to produce minor amounts of reduction metabolites. **Acute toxicity**: Oral LD50 values ranged from 887 to greater than 5,000 mg/kg bw demonstrating the low to moderate toxicity of these compounds. **Repeat dose toxicity:** Overall, numerous repeat-dose studies using various routes of exposure have been conducted in different animal species with members of this chemical category or their close structural relatives. It is important to note that all the benzyl derivatives in this category are eventually metabolised to a common metabolite, benzoic acid, and are rapidly excreted in the

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	urine as benzoic acid or as its glycine, sulfate, or glucuronic acid conjugate. For this reason, the repeat-dose studies currently available provide adequate support for the safety of the benzyl derivatives. Moreover, the levels at which no adverse effects were reported were sufficiently high to accommodate any potential differences among the members of the category. **Reproductive toxicity:** Several reproductive toxicity studies have been conducted with representatives of this group and produced no evidence of reproductive toxicity As with the repeat-dose studies, the benzyl derivatives generally follow the similar metabolic pathways and the studies conducted provide an adequate database for this endpoint. In addition, the dose levels tested provide margins of safety large enough to accommodate any differences among the group. **Developmental toxicity:** Representative substances from this group were tested for developmental toxicity with uniform results, and indicated no teratogenic potential in the absence of maternal toxicity. Again, the representative substances undergo similar metabolism to the entire benzyl derivative group and therefore, provide an adequate representation for this endpoint. **Genetic toxicity:** Overall, *in vitro* and *in vivo* genotoxicity studies have been conducted with substances representing the structural characteristics of the benzyl category. The results of these studies were predominantly negative demonstrating a low order of genotoxic potential.. Limited positive and/or equivocal findings have been reported for 3 aldehydes and benzyl acetate, but, in most cases, other studies of the same endpoint with same test substance show no activity. Most importantly, *in vivo* studies on benzaldehyde derivatives and closely related benzyl esters have all yielded negative results. These negative *in vivo* genotoxicity assays are supported by the lack of tumorigenicity in chronic animal studies with representatives of this group. Data available for more than 100 *in vitro* genotoxicity assays for 9 members of the category and five metabolic precursors or metabolites of benzyl derivatives indicate a low genotoxic potential for members of this chemical category Equivocal results have been reported mainly for aromatic aldehydes in the MLA and ABS assays. The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.
12-MUSK DECENONE	NOAEL (rat) 4-week oral gavage: 1000 mg/kg/day NOAEL (rat) oral repeateddose toxicity:1000 mg/kg/day Not mutagenic; reverse mutation assay S. typhimurium * Not clastogenic: chromosome aberration - human lymphocytes * Not genotoxic in mouse lymphoma assay * Not sensitising to Guinea pig skin * No evidence of sensitising effects in human volunteers in insult test repeated patch * * NICNAS Full Public Report Habanolide Current opinion holds that there are no safety concerns for the Macrocyclic Lactone and Lactide (MLs, natural and synthetic musks) derivatives at reported levels of use and exposure as fragrance ingredients. · The MLs had low acute toxicity and no significant toxicity in repeat dose oral or dermal toxicity studies. Effects on blood biochemistry were reversible after 2 weeks of no treatment · Human dermatological studies show MLs are generally not irritating after one application. Minor irritation was observed in a few individuals following multiple applications.For high end users, calculated maximum dermal exposures vary from 0.47% to 11.15%; systemic exposures vary from 0.0008 to 0.25 mg/kg/day. . · In animal studies, the MLs are not sensitizers at lower exposures from consumer products. Eleven ML materials were evaluated for human sensitization. Of these, only ethylene brassylate showed evidence of sensitization in 2/27 studies (sensitization frequency 4/2059 total). · At rates consistent with reported levels for current human exposure, no phototoxicity or photosensitization was observed. · No mutagenic or genotoxic activity in bacteria and mammalian cell line assays was observed. The common structural element of the ML group of fragrance ingredients is a mono- or diester-lactone group, R–C (=O) O–R' , contained within a macrocyclic ring of C14 to C16 carbon chain length. . The naturally occurring macrocyclic lactones are generally derived from various plant, rather than animal, sources The macrocyclic lactone fragrance ingredients are generally lipophilic and log Kow increases with increasing ring size. log Kow values range from 6.7 for the mono C16 saturated lactone oxacycloheptadec-10-ene-2-one (CAS RN 28645-51-4) to 3.65 for the saturated C14 diester ethylene dodecanedioate (CAS RN 54982-83.-1). As a class, the macrocyclic lactone fragrance ingredients have a low volatility and are not appreciably water soluble. The initial and primary metabolism would be hydrolysis of the lactone functionality to generate the corresponding long chain open carboxylic acid and alcohol which should undergo fatty acid type beta-oxidation. It is believed that all the materials in this group have similar metabolism and are detoxified in the same manner. Their toxicological profiles would, then, be similar The Research Institute for Fragrance Materials (RIFM) Expert Panel
D-PANTHENOL	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
CITRIC ACID	for citric acid (and its inorganic citrate salts) Based on many experimental data in animals and on human experience, citric acid is of low acute toxicity. The NOAEL for repeated dose toxicity for rats is 1200 mg/kg/d. The major, reversible (sub)chronic toxic effects seem to be limited to changes in blood chemistry and metal absorption/excretion kinetics. Citric acid is not suspected of being a carcinogen nor a reprotoxic or teratogenic agent. The NOAEL for reproductive toxicity for rats is 2500 mg/kg/d. Further, it is not mutagenic *in vitro* and *in vivo*. Also, the sensitising potential is seen as low. In contrast, irritation, in particular of the eyes but also of the respiratory pathways and the skin, is the major toxicological hazard presented by citric acid The CIR Expert Panel (Panel) assessed the safety of citric acid, 12 inorganic citrate salts, and 20 alkyl citrate esters as used in cosmetics, concluding that these ingredients are safe in the present practices of use and concentration. Citric acid is reported to function as a pH adjuster, chelating agent, or fragrance ingredient. Some of the salts are also reported to function as chelating agents, and a number of the citrates are reported to function as skin-conditioning agents but other functions are also reported. The Panel reviewed available animal and clinical data, but because citric acid, calcium citrate, ferric citrate, manganese citrate, potassium citrate, sodium citrate, diammonium citrate, isopropyl citrate, stearyl citrate, and triethyl citrate are generally recognized as safe direct food additives, dermal exposure was the focus for these ingredients in this cosmetic ingredient safety assessment.

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ALOES, EXTRACT	Aloe barbadensis Mill., extract **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. Whole leaf extract of Aloe vera was tested for carcinogenicity after oral administration in one 2-year study in mice, and one 2-year study in rats. In male and female rats, drinking-water containing whole leaf extract of Aloe vera caused significantly increased incidences of adenoma of the large intestine (colon and caecum) and carcinoma of the large intestine (colon and caecum), tumours rarely developed spontaneously in rats. In the 2-year study in mice, there was no significantly increased incidence of any type of tumours in males or females given drinking-water containing whole leaf extract of Aloe vera. In a study of photo-co-carcinogenesis with simulated sunlight, four articles were studied by skin application in hairless mice: three test articles containing Aloe vera that included gel, whole leaf extract, and decolourised whole leaf extract; and an aloe-emodin preparation. Almost all mice exposed to simulated sunlight developed skin neoplasms. No increase in the incidence of skin neoplasms was observed in the groups receiving any of the four test articles applied as a cream followed by simulated sunlight when compared with the group receiving control cream followed by simulated sunlight. There was a significant enhancing effect of Aloe vera gel cream or aloe-emodin cream on the photocarcinogenic activity of simulated sunlight in female mice based on an increase in the multiplicity of squamous cell papilloma, carcinoma or carcinoma in situ (combined). There was a significant enhancing effect of the whole leaf extract cream or decolourized whole leaf extract cream on the photocarcinogenic activity of simulated sunlight in both male and female mice, based on an increase in the multiplicity of squamous cell papilloma, carcinoma or carcinoma in situ (combined). Mechanistic and other relevant data The C-glycosides aloin A and aloin B, which are components of Aloe vera latex, are converted to aloe-emodin-9-anthrone by bacteria present in the gastrointestinal tract of rats and humans. Aloe-emodin-9-anthrone undergoes sequential oxidation to aloe-emodin and rhein. Preparations of Aloe vera, acemannan, and aloin A, do not display genotoxic activity in assays for mutagenesis in bacteria and/or other assays for genotoxicity. In contrast, aloe-emodin has genotoxic activity. These data suggest that the neoplastic response observed with Aloe vera is a consequence of the conversion of the anthrone C-glycosides to aloe-emodin, which by itself or in combination with other components of Aloe vera is responsible for the adenomas and carcinomas in the large intestine of rats.
BUGALUGS Baby Fresh Cologne & BETA-CITRONELLOL & COUMARIN & CITRIC ACID	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.
BUGALUGS Baby Fresh Cologne & ISOMETHYL-ALPHA-IONONE	Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. 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.
BUGALUGS Baby Fresh Cologne & ISOMETHYL-ALPHA-IONONE & BETA-CITRONELLOL & COUMARIN & AMYL SALICYLATE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and connubial contact dermatitis occur. Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes. Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits. Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of

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	the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water. Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis. Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a suffcient degree of fragrance contact allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the allergen. As a consequence, symptoms, i.e. allergic contact dermatitis, may occur upon re-exposure to the fragrance allergen(s) in question. Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or hands. It may affect fitness for work and the quality of life of the individual. Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure. **Hands:** Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema may often be complicated by sensitisation .Fragrance allergy may be a relevant problem in patients with hand eczema; perfumes are present in consumer products to which their hands are exposed. A significant relationship between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe) chronic hand eczema may not be clear. **Axillae Bilateral axillary** (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy. **Face** Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of of being fragrance allergic. **Irritant reactions (including contact urticaria):** Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this, Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported . The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested , but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen. **Pigmentary anomalies:** The term “pigmented cosmetic dermatitis” was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified.. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil. **Photo-reactions** Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon . Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. Phototoxic reactions still occur but are rare. **General/respiratory:** Fragrances are volatile and therefore, in addition to skin exposure, a perfume also exposes the eyes and naso-respiratory tract. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma . Asthma-like symptoms can be provoked by sensory mechanisms. In an epidemiological investigation, a significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.
BUGALUGS Baby Fresh Cologne & COUMARIN & AMYL SALICYLATE	Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A prehapten is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems. A prohapten is a chemical that itself is non- or low-sensitising but that is transformed into a hapten in the skin (bioactivation) usually via enzyme catalysis. It is not always possible to know whether a particular allergen that is not directly reactive acts as a prehapten or as a prohapten, or both, because air oxidation and bioactivation can often give the same product (geraniol is an example). Some chemicals might act by all three pathways. **Prohaptens** Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohaptens. In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal. The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to

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	increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin . These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohaptens can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity. **QSAR prediction:** The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptens) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptens. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation
BUGALUGS Baby Fresh Cologne & AMYL SALICYLATE	The Research Institute for Fragrance Materials (RIFM) Expert Panel study of fragrance salicylates concluded. The salicylates are well absorbed by the oral route, and oral bioavailability is assumed to be 100%. Absorption by the dermal route in humans is more limited with bioavailability in the range of 11.8-30.7%. The salicylates are expected to undergo extensive hydrolysis, primarily in the liver, to salicylic acid which is conjugated with either glycine or glucuronide and is excreted in the urine as salicyluric acid and acyl and phenolic glucuronides. The hydrolyzed side chains are metabolized by common and well-characterized metabolic pathways leading to the formation of innocuous end products. The expected metabolism of the salicylates does not present toxicological concerns. The acute dermal toxicity of the salicylates is very low, with LD50 values in rabbits reported to be greater than 5000 mg/kg body weight. The acute oral toxicity of the salicylates is moderate, with toxicity generally decreasing with increasing size of the ester R-group and with LD50‘s between 1000 and >5000 g/kg. In dermal subchronic toxicity studies, extreme doses of methyl salicylate (5 g/kg body weight/day) possibly were nephrotoxic but the data were minimal. The subchronic oral NOAEL is concluded to be 50 mg/kg body weight/day. Genetic toxicity data, for methyl salicylate, a few other salicylates and for structurally related alkyl- and alkoxy-benzyl derivatives are negative for genotoxicity. Given the metabolism of salicylate and the evidence that they are non-genotoxic, it can be concluded that the salicylates are without carcinogenic potential. The reproductive and developmental toxicity data on methyl salicylate demonstrate that high, maternally toxic doses result in a pattern of embryotoxicity and teratogenesis similar to that characterized for salicylic acid. At concentrations likely to be encountered by humans through the use of the salicylates as fragrance ingredients, these chemicals are considered to be non-irritating to the skin. The salicylates (with the exception of benzyl salicylate) in general have no or very limited skin sensitization potential. The salicylates are non-phototoxic and have no photoirritant or photoallergenic activity The use of the salicylates in fragrances produces low levels of exposure relative to doses that elicit adverse systemic effects in laboratory animals exposed by the dermal or oral route. Based on NOAEL values of 50 mg/kg body weight/day identified in the subchronic and the chronic toxicity studies , a margin of safety for systemic exposure of humans to the individual salicylates in cosmetic products, may be calculated to range from 125 to 2,500,000 (depending upon the assumption of either 12–30% or 100% bioavailability following dermal application) times the maximum daily exposure. The acute dermal toxicity of the salicylates is very low. Rabbit dermal LD50 values have been reported to be >5000 mg/kg body weight for 15 of the 16 salicylates tested, findings likely related to the limited degree of dermal absorption, the retention of salicylate in the skin, and the relatively moderate toxicity of salicylic acid itself upon systemic exposure (i.e., oral LD50 value of 891 mg/kg body weight in rats) . Overall, the acute oral toxicity of the salicylates is moderate, with toxicity generally decreasing with increasing size of the ester R-group. For the longer carbon chain salicylates, acute oral LD50 s range from 1320 to >5000 mg/kg body weight. The acute oral toxicity of the unsaturated salicylates is likewise low to moderate with rat oral LD50 s in the 3200 to >5000 mg/kg body weight range as are the acute oral toxicities of the aromatic salicylates (1300 to >5000 mg/kg body weight) The 17 compounds assessed in this report include the core salicylate moiety that upon hydrolysis yield salicylic acid and the alcohol of the corresponding alkyl, alkenyl, benzyl, phenyl, phenethyl, etc. side chain. This is consistent with information on other alkyl- and alkoxy- benzyl derivatives whereby aromatic esters are hydrolyzed in vivo by carboxylesterases, or esterases, especially the A-esterases. Potential differences in the metabolism of the individual salicylates would be related to the manner in which the hydrolyzed side chain undergoes further oxidation/reduction and/or conjugation reactions. Salicylic acid undergoes metabolism primarily in the liver. At low, non-toxic doses, approximately 80% of salicylic acid is further metabolized in the liver via conjugation with glycine and subsequent formation of salicyluric acid. For each of the salicylates, following hydrolysis to salicylic acid, the resulting side chains, hydroxylated alkyl, alkenyl, and phenyl moieties, could be expected to be further metabolized. In the case of the alcohols formed following hydrolysis. Further metabolism would result in the formation of the corresponding aldehydes and acids, with eventual degradation to CO2 by the

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	fatty acid pathway and the tricarboxylic acid cycle. The secondary alcohols formed by hydrolysis of isobutyl and isoamyl salicylate, would primarily be conjugated with glucuronic acid and excreted. They could also interconvert to the corresponding ketones. Salicylates bearing alkenyl side chains, may undergo epoxidation and subsequent hydroxylation at points of unsaturation. However, since both the alkyl and alkenyl side chains would be hydroxylated at one terminus following hydrolysis of the corresponding salicylate, a significant proportion of these hydrolysis products would be excreted in the urine precluding further metabolism and epoxidation. In the case of hydrolysis of the salicylates containing aromatic side chains, phenyl salicylate and benzyl salicylate, phenol and benzyl alcohol, respectively, would be formed. Salicylates were potent and selective inhibitors for AKR1C1 enzymes , a family of aldo-keto reductases implicated in biosynthesis, intermediary metabolism and detoxification.
ISOMETHYL-ALPHA-IONONE & AMYL SALICYLATE & ALOES, EXTRACT	No significant acute toxicological data identified in literature search.
ISOMETHYL-ALPHA-IONONE & BETA-CITRONELLOL	Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A **prehapten** is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems. In the case of prehaptens, it is possible to prevent activation outside the body to a certain extent by different measures, e.g. prevention of air exposure during handling and storage of the ingredients and the final product, and by the addition of suitable antioxidants. When antioxidants are used, care should be taken that they will not be activated themselves and thereby form new sensitisers. **Prehaptens** Most terpenes with oxidisable allylic positions can be expected to autoxidise on air exposure due to their inherent properties. Depending on the stability of the oxidation products that are formed, a difference in the sensitisation potency of the oxidised terpenes can be seen Autoxidation is a free radical chain reaction in which hydrogen atom abstraction in combination with addition of oxygen forms peroxyl radicals. The reaction shows selectivity for positions where stable radicals can be formed. So far, all fragrance substances that have been investigated with regard to the influence of autoxidation on the allergenic potential, including identification of formed oxidation products, have oxidisable allylic positions that are able to form hydroperoxides and/or hydrogen peroxide as primary oxidation products upon air exposure. Once the hydroperoxides have been formed outside the skin they form specific antigens and act as skin sensitisers. Secondary oxidation products such as aldehydes and epoxides can also be allergenic, thus further increasing the sensitisation potency of the autoxidation mixture. The process of photoactivation may also play a role, but further research is required to establish whether this activation route is currently underestimated in importance due to insufficient knowledge of the true haptens in this context. It should be noted that activation of substances via air oxidation results in various haptens that might be the same or cross-reacting with other haptens (allergens). The main allergens after air oxidation of linalool and linalyl acetate are the hydroperoxides. If linalyl acetate is chemically hydrolysed outside the skin it can thereafter be oxidised to the same haptens as seen for linalool. A corresponding example is citronellol and citronellyl acetate. In clincal studies, concomitant reactions to oxidised linalool and oxidised linalyl acetate have been observed. Whether these reactions depend on cross-reactivity or are due to exposure to both fragrance substances cannot be elucidated as both have an allergenic effect themselves. Linalool and linalyl acetate are the main components of lavender oil. They autoxidise on air exposure also when present in the essential oil, and form the same oxidation products found in previous studies of the pure synthetic terpenes. Experimental sensitisation studies showed that air exposure of lavender oil increased the sensitisation potency. Patch test results in dermatitis patients showed a connection between positive reactions to oxidised linalool, linalyl acetate and lavender oil. **Prohaptens** Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohaptens. In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal. The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin . These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohaptens can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity. **QSAR prediction:** The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptens) is more complex compared to that of

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	compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptens. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation.
D-PANTHENOL & CITRIC ACID	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

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

BUGALUGS Baby Fresh Cologne

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

ethanol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	275mg/l	2
EC50	48h	Crustacea	2mg/l	4
EC50	96h	Algae or other aquatic plants	<0.001mg/L	4
LC50	96h	Fish	42mg/l	4
EC50(ECx)	96h	Algae or other aquatic plants	<0.001mg/L	4

isomethyl-alpha-ionone

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>20mg/l	2
EC50	48h	Crustacea	9mg/l	2
LC50	96h	Fish	6.8mg/l	2
NOEC(ECx)	48h	Crustacea	1mg/l	2

beta-citronellol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	2.4mg/l	2
EC50	48h	Crustacea	17.48mg/l	2
LC50	96h	Fish	14.66mg/l	2
EC20(ECx)	72h	Algae or other aquatic plants	1.1mg/l	2

coumarin

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	96h	Algae or other aquatic plants	1.452mg/l	2
EC50	48h	Crustacea	8.012mg/l	2
NOEC(ECx)	1440h	Fish	0.119mg/l	2
LC50	96h	Fish	1.324mg/l	2

amyl salicylate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.77mg/l	Not Available
EC50	48h	Crustacea	0.88mg/l	Not Available
EC50	96h	Algae or other aquatic plants	0.65mg/l	2

Continued...

	EC50(ECx)	72h	Algae or other aquatic plants	0.77mg/l	Not Available
	LC50	96h	Fish	1.34mg/l	Not Available

12-musk decenone

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

d-panthenol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>100mg/l	2
EC50	48h	Crustacea	>100mg/l	2
NOEC(ECx)	48h	Crustacea	100mg/l	2
LC50	96h	Fish	>1000mg/l	2

citric acid

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	990mg/l	2
EC50	48h	Crustacea	>50mg/l	2
LC50	96h	Fish	>100mg/l	2
EC50(ECx)	48h	Crustacea	>50mg/l	2

Aloes, extract

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

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*

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.
For Ethanol:
log Kow: -0.31 to -0.32;
Koc 1: Estimated BCF= 3;
Half-life (hr) air: 144;
Half-life (hr) H2O surface water: 144;
Henry's atm m3 /mol: 6.29E-06;
BOD 5 if unstated: 0.93-1.67,63%
COD: 1.99-2.11,97%;
ThOD : 2.1.
Environmental Fate: Terrestrial - Ethanol quickly biodegrades in soil but may leach into ground water; most is lost by evaporation. Ethanol is expected to have very high mobility in soil. Volatilization of ethanol from moist soil surfaces is expected to be an important fate process. The potential for volatilization of ethanol from dry soil surfaces may exist. Biodegradation is expected to be an important fate process for ethanol based on half-lives on the order of a few days for ethanol in sandy soil/groundwater microcosms.
Atmospheric Fate: Ethanol is expected to exist solely as a vapour in the ambient atmosphere. Vapour-phase ethanol is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 5 days. Ethanol readily degraded by reaction with photochemically produced hydroxy radicals; release into air will result in photodegradation and wet deposition.
Aquatic Fate: When released into water ethanol readily evaporates and is biodegradable. Ethanol is not expected to adsorb to suspended solids and sediment. Volatilization from water surfaces is expected and volatilization half-lives for a model river and model lake are 3 and 39 days, respectively. Bioconcentration in aquatic organisms is considered to be low. Hydrolysis and photolysis in sunlit surface waters is not expected to be an important environmental fate process for ethanol and is unlikely to be persistent in aquatic environments.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ethanol	LOW (Half-life = 2.17 days)	LOW (Half-life = 5.08 days)
isomethyl-alpha-ionone	HIGH	HIGH
beta-citronellol	LOW	LOW
coumarin	LOW	LOW
amyl salicylate	LOW	LOW
d-panthenol	LOW	LOW

Continued...

Ingredient	Persistence: Water/Soil	Persistence: Air
citric acid	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
ethanol	LOW (LogKOW = -0.31)
isomethyl-alpha-ionone	HIGH (LogKOW = 4.8411)
beta-citronellol	MEDIUM (LogKOW = 3.91)
coumarin	LOW (LogKOW = 1.39)
amyl salicylate	HIGH (LogKOW = 4.568)
d-panthenol	LOW (LogKOW = -1.9222)
citric acid	LOW (LogKOW = -1.64)

Mobility in soil

Ingredient	Mobility
ethanol	HIGH (KOC = 1)
isomethyl-alpha-ionone	LOW (KOC = 1034)
beta-citronellol	LOW (KOC = 70.79)
coumarin	LOW (KOC = 146.1)
amyl salicylate	LOW (KOC = 1483)
d-panthenol	LOW (KOC = 10)
citric acid	LOW (KOC = 10)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. 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. ▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▸ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material). ▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 Transport information

Labels Required

Continued...

	
Marine Pollutant	NO
HAZCHEM	•2Y

Land transport (ADG)

UN number or ID number	1170
UN proper shipping name	ETHANOL (ETHYL ALCOHOL) or ETHANOL SOLUTION (ETHYL ALCOHOL SOLUTION)
Transport hazard class(es)	Class: 3 Subsidiary risk: Not Applicable
Packing group	III
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: 144 223 Limited quantity: 5 L

Air transport (ICAO-IATA / DGR)

UN number	1170
UN proper shipping name	Ethanol or Ethanol. solution
Transport hazard class(es)	ICAO/IATA Class: 3 ICAO / IATA Subrisk: Not Applicable ERG Code: 3L
Packing group	III
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: A3 A58 A180 Cargo Only Packing Instructions: 366 Cargo Only Maximum Qty / Pack: 220 L Passenger and Cargo Packing Instructions: 355 Passenger and Cargo Maximum Qty / Pack: 60 L Passenger and Cargo Limited Quantity Packing Instructions: Y344 Passenger and Cargo Limited Maximum Qty / Pack: 10 L

Sea transport (IMDG-Code / GGVSee)

UN number	1170
UN proper shipping name	ETHANOL (ETHYL ALCOHOL) or ETHANOL SOLUTION (ETHYL ALCOHOL SOLUTION)
Transport hazard class(es)	IMDG Class: 3 IMDG Subrisk: Not Applicable
Packing group	III
Environmental hazard	Not Applicable
Special precautions for user	EMS Number: F-E, S-D Special provisions: 144 223 Limited Quantities: 5 L

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

Not Applicable

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

Continued...

Product name	Group
ethanol	Not Available
isomethyl-alpha-ionone	Not Available
beta-citronellol	Not Available
coumarin	Not Available
amyl salicylate	Not Available
12-musk decenone	Not Available
d-panthenol	Not Available
citric acid	Not Available
Aloes, extract	Not Available

Transport in bulk in accordance with the IGC Code

Product name	Ship Type
ethanol	Not Available
isomethyl-alpha-ionone	Not Available
beta-citronellol	Not Available
coumarin	Not Available
amyl salicylate	Not Available
12-musk decenone	Not Available
d-panthenol	Not Available
citric acid	Not Available
Aloes, extract	Not Available

SECTION 15 Regulatory information

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

ethanol is found on the following regulatory lists

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

isomethyl-alpha-ionone is found on the following regulatory lists

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

beta-citronellol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

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

amyl salicylate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

12-musk decenone is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

d-panthenol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

citric acid is found on the following regulatory lists

Continued...

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

Aloes, extract is found on the following regulatory lists

Australia Industrial Chemicals Introduction Scheme Comparable Chemicals Table

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

Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (ethanol; isomethyl-alpha-ionone; beta-citronellol; coumarin; amyl salicylate; 12-musk decenone; d-panthenol; citric acid; Aloes, extract)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (12-musk decenone)
Japan - ENCS	No (Aloes, extract)
Korea - KECI	No (Aloes, extract)
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	No (12-musk decenone; Aloes, extract)
Taiwan - TCSI	Yes
Mexico - INSQ	No (amyl salicylate; 12-musk decenone)
Vietnam - NCI	Yes
Russia - FBEPH	No (12-musk decenone; Aloes, extract)
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	10/07/2023
Initial Date	10/07/2023

Other information

Ingredients with multiple cas numbers

Name	CAS No
ethanol	64-17-5, 2348-46-1
beta-citronellol	106-22-9, 1117-61-9, 7540-51-4, 26489-01-0, 1335-43-9, 68916-43-8
d-panthenol	81-13-0, 16485-10-2, 17307-32-3
citric acid	77-92-9, 1192555-95-5, 12262-73-6, 136108-93-5, 245654-34-6, 43136-35-2, 623158-96-3, 856568-15-5, 878903-72-1, 890704-54-8, 896506-46-0, 906507-37-7
Aloes, extract	85507-69-3, 94349-62-9

Classification of the preparation and its individual components has drawn on official and authoritative sources 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。

Continued...

IDLH: Immediately Dangerous to Life or Health Concentrations
ES: Exposure Standard
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index
AIIC: Australian Inventory of Industrial Chemicals
DSL: Domestic Substances List
NDSL: Non-Domestic Substances List
IECSC: Inventory of Existing Chemical Substance in China
EINECS: European INventory of Existing Commercial chemical Substances
ELINCS: European List of Notified Chemical Substances
NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory
KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals
PICCS: Philippine Inventory of Chemicals and Chemical Substances
TSCA: Toxic Substances Control Act
TCSI: Taiwan Chemical Substance Inventory
INSQ: Inventario Nacional de Sustancias Químicas
NCI: National Chemical Inventory
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances