Pebeo Gedeo Create Your Own Candles Set B

Jasco Pty Limited

Chemwatch Hazard Alert Code: **2**

Chemwatch: **5491-63**
Version No: **4.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Issue Date: **10/03/2023**
Print Date: **10/08/2024**
L.GHS.AUS.EN

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

Product Identifier

Product name	Pebeo Gedeo Create Your Own Candles Set B
Chemical Name	Not Applicable
Synonyms	0083820; FDS030 - Frosted effect candle scent
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains geranyl acetate)
Chemical formula	Not Applicable
Other means of identification	UFI:9YE0-J0SC-400S-TF1J

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

Relevant identified uses	Use according to manufacturer's directions.

Details of the manufacturer or supplier of the safety data sheet

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

Emergency telephone number

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

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

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Label elements

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

Hazard statement(s)

H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H319	Causes serious eye irritation.
H335	May cause respiratory irritation.
H341	Suspected of causing genetic defects.
H351	Suspected of causing cancer.
H361d	Suspected of damaging the unborn child.
H411	Toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P302+P352	IF ON SKIN: Wash with plenty of water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
78-70-6	2.5-<10	linalool
115-95-7	2.5-<10	linalyl acetate
105-87-3	2.5-<10	geranyl acetate
91-64-5	2.5-<10	coumarin

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CAS No	%[weight]	Name
32210-23-4	2.5-<10	4-tert-butylcyclohexyl acetate
18479-58-8	2.5-<10	dihydromyrcenol
80-26-2	2.5-<10	terpinyl acetate
Not Available	balance	Ingredients determined not to be hazardous

Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available***

SECTION 4 First aid measures

Description of first aid measures

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

Indication of any immediate medical attention and special treatment needed

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.
Treat symptomatically.
for simple esters:

BASIC TREATMENT

- Establish a patent airway with suction where necessary.
- Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- Monitor and treat, where necessary, for pulmonary oedema .
- Monitor and treat, where necessary, for shock.
- **DO NOT** use emetics. Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.
- Give activated charcoal.

ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Treat seizures with diazepam.
- Proparacaine hydrochloride should be used to assist eye irrigation.

Continued...

EMERGENCY DEPARTMENT

- Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- Consult a toxicologist as necessary.

BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

To treat poisoning by the higher aliphatic alcohols (up to C7):

- Gastric lavage with copious amounts of water.
- It may be beneficial to instill 60 ml of mineral oil into the stomach.
- Oxygen and artificial respiration as needed.
- Electrolyte balance: it may be useful to start 500 ml. M/6 sodium bicarbonate intravenously but maintain a cautious and conservative attitude toward electrolyte replacement unless shock or severe acidosis threatens.
- To protect the liver, maintain carbohydrate intake by intravenous infusions of glucose.
- Haemodialysis if coma is deep and persistent. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products, Ed 5)

BASIC TREATMENT

- Establish a patent airway with suction where necessary.
- Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- Monitor and treat, where necessary, for shock.
- Monitor and treat, where necessary, for pulmonary oedema.
- Anticipate and treat, where necessary, for seizures.
- **DO NOT** use emetics. Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.
- Give activated charcoal.

ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- If the patient is hypoglycaemic (decreased or loss of consciousness, tachycardia, pallor, dilated pupils, diaphoresis and/or dextrose strip or glucometer readings below 50 mg), give 50% dextrose.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Treat seizures with diazepam.
- Proparacaine hydrochloride should be used to assist eye irrigation.

EMERGENCY DEPARTMENT

- Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- Acidosis may respond to hyperventilation and bicarbonate therapy.
- Haemodialysis might be considered in patients with severe intoxication.
- Consult a toxicologist as necessary. BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

For C8 alcohols and above.
Symptomatic and supportive therapy is advised in managing patients.

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

Continued...

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves in the event of a fire. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ Use fire fighting procedures suitable for surrounding area. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire. ▸ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	▸ The material is not readily combustible under normal conditions. ▸ However, it will break down under fire conditions and the organic component may burn. ▸ Not considered to be a significant fire risk. ▸ Heat may cause expansion or decomposition with violent rupture of containers. ▸ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke. Combustion products include: carbon dioxide (CO_2) other pyrolysis products typical of burning organic material.
HAZCHEM	•3Z

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	Environmental hazard - contain spillage. ▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Control personal contact with the substance, by using protective equipment. ▸ Contain and absorb spill with sand, earth, inert material or vermiculite. ▸ Wipe up. ▸ Place in a suitable, labelled container for waste disposal.
Major Spills	Environmental hazard - contain spillage. ▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Stop leak if safe to do so. ▸ Contain spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Neutralise/decontaminate residue (see Section 13 for specific agent). ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▸ If contamination of drains or waterways occurs, advise emergency services. **CARE**: Absorbent materials wetted with occluded oil must be moistened with water as they may auto-oxidize, become self heating and ignite. Some oils slowly oxidise when spread in a film and oil on cloths, mops, absorbents may autoxidise and generate heat, smoulder, ignite and burn. In the workplace oily rags should be collected and immersed in water.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ **DO NOT** allow clothing wet with material to stay in contact with skin The 38th Amendment to the IFRA Standard (Nov 2003) states that "linalool and natural products known to be rich in linalool should only be used when the level of peroxides is kept to the lowest practical value. It is recommended to add antioxidants at the time of production of the raw material. The addition of 0.1% BHT or a-tocopherol has shown great efficiency. The maximum peroxide level for products in use should be 20mmol/l." ▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Avoid contact with moisture. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers.

Continued...

	▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	Consider storage under inert gas. ▸ Store in original containers. ▸ Keep containers securely sealed. ▸ No smoking, naked lights or ignition sources. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Polyethylene or polypropylene container. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ Avoid reaction with oxidising agents, bases and strong reducing agents. ▸ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
coumarin	0.88 mg/m3	9.7 mg/m3	58 mg/m3

Ingredient	Original IDLH	Revised IDLH
linalool	Not Available	Not Available
linalyl acetate	Not Available	Not Available
geranyl acetate	Not Available	Not Available
coumarin	Not Available	Not Available
4-tert-butylcyclohexyl acetate	Not Available	Not Available
dihydromyrcenol	Not Available	Not Available
terpinyl acetate	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
linalool	E	≤ 0.1 ppm
linalyl acetate	E	≤ 0.1 ppm
geranyl acetate	E	≤ 0.1 ppm
coumarin	E	≤ 0.01 mg/m³
4-tert-butylcyclohexyl acetate	E	≤ 0.1 ppm
dihydromyrcenol	E	≤ 0.1 ppm
terpinyl acetate	E	≤ 0.1 ppm
Notes:	***Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.***	

MATERIAL DATA

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are:

Continued...

Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.

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

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

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
Care: Atmospheres in bulk storages and even apparently empty tanks may be hazardous by oxygen depletion. Atmosphere must be checked before entry.

Requirements of State Authorities concerning conditions for tank entry must be met. Particularly with regard to training of crews for tank entry; work permits; sampling of atmosphere; provision of rescue harness and protective gear as needed

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.

For esters:

- Do NOT use natural rubber, butyl rubber, EPDM or polystyrene-containing materials.

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.

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

Respiratory protection

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

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	A-AUS / Class1 P2	-
up to 50	1000	-	A-AUS / Class 1 P2
up to 50	5000	Airline *	-
up to 100	5000	-	A-2 P2
up to 100	10000	-	A-3 P2
100+			Airline**

* - Continuous Flow ** - Continuous-flow or positive pressure demand
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	Liquid; dilutable in water.

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Physical state	Liquid	**Relative density (Water = 1)**	1
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Applicable
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Applicable	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Applicable	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Applicable	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Dilutable	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	350

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. 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. The main effects of simple aliphatic esters are narcosis and irritation and anaesthesia at higher concentrations. These effects become greater as the molecular weights and boiling points increase. Central nervous system depression , headache, drowsiness, dizziness, coma and neurobehavioral changes may also be symptomatic of overexposure. Respiratory tract involvement may produce mucous membrane irritation, dyspnea, and tachypnea, pharyngitis, bronchitis, pneumonitis and, in massive exposures, pulmonary oedema (which may be delayed). Gastrointestinal effects include nausea, vomiting, diarrhoea and abdominal cramps. Liver and kidney damage may result from massive exposures. Exposure to aliphatic alcohols with more than 3 carbons may produce central nervous system effects such as headache, dizziness, drowsiness, muscle weakness, delirium, CNS depression, coma, seizure, and neurobehavioural changes. Symptoms are more acute with higher alcohols. Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, respiratory depression secondary to CNS depression, pulmonary oedema, chemical pneumonitis and bronchitis. Cardiovascular involvement may result in arrhythmias and hypotension. Gastrointestinal effects may include nausea and vomiting. Kidney and liver damage may result following massive exposures. The alcohols are potential irritants being, generally, stronger irritants than similar organic structures that lack functional groups (e.g. alkanes) but are much less irritating than the corresponding amines, aldehydes or ketones. Alcohols and glycols (diols) rarely represent serious hazards in the workplace, because their vapour concentrations are usually less than the levels which produce significant irritation which, in turn, produce significant central nervous system effects as well. Inhalation hazard is increased at higher temperatures.

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Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. Effects on the nervous system characterise over-exposure to higher aliphatic alcohols. These include headache, muscle weakness, giddiness, ataxia, (loss of muscle coordination), confusion, delirium and coma. Gastrointestinal effects may include nausea, vomiting and diarrhoea. In the absence of effective treatment, respiratory arrest is the most common cause of death in animals acutely poisoned by the higher alcohols. Aspiration of liquid alcohols produces an especially toxic response as they are able to penetrate deeply in the lung where they are absorbed and may produce pulmonary injury. Those possessing lower viscosity elicit a greater response. The result is a high blood level and prompt death at doses otherwise tolerated by ingestion without aspiration. In general the secondary alcohols are less toxic than the corresponding primary isomers. As a general observation, alcohols are more powerful central nervous system depressants than their aliphatic analogues. In sequence of decreasing depressant potential, tertiary alcohols with multiple substituent OH groups are more potent than secondary alcohols, which, in turn, are more potent than primary alcohols. The potential for overall systemic toxicity increases with molecular weight (up to C7), principally because the water solubility is diminished and lipophilicity is increased. Within the homologous series of aliphatic alcohols, narcotic potency may increase even faster than lethality Only scanty toxicity information is available about higher homologues of the aliphatic alcohol series (greater than C7) but animal data establish that lethality does not continue to increase with increasing chain length. Aliphatic alcohols with 8 carbons are less toxic than those immediately preceding them in the series. 10 -Carbon n-decyl alcohol has low toxicity as do the solid fatty alcohols (e.g. lauryl, myristyl, cetyl and stearyl). However the rat aspiration test suggests that decyl and melted dodecyl (lauryl) alcohols are dangerous if they enter the trachea. In the rat even a small quantity (0.2 ml) of these behaves like a hydrocarbon solvent in causing death from pulmonary oedema. Primary alcohols are metabolised to corresponding aldehydes and acids; a significant metabolic acidosis may occur. Secondary alcohols are converted to ketones, which are also central nervous system depressants and which, in he case of the higher homologues persist in the blood for many hours. Tertiary alcohols are metabolised slowly and incompletely so their toxic effects are generally persistent. Coumarin, can cause liver damage in highly sensitive individuals. However, the effect can be reversed once coumarin intake is halted. This plant ingredient is found in woodruff and sweet clover and there are higher levels in cassia cinnamon, too. Consumers mainly ingest coumarin from food containing high levels of cassia cinnamon. In animal experiments high concentrations of the substance have triggered cancer. Based on hepatotoxicity a tolerable daily intake (TDI) of 0.1 milligram coumarin per kilogram bodyweight was derived for coumarin. This should not be exceeded in the long term At sufficiently high doses the material may be hepatotoxic (i.e. poisonous to the liver). Signs may include nausea, stomach pains, low fever, loss of appetite, dark urine, clay-coloured stools, jaundice (yellowing of the skin or eyes) Terpenes and their oxygen-containing counterparts, the terpenoids, produce a variety of physiological effects. Pine oil monoterpenes, for example, produce a haemorrhagic gastritis characterised by stomach pain and bleeding and vomiting. Systemic effects of pine oils include weakness and central nervous depression, excitement, loss of balance, headache, with hypothermia and respiratory failure. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Synthetically produced coumarin is added as a fragrance to cosmetics and can reach the body through the skin. Consumers may already exceed the tolerable daily intake (TDI) of coumarin just by using cosmetics with high coumarin levels. It has not been fully elucidated whether coumarin taken in via the skin has a similarly harmful effect on the liver to coumarin ingested from the gastro-intestinal tract. Whereas a maximum of two milligram coumarin per kilogram may be added to foods as a flavouring, there are no maximum limits for coumarin in cosmetics. However, as coumarin, in common with other fragrances, can trigger allergies in highly sensitive individuals, too, it must be labelled as an ingredient in cosmetics from specific concentrations upwards. Coumarin easily reaches the human body through the skin. Cosmetics can, therefore, contribute to the total exposure of consumers to coumarin. Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.

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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.

Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.
Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.
Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.

Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.
A number of common flavor and fragrance chemicals can form peroxides surprisingly fast in air. Antioxidants can in most cases minimize the oxidation.
Fragrance terpenes are easily oxidized in air. Non-oxidised forms are very weak sensitizers; however, after oxidation, the hyproperoxides are strong sensitisers which may cause allergic reactions. Autooxidation of fragrance terpenes contributes greatly to fragrance allergy. There is the need to test for compounds the patients are actually exposed to, not only the ingredients originally applied in commercial formulations.

Linalool (a terpinoid) is an unsaturated tertiary alcohol. It is a naturally occurring component together with linalyl esters in a variety of fruits, fruit peels, fruit juices, vegetables and spices as for example laurel, coriander seeds and clary sage. The annual worldwide use of linalool and linalyl acetate in fragrances exceeds 1000 metric tons.

For consideration of potential sensitization the exposure is calculated as a percent concentration used on the skin. Exposure to linalool used in fine fragrance products is reported as 4.3% based on the use of 20% of the fragrance mixture in the fine fragrance consumer product.

Experimental studies in laboratory animals combined with advanced chemical analyses have shown that linalool is easily oxidized, and that the content of linalool decreased to about 80% after oxidation for 10 weeks at standardized conditions. One of the major oxidation products was identified as 7-hydroperoxy-3,7-dimethyl-octal-1,5-diene-3-ol. In guinea pig sensitisation studies a sample of oxidized linalool was a significant allergen sensitizing 8 of 15 test animals, whereas controls were negative. Linalyl hydroperoxide is a very strong sensitiser at the 1% level. Further studies have documented the sensitising capacity of linalool and derivatives found commercially available grade of linalool (97% purity) to be a weak sensitiser. When impurities were identified and removed the sensitising capacity was reduced but not eliminated. During storage, linalool undergoes autoxidation, building up products including hydroperoxides such as 7-hydroperoxy-3,7-dimethyl-octal-1,5-diene-3-ol , which has been identified as the apparent cause allergic reactions on exposed skin. Animal testing data found that with guinea pigs, ten week old samples of linalool sensitized the animals skin, but highly purified linalool produces no reaction. Auto-oxidation was therefore identified by the authors as necessary for the sensitising process.
Peroxidisable terpenes and terpenoids should only be used when the level of peroxides is kept to the lowest practicable level, for instance by adding antioxidants at the time of production. Such products should have a peroxide value of less than 10 millimoles peroxide per liter. This requirement is based on the published literature mentioning sensitising properties when containing peroxides.

Pebeo Gedeo Create Your Own Candles Set B	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

linalool	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: 5610 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
	Oral (Rat) LD50: 2790 mg/kg[2]	Skin (guinea pig):100mg/24h-mild
		Skin (man): 16 mg/48h-mild
		Skin (rabbit): 100 mg/24h-SEVERE
		Skin (rabbit): 500 mg/24h - mild
		Skin: adverse effect observed (irritating)[1]

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linalyl acetate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >5000 mg/kg[2]	Skin (guinea pig): 100mg/24h-mod
	Oral (Mouse) LD50; 12000 mg/kg[2]	Skin (rabbit): 100 mg/24h-SEVERE
geranyl acetate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: 2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: 6330 mg/kg[2]	Skin (guinea pig):100mg/24h mod
		Skin (man): 16 mg/48h - mild
		Skin (rabbit): 100 mg/24h-SEVERE
		Skin: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
coumarin	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: 293 mg/kg[1]	Not Available
	Oral (Rat) LD50: ~290 mg/kg[1]	
4-tert-butylcyclohexyl acetate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >4670 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >300<2000 mg/kg[1]	Skin (rabbit): 500 mg/24h mod
		Skin: no adverse effect observed (not irritating)[1]
dihydromyrcenol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >5000 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
	Oral (Rat) LD50: 3600 mg/kg[2]	Skin (rabbit): 500 mg/24h - mild
		Skin: adverse effect observed (irritating)[1]
terpinyl acetate	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 5075 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]

Legend:	***1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances***

LINALOOL

For monoterpenes:
The chemical category designated terpenoid hydrocarbons includes three simple C10 isomeric monocyclic terpene hydrocarbons (***d-***limonene, ***dl-***limonene, and terpinolene) two simple C10 acyclic terpene hydrocarbons (***beta-***myrcene and dihydromyrcene) and mixtures composed primarily of ***d-***limonene, ***dl-***limonene (dipentene), terpinolene, myrcene, and ***alpha***and ***beta***-pinene
Monoterpene hydrocarbons are mainly released by coniferous woodland such as pine trees, cedars, redwood and firs. To a lesser extent, they are also produced and released by deciduous plants. They are common components of traditional foods occurring in essentially all fruits and vegetables.
Members of this chemical category are of very low acute toxicity
Studies of terpene hydrocarbons indicate that they are rapidly absorbed, distributed, metabolised and excreted. The principal metabolic pathway involves side chain oxidation to yield monocyclic terpene alcohols and carboxylic acids. These metabolites are mainly conjugated with glucuronic acid and excreted in the urine, or to a lesser extent in the feces. A secondary pathway involves epoxidation of either the exocyclic or endocyclic double bond yielding an epoxide that is subsequently detoxicated ***via*** formation of the corresponding diol or conjugation with glutathione. Although some species- and sex-specific differences exist, studies for ***d-***limonene and ***beta***-myrcene indicate that the monoterpene hydrocarbons in this chemical category will participate in common pathways of absorption, distribution, metabolism and excretion.
Genotoxicity: Based on the results of this ***in vivo*** genotoxicity assay and the numerous ***in vitro*** genotoxicity assays, it is unlikely that any of these materials would exhibit a significant genotoxic potential ***in vivo***.
Carcinogenicity: Under the conditions of 2-year gavage studies, conducted by NTP, there was clear evidence of carcinogenic activity of ***d-***limonene for male F344/N rats as shown by increased incidences in tubular cell hyperplasia, adenomas, and adenocarcinomas of the kidney. There was no evidence of carcinogenic activity of ***d-***limonene for female rats receiving 300 or 600 mg/kg bw/d. It has been demonstrated that renal lesions, which were observed in the NTP study, resulted from the accumulation of aggregates of ***alpha***-2 microglobulin (a low molecular-weight protein synthesised in the liver) and limonene-1,2-epoxide in the P2 segment of the renal proximal tubule. While humans produce low molecular weight serum proteins, which are reabsorbed by the kidney, there is no evidence that a similar ***alpha-***2 microglobulin is produced.
The kidney changes seen in male rats administered limonene have been well characterized, and are known to be specific to the male rat and of no significance in human risk assessment.
Reproductive toxicity: Substances within this chemical category exhibit low reproductive toxicity potential. This is based on the results of three reproductive toxicity assays. using sweet orange peel oil predominantly composed of ***d-***limonene and ***beta-***

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	myrcene. **Developmental toxicity:** Given the results of six developmental toxicity assays using limonene, sweet orange oil and ***beta***-myrcene, it may be concluded that the substances within this chemical category exhibit low developmental toxicity potential 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
GERANYL ACETATE	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 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.

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	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)
COUMARIN	umarin is moderately toxic to the liver and kidneys of rodents, with a median lethal dose (LD50) of 293 mg/kg in the rat,[] a low toxicity compared to related compounds. Coumarin is hepatotoxic in rats, but less so in mice. Rodents metabolize it mostly to 3,4-coumarin epoxide, a toxic, unstable compound that on further differential metabolism may cause liver cancer in rats and lung tumors in mice] Humans metabolize it mainly to 7-hydroxycoumarin, a compound of lower toxicity, and no adverse affect has been directly measured in humans.[The German Federal Institute for Risk Assessment has established a tolerable daily intake (TDI) of 0.1 mg coumarin per kg body weight, but also advises that higher intake for a short time is not dangerous.[] The Occupational Safety and Health Administration (OSHA) of the United States does not classify coumarin as a carcinogen for humans European health agencies have warned against consuming high amounts of cassia bark, one of the four main species of cinnamon, because of its coumarin content] According to the German Federal Institute for Risk Assessment (BFR), 1 kg of (cassia) cinnamon powder contains about 2.1 to 4.4 g of coumarin.] Powdered cassia cinnamon weighs 0.56 g/cm3,[33] so a kilogram of cassia cinnamon powder equals 362.29 teaspoons. One teaspoon of cassia cinnamon powder therefore contains 5.8 to 12.1 mg of coumarin, which may be above the tolerable daily intake value for smaller individuals.[32] However, the BFR only cautions against high daily intake of foods containing coumarin. Its repor] specifically states that Ceylon cinnamon (Cinnamomum verum) contains "hardly any" coumarin he European Regulation (EC) No 1334/2008 describes the following maximum limits for coumarin: 50 mg/kg in traditional and/or seasonal bakery ware containing a reference to cinnamon in the labeling, 20 mg/kg in breakfast cereals including muesli, 15 mg/kg in fine bakery ware, with the exception of traditional and/or seasonal bakery ware containing a reference to cinnamon in the labeling, and 5 mg/kg in desserts. An investigation from the Danish Veterinary and Food Administration in 2013 shows that bakery goods characterized as fine bakery ware exceeds the European limit (15 mg/kg) in almost 50% of the cases.[34] The paper also mentions tea as an additional important contributor to the overall coumarin intake, especially for children with a sweet habit. Coumarin was banned as a food additive in the United States in 1954, largely because of the hepatotoxicity results in rodent] Coumarin is currently listed by the Food and Drug Administration (FDA) of the United States among "Substances Generally Prohibited From Direct Addition or Use as Human Food," according to 21 CFR 189.130,[36][37] but some natural additives containing coumarin, such as the flavorant sweet woodruff are allowed "in alcoholic beverages only" under 21 CFR 172.510. In Europe, popular examples of such beverages are Maiwein, white wine with woodruff, and Zubrówka, vodka flavoured with bison grass. umarin is subject to restrictions on its use in perfumery,[39] as some people may become sensitized to it, however the evidence that coumarin can cause an allergic reaction in humans is disputed nor neurological dysfunction was found in children exposed to the anticoagulants acenocoumarol or phenprocoumon during pregnancy. A group of 306 children were tested at ages 7–15 years to determine subtle neurological effects from anticoagulant exposure. Results showed a dose–response relationship between anticoagulant exposure and minor neurological dysfunction. Overall, a 1.9 (90%) increase in minor neurological dysfunction was observed for children exposed to these anticoagulants, which are collectively referred to as "coumarins." In conclusion, researchers stated, "The results suggest that coumarins have an influence on the development of the brain which can lead to mild neurologic dysfunctions in children of school age. Alcoholic beverages sold in the European Union are limited to a maximum of 10 mg/L coumarin by law. Cinnamon flavor is generally cassia bark steam-distilled to concentrate the cinnamaldehyde, for example, to about 93%. Clear cinnamon-flavored alcoholic beverages generally test negative for coumarin, but if whole cassia bark is used to make mulled wine, then coumarin shows up at significant levels Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies. Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A prehapten is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems. A prohapten is a chemical that itself is non- or low-sensitising but that is transformed into a hapten in the skin (bioactivation) usually via enzyme catalysis. It is not always possible to know whether a particular allergen that is not directly reactive acts as a prehapten or as a prohapten, or both, because air oxidation and bioactivation can often give the same product (geraniol is an example). Some chemicals might act by all three pathways. **Prohaptens** Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohaptens. In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal. The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I

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	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 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.
4-TERT-BUTYLCYCLOHEXYL ACETATE	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. There are no safety concerns regarding cyclic acetates, as fragrance ingredients, under the present declared levels of use. This opinion was based on the following reasons: · The cyclic acetates have a low order of acute toxicity. · The cyclic acetates and the cyclic alcohols tested are of low systemic toxicity upon repeated dermal application. · Minimal, if any, evidence of skin irritation in humans is associated with current levels of use at 2-20% for individual cyclic acetates. · These materials have no, or a low, sensitizing potential. · The cyclic acetates and the cyclic alcohols tested are of low systemic toxicity upon repeated dermal application. NOAELs for compounds or their metabolites are in the range of 50-500 mg/kg bw/day in rats · Data on carcinogenicity of cyclic acetates are not available but in view of the negative mutagenicity tests so far obtained. This is not of primary concern. · Available genotoxicity data do not show a genotoxic potential of the substances · Compared to the estimated highest daily uptake of 0.246 mg/kg bw/day of d-cy clocitronellene acetate (100% dermal absorption) the margin of safety for this compound is at least 200 (estimated 50% oral absorption). For the other acetates with estimated daily doses the margin of safety ranges from 269 (myraldyl acetate) to 166,666 (cyclohexyl acetate). The common characteristic structural element of cyclic acetates is the acetate unit bound to a mono-, bi- or tri-cyclic alcohol. The present group comprises 25 substances which include 15 esters of monocyclic alcohols, three of bicyclic alcohols, and seven of tricyclic alcohols. The only substituents at the alcohol moiety are alkyl groups. Some esters contain cyclic or exocyclic double bonds or a terminal triple bond. Although data on metabolism are lacking and the toxicity of only a few compounds has been studied, cyclic acetates are assumed to be rapidly hydrolyzed to the alcohol and the carboxylic acid by carboxylesterases. The local and systemic toxicity of the compounds under review is exerted either by the parent compound or by the hydrolysis products. This hydrolysis is on the one hand a detoxification step. It transforms the poorly water. soluble ester into an alcohol, which can subsequently be conjugated and excreted faster than the parent compound, thereby diminishing the potential toxicity of the ester itself. On the other hand, the resulting acid (in this case acetic acid for all but one substance) may cause irritation due to a pH-shift in tissues where hydrolysis initially occurs. Even esters (octahydro-4,7-methano-1H-indenemethyl acetate and myraldyl acetate) that may be metabolized to both acetic acid and a cyclic acid (via the primary alcohol), have no effect concentrations for skin irritation in humans of 10-20%.The same is expected for abietyl acetate, because its possible metabolite abietic acid is a weak acid. Therefore, due to the structural similarities, compounds not tested for skin irritation in humans are expected to be of no concern provided concentrations in end products are in the range of 2–20% The Research Institute for Fragrance Materials (RIFM) Expert Panel
DIHYDROMYRCENOL	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.
LINALOOL & LINALYL ACETATE & GERANYL ACETATE & COUMARIN	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.
LINALOOL & LINALYL ACETATE	For linalool: Linalool gradually breaks down when in contact with oxygen, forming an oxidized by-product that may cause allergic reactions such as eczema in susceptible individuals. Between 5 and 7% of patients undergoing patch testing in Sweden were found to be allergic to the oxidized form of linalool.[Linalool has an acute oral mammalian LD50 close to 3,000 mg/kg bw; the acute dermal toxicity is ~ 2,000 mg/kg bw. After inhalation exposure of mice and man, slight sedative effects were observed; however a dose response characteristic could not

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	be determined. Linalool is irritating to the skin, based on animal studies, and is a mild irritant from human experience. It may be moderately irritant to the eyes at the same concentration where it produces nasal pungency. Linalool is considered not to be a sensitiser. The incidence of dermal reaction to inalool is below 1% in naïve probands (not knowingly pre-sensitised) while in subjects pre-sensitised to fragrances it is up to 10%. In a 28-day oral rat study (72.9% linalool) findings were increased liver and kidney weight, thickened liver lobes and pale areas on the kidneys and in females only hepatocellular cytoplasmic vacuolisation. Other findings were related to local irritation of the gastro-intestinal tract. Based on the effects on liver and kidney a NOAEL of 160 mg/kg bw/d (equivalent to 117 mg/kg bw/d linalool) was derived. In this study no effects on male and female gonads were found. Linalool was not mutagenic in seven out of eight bacterial tests nor in two (one ***in vitro*** and one ***in vivo***) mammalian tests; the one positive bacterial result is estimated to be a chance event. Linalool (72.9%) was tested in a reproduction screening test (non-OECD). The NOAEL for maternal toxicity based on clinical signs and effects on body weight and food consumption was 500 mg/kg bw/d (equivalent to 365 mg/kg bw/d linalool). The NOAEL on reproduction toxicity and developmental toxicity is 500 mg/kg bw/d (equivalent to 365 mg/kg bw linalool), based on the decreased litter size at birth and pup morbidity/mortality thereafter. Linalool seems not to be an immunotoxicant according to one animal study. Opinion holds that there are no safety concerns for linalool and the linalyl esters, as fragrance ingredients, under the present declared levels of use and exposure for the following reasons: · Linalool and the linalyl esters have a low order of acute toxicity. · No significant toxicity was observed in subchronic tests; it is concluded that these materials have dermal and oral NOAELS of 50 mg/kg/day or greater. · Based on a critical review of all available mutagenicity and genotoxicity studies,it has been determined that these materials are negative in short-term tests and therefore would have no significant potential to produce genotoxic effects. · The metabolic fate of linalool and the linalyl esters is either known or assumed from analogies with structurally related substances that indicate no production of toxic or persistent metabolites and the structural analogies indicate no concern. · Human dermatological studies show that these materials are not irritating, phototoxic or sensitizing. · These materials are used at low levels of exposure relative to doses that elicit adverse effects. The estimate for maximum systemic exposure by humans using cosmetic products is 0.3 mg/kg/ day for linalool and linalyl acetate and 0.1 mg/ kg/day or lower for the other linalyl esters. Using the NOAELs (50 mg/kg/day or greater) and the maximum exposure estimates and assuming 100% absorption,a margin of safety for the exposure of humans to linalool and the linalyl esters may conservatively be calculated as 167 times the maximum daily exposure for linalool and linalyl acetate (50 mg/kg/day 0.3 mg/kg/day for linalool or linalyl acetate=167) and 500 times the maximum daily exposure for the other individual linalyl esters (50 mg/kg/day / 0.1 mg/kg/day for the other individual linalyl esters=500). In general, linalool esters are hydrolyzed to their corresponding alcohol (linalool) and carboxylic acid. Hydrolysis is catalyzed by carboxylesterases or esterases . Tertiary alcohols such as linalool are metabolized primarily through conjugation with glucuronic acid and are excreted in the urine and to a lesser extent faeces. Alkyl or alkenyl substituents may undergo oxidation to form polar metabolites that may also be excreted free or in the conjugated form. Oxidation is mediated by cytochrome P-450 dependant mono-oxygenases. The carboxylic acids formed by hydrolysis of the linalyl esters included in this summary are all known to be easily and rapidly metabolized. The linear saturated carboxylic acids are metabolized normally as fatty acids that undergo beta-oxidation. The branched-chain carboxylic acids from linalyl isovalerate and isobutyrate are similarly oxidized,but the end product is acetone. The carboxylic acids from linalyl benzoate and phenylacetate are conjugated and excreted. The cinnamic acid from linalyl cinnamate is conjugated and excreted,or metabolized to benzoic acid. No sensitization was observed with linalool in guinea pig sensitization studies at concentrations up to 20%. With linalyl acetate at a concentration of 10%,weak to moderate sensitization effects were observed in guinea pig sensitization studies. Linalyl acetate was non-sensitizing when tested at 5% in these same guinea pig sensitization studies. No sensitization reactions were observed with linalyl isobutyrate and linalyl propionate (data were not available for the other linalyl esters) when tested at 8% in open epicutaneous tests in guinea pigs The Research Institute for Fragrance Materials (RIFM) Expert Panel
LINALOOL & LINALYL ACETATE & TERPINYL ACETATE	A member or analogue of a group of aliphatic and alicyclic terpenoid tertiary alcohols and structurally related substances generally regarded as safe (GRAS based, in part, on their self-limiting properties as flavouring substances in food; their rapid absorption, metabolic conversion, and excretion in humans and experimental animals; their low level of flavour use; the wide margins of safety between the conservative estimates of intake and the no-observed-adverse effect levels (NOAEL) determined from subchronic and chronic studies and the lack of genotoxic and mutagenic potential. This evidence of safety is supported by the fact that the intake of aliphatic acyclic and alicyclic terpenoid tertiary alcohols and structurally related substances as natural components of traditional foods is greater than their intake as intentionally added flavoring substances. Oral median lethal dose (LD50) values have been reported for 24 of the 43 substances in this group. LD50 values range from 1300 to greater than 36300 mg/kg bw, demonstrating that the oral acute toxicity of tertiary alcohols and related esters is extremely low. Genotoxicity: the testing of representative materials in vitro in bacterial test systems (Ames assay) and in vivo in mammalian test systems (micronucleus assay) showed no evidence of mutagenic or genotoxic potential. Based on the results of studies under a wide variety of conditions, including aqueous buffered media, simulated gastric juice, simulated human intestinal fluid, blood plasma, whole hepatocytes and liver microsome preparations, terpene esters formed from tertiary alcohols (for example, linalool), and simple aliphatic carboxylic acids are expected to undergo hydrolysis.Bicyclic tertiary alcohols are relatively stable in vivo, but are eventually conjugated with glucuronic acid and excreted Although differences in the rates of hydrolysis occur under in vitro conditions in gastric juice and intestinal fluids, ready hydrolysis is observed in tissue preparations that have an abundant concentration of carboxylesterases (CES), especially the liver The most important class of these enzymes is the B-esterases, which are members of the serine esterase superfamily. Generally, CES enzymes are ubiquitous throughout mammalian tissues and are found at the highest levels in hepatocytes In general, the esters are hydrolysed to their corresponding alcohol and carboxylic acid. It is expected that the tertiary aromatic alcohols will undergo direct conjugation of the hydroxyl group with glucuronic acid while the tertiary terpenoid alcohols formed as a result of hydrolysis are rapidly absorbed and converted to the glucuronic acid conjugates which are excreted in the urine, or are further oxidised to CO2 that is subsequently expired Aliphatic acyclic and alicyclic terpenoid tertiary alcohols and structurally related substances often have a sweet floral rose to a fruity citrus green organoleptic profile.Twenty-two of the 44 flavor ingredients in this group have been reported to occur naturally, and can be found in chamomile, cocoa, coffee, a variety of fruits and especially citrus fruit varieties and vegetables, lemon juice, black and green teas, calamus, soybean, pepper, strawberry guava, beer and wine

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	Flavor and Extract Manufacturers' Association (FEMA)
LINALOOL & LINALYL ACETATE & DIHYDROMYRCENOL & TERPINYL ACETATE	For terpenoid tertiary alcohols and their related esters: Substances assigned to this category, as part of the HPV Challenge Program,possess close structural relationships, similar physicochemical properties and participate in the same pathways of metabolic detoxification and have similar toxicologic potential. **Acute Toxicity:** Oral and dermal LD50 values for members of this chemical category indicate a low order of both oral and dermal toxicity. All rabbit dermal, and mouse and rat oral LD50 values exceed 2000 mg/kg with the majority of values greater than 5000 mg/kg **Repeat dose toxicity:** In a safety evaluation study, a 50/50 mixture of linalool and citronellol was fed to male and female rats (number and strain not specified) in the diet. The daily intake was calculated to be 50 mg/kg bw of each. Measurements of haematology, clinical chemistry, and urinalysis at weeks 6 and 12 showed no statistically significant differences between test and control groups. Histopathology revealed no dose-related lesions. A slight retardation of growth was observed in males only, but was concluded by the authors to be biologically insignificant **Reproductive toxicity:** Four groups of 10 virgin Crl CD rats were administered 0,250,500, or 1000 mg/kg bw of an essential oil (coriander oil) known to contain 73% linalool by mass. The test material was given by gavage once daily, 7 days prior to cohabitation, through cohabitation (maximum of 7 days), gestation, delivery, and a 4-day post-parturition period. The duration of the study was 39 days. Maternal effects reported included increased body weight and increased food consumption at 250 mg/kg/d, a non-statistically significant decrease in body weight and food consumption and decreased gestation index and decreased length of gestation at 500 mg/kg/d, and a statistically significant decrease in body weight and food consumption, statistically significant decrease in gestation index, length of gestation, and litter size at 1000 mg/kg/d. The only effect on pups was a decrease in viability of pups at the highest dose level. The authors concluded that there were no effects observed in the dams at the low dose of 250 mg/kg bw/d or in the offspring at the 250 and 500 mg/kg bw/d levels. The authors concluded that the maternal NOAEL was 250 mg/kg/d and the developmental NOAEL was 500 mg/kg/d. Four groups of 10 virgin Crl CD rats were administered 0,375,750, or 1500 mg/kg bw of an essential oil (cardamom oil) known to contain greater than 65 % tertiary terpenoid alcohols with 5 1% alpha-terpineol acetate by mass. Maternal observations included a non-statistically significant decrease in body weight gain and food consumption at 375 mg/kg/d. Mortality, clinical signs, a statistically significant decrease in body weight gain and food consumption, and gross lesions at necropsy were seen at 750 and 1500 mg/kg/d. The only effects on pups were a reduced body weight gain in pups at 750 and 1500 mg/kg/d and increased mortality at 1500 mg/kg/d. The authors concluded that there were no significant adverse effects in the dams or offspring at the 375 mg/kg/d dose. A maternal NOEL was reported to be less than 375 mg/kg/d based on reduced body weight gain and food consumption at 375 mg/kg/d and a developmental NOAEL was reported to be 375 mg/kg/d **Developmental toxicity**: A range finding study and follow-up teratology study was performed with pine oil. Pregnant Crl:CD(SD) BR rats were given 0, 50, 100, 500,750,or 1000 mg/kg/d by gavage in corn oil on days 6 to 20 of gestation. Laparotomies were performed, corpora lutea were counted, and the uterus of each rat was removed, weighed and then examined for number, placement and viability of implantations. Live foetuses were weighed, sexed and gross external alternations were identified. There were no deaths or abortions during the course of this study. Necropsy revealed no gross lesions. Maternal effects included local alopecia, decreased body weight gain and food consumption for the 3 highest dose levels. At 750 and 1000 mg/kg, average gravid uterine weight was reduced. In foetuses, decreased body weight was observed at dose levels of 100 mg/kg and above, and at dose levels of 500 and above there was a slight increase in average number of resorptions/litter. In the follow-up teratology study, pregnant Crl:CD(SD) BR rats were given 0, 50, 600, or 1200 mg/kg/d by gavage in corn oil on days 6 to 20 of gestation. Six of the 25 rats in 1200 mg/kg dose group died and necropsies revealed that adrenal weights were significantly increased in these rats. At 1200 mg/kg/d, foetuses exhibited increased incidences of delayed ossification, delayed brain development, decreased weights, increased embryo -foetal mortality, and sunken eye bulge with associated soft and hard tissue findings, a dose that also resulted in maternal death and a low incidence of embryo-foetal death (resorption). The maternal and developmental NOEL for pine oil was greater than 50 mg/kg/d but less than 600 mg/kg/d **Genotoxicity:** Mutagenicity/genotoxicity testing has been performed on six members of this chemical category, including a complete battery of in vitro genotoxicity tests using linalool. In nineteen separate in vitro tests on the mutagenicity and genotoxicity of terpenoid tertiary alcohols and related esters, all but two were negative. One of the positive results for linalool was observed in a rec assay using differences in growth rates in two strains of Bacillus subtilis as a measure of DNA changes In contrast, no evidence of mutagenicity was observed in the same test at a higher concentrations nor was DNA damage observed in a rat hepatocyte UDS assay. The authors of the mouse lymphoma assay which gave a weak positive result for linalool, emphasized that positive results in this assay are commonly observed for polar substances in the absence of S-9 and may be associated with changes in physiologic culture conditions (pH and osmolality). Based on a weight of evidence evaluation of the available in vitro and in vivo mutagenicity and genotoxicity assays on terpenoid tertiary alcohols and related esters, this group of flavouring substances would not be expected to exhibit a low genotoxic potential in vivo **Metabolic fate:** Based on the results of hydrolysis, the reactivity of linalool in aqueous media, and data on metabolism it is concluded that members of this chemical category exhibit similar chemical and biochemical fate. The esters are readily hydrolyzed to the corresponding alcohols, linalool and alpha-terpineol. Linalool is then partial converted to alpha-terpineol mainly under acidic1conditions. Alicyclic and aliphatic tertiary alcohols are efficiently detoxicated by two principal pathways: conjugation primarily with glucuronic acid and excretion primarily in urine, and omega-oxidation to eventually yield diacids and their reduced or hydrated analogs. These polar metabolites will be efficiently excreted primarily in the urine either unchanged or as the glucuronic acid conjugates. The physiochemical and toxicological properties of these substances are consistent with their known reactivity and common metabolic fate. Esters belonging to this category can be hydrolysed to their corresponding terpenoid alcohol and organic acid. Hydrolysis can also be catalysed by a class of esters known as carboxylesterases or B-type esterases that predominated in hepatocytes. Esters of tertiary terpenoid alcohols are readily hydrolyzed in animals, including fish. Once hydrolysed, the resulting alcohols undergo excretion unchanged or as the glucuronic acid conjugate. To a minor extent, CYP-450 mediated oxidation at the omega or omega-1 position yields polar oxidized metabolites capable of excretion primarily in the urine Terpenoid alcohols formed in the gastrointestinal tract are readily absorbed. During hydrolysis under acidic condition cyclisation may occur. In humans and animals, terpenoid tertiary alcohols primarily conjugate with glucuronic acid and are excreted in the urine and feces. Terpenoid alcohols with unsaturation may also undergo allylic oxidation to form polar diol metabolites that may be excreted either free or conjugated. If the diol contains a primary alcohol function, it may undergo further oxidation to the corresponding carboxylic acid. In a minor pathway, the endocyclic alkene of alpha-terpineol is epoxidised and then hydrolyzed to yield a triol metabolite 1,2,8-trihydroxy--p-menthane which also has been reported in humans following inadvertent oral ingestion of a pine oil disinfectant containing alpha-terpineol.

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	Bicyclic tertiary alcohols are conjugated with glucuronic acid and excreted primarily in the urine. In rabbits the structurally related bicyclic tertiary alcohols thujyl alcohol (4-methyl-1-(l-methylethyl)bicyclo[3.1.0]-hexan-3-ol) and beta-santenol (2,3,7-trimethylbicyclo[2.2.1]-heptan-2-ol) are conjugated with glucuronic acid. In a metabolism study using the terpenoid tertiary alcohol trans-sobrerol , in humans, dogs, and rats, ten metabolites were isolated in urine, eight of which were characterised in humans. Two principle modes of metabolism were observed, allylic oxidation of the ring positions and alkyl substituents, and conjugation of the tertiary alcohol fractions with glucuronic acid. These metabolic patterns are common modes of converting tertiary and secondary terpenoid alcohols to polar metabolites, which are easily excreted in the urine and faeces . Menthol forms similar conjugation products in rats
LINALOOL & DIHYDROMYRCENOL	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. 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

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	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.
LINALOOL & LINALYL ACETATE & GERANYL ACETATE & COUMARIN & DIHYDROMYRCENOL	Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and connubial contact dermatitis occur. Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes. Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits. Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water. Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis. Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a suffcient degree of fragrance contact allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the allergen. As a consequence, symptoms, i.e. allergic contact dermatitis, may occur upon re-exposure to the fragrance allergen(s) in question. Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or hands. It may affect fitness for work and the quality of life of the individual. Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure. **Hands:** Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema may often be complicated by sensitisation .Fragrance allergy may be a relevant problem in patients with hand eczema; perfumes are present in consumer products to which their hands are exposed. A significant relationship between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe) chronic hand eczema may not be clear. **Axillae Bilateral axillary** (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy. **Face** Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of of being fragrance allergic. **Irritant reactions (including contact urticaria):** Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this, Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported . The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested , but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen. **Pigmentary anomalies:** The term “pigmented cosmetic dermatitis” was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified.. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil. **Photo-reactions** Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon . Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. Phototoxic reactions still occur but are rare. **General/respiratory:** Fragrances are volatile and therefore, in addition to skin exposure, a perfume also exposes the eyes and naso-respiratory tract. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma . Asthma-like symptoms can be provoked by sensory mechanisms. In an epidemiological investigation, a significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.

Continued...

LINALOOL & LINALYL ACETATE & GERANYL ACETATE & DIHYDROMYRCENOL	Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A **prehapten** is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems. In the case of prehaptens, it is possible to prevent activation outside the body to a certain extent by different measures, e.g. prevention of air exposure during handling and storage of the ingredients and the final product, and by the addition of suitable antioxidants. When antioxidants are used, care should be taken that they will not be activated themselves and thereby form new sensitisers. **Prehaptens** Most terpenes with oxidisable allylic positions can be expected to autoxidise on air exposure due to their inherent properties. Depending on the stability of the oxidation products that are formed, a difference in the sensitisation potency of the oxidised terpenes can be seen Autoxidation is a free radical chain reaction in which hydrogen atom abstraction in combination with addition of oxygen forms peroxyl radicals. The reaction shows selectivity for positions where stable radicals can be formed. So far, all fragrance substances that have been investigated with regard to the influence of autoxidation on the allergenic potential, including identification of formed oxidation products, have oxidisable allylic positions that are able to form hydroperoxides and/or hydrogen peroxide as primary oxidation products upon air exposure. Once the hydroperoxides have been formed outside the skin they form specific antigens and act as skin sensitisers. Secondary oxidation products such as aldehydes and epoxides can also be allergenic, thus further increasing the sensitisation potency of the autoxidation mixture. The process of photoactivation may also play a role, but further research is required to establish whether this activation route is currently underestimated in importance due to insufficient knowledge of the true haptens in this context. It should be noted that activation of substances via air oxidation results in various haptens that might be the same or cross-reacting with other haptens (allergens). The main allergens after air oxidation of linalool and linalyl acetate are the hydroperoxides. If linalyl acetate is chemically hydrolysed outside the skin it can thereafter be oxidised to the same haptens as seen for linalool. A corresponding example is citronellol and citronellyl acetate. In clincal studies, concomitant reactions to oxidised linalool and oxidised linalyl acetate have been observed. Whether these reactions depend on cross-reactivity or are due to exposure to both fragrance substances cannot be elucidated as both have an allergenic effect themselves. Linalool and linalyl acetate are the main components of lavender oil. They autoxidise on air exposure also when present in the essential oil, and form the same oxidation products found in previous studies of the pure synthetic terpenes. Experimental sensitisation studies showed that air exposure of lavender oil increased the sensitisation potency. Patch test results in dermatitis patients showed a connection between positive reactions to oxidised linalool, linalyl acetate and lavender oil. **Prohaptens** Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohaptens. In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal. The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin . These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohaptens can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity. **QSAR prediction:** The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptens) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptens. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation.
LINALOOL & LINALYL ACETATE & GERANYL ACETATE	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.
LINALYL ACETATE & GERANYL ACETATE & COUMARIN & 4-TERT-BUTYLCYCLOHEXYL ACETATE & TERPINYL ACETATE	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.

Continued...

LINALYL ACETATE & GERANYL ACETATE	Cross-reactivity is also expected between ester derivatives and their parent alcohols, as the esters will be hydrolysed by esterases in the skin. Esters of important contact allergens that can be activated by hydrolysis in the skin are isoeugenol acetate, eugenyl acetate and geranyl acetate all of which are known to be used as fragrance ingredients.

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

Pebeo Gedeo Create Your Own Candles Set B

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

linalool

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	20mg/l	1
LC50	96h	Fish	<19.9mg/l	1
EC50	96h	Algae or other aquatic plants	88.3mg/l	1
NOEC(ECx)	96h	Fish	<3.5mg/l	1

linalyl acetate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	13.1mg/l	2
EC50	48h	Crustacea	10.8mg/l	2
LC50	96h	Fish	11mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	1mg/l	2
EC50	96h	Algae or other aquatic plants	13.1mg/l	2

geranyl acetate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	3.72mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	0.585mg/l	2
EC50	48h	Crustacea	14.1mg/l	2
LC50	96h	Fish	62mg/l	2

coumarin

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

4-tert-butylcyclohexyl acetate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	22mg/l	2
EC50	48h	Crustacea	5.3mg/l	2
LC50	96h	Fish	8.6mg/l	2
EC50(ECx)	48h	Crustacea	5.3mg/l	2

dihydromyrcenol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	65mg/l	2
EC50	48h	Crustacea	38mg/l	2
LC50	96h	Fish	27.8mg/l	2
NOEC(ECx)	96h	Fish	<3.5mg/l	2

terpinyl acetate

Endpoint	Test Duration (hr)	Species	Value	Source

Continued...

	EC50	72h	Algae or other aquatic plants	4.3mg/l	2
	EC50	48h	Crustacea	>10mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	2.7mg/l	2
	LC50	96h	Fish	>11mg/l	2

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

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
linalool	HIGH	HIGH
linalyl acetate	HIGH	HIGH
geranyl acetate	LOW	LOW
coumarin	LOW	LOW
4-tert-butylcyclohexyl acetate	HIGH	HIGH
dihydromyrcenol	HIGH	HIGH
terpinyl acetate	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
linalool	LOW (LogKOW = 2.97)
linalyl acetate	MEDIUM (LogKOW = 3.93)
geranyl acetate	MEDIUM (LogKOW = 4.4754)
coumarin	LOW (LogKOW = 1.39)
4-tert-butylcyclohexyl acetate	MEDIUM (LogKOW = 4.4225)
dihydromyrcenol	LOW (LogKOW = 3.4666)
terpinyl acetate	MEDIUM (LogKOW = 3.96)

Mobility in soil

Ingredient	Mobility
linalool	LOW (Log KOC = 56.32)
linalyl acetate	LOW (Log KOC = 517.9)
geranyl acetate	LOW (Log KOC = 604.3)
coumarin	LOW (Log KOC = 146.1)
4-tert-butylcyclohexyl acetate	LOW (Log KOC = 517.4)
dihydromyrcenol	LOW (Log KOC = 54.78)
terpinyl acetate	LOW (Log KOC = 531.9)

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

Continued...

	▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	•3Z

Land transport (ADG)

14.1. **UN number or ID number**	3082
14.2. **UN proper shipping name**	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains geranyl acetate)
14.3. **Transport hazard class(es)**	Class: 9 Subsidiary Hazard: Not Applicable
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: 274 331 335 375 AU01 Limited quantity: 5 L

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082
are not subject to this Code when transported by road or rail in;
(a) packagings;
(b) IBCs; or
(c) any other receptacle not exceeding 500 kg(L).
- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	3082
14.2. **UN proper shipping name**	Environmentally hazardous substance, liquid, n.o.s. (contains geranyl acetate)
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 9 ICAO / IATA Subsidiary Hazard: Not Applicable ERG Code: 9L
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: A97 A158 A197 A215 Cargo Only Packing Instructions: 964 Cargo Only Maximum Qty / Pack: 450 L

Continued...

Passenger and Cargo Packing Instructions	964
Passenger and Cargo Maximum Qty / Pack	450 L
Passenger and Cargo Limited Quantity Packing Instructions	Y964
Passenger and Cargo Limited Maximum Qty / Pack	30 kg G

Sea transport (IMDG-Code / GGVSee)

14.1. **UN number**	3082
14.2. **UN proper shipping name**	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains geranyl acetate)
14.3. **Transport hazard class(es)**	IMDG Class: 9 IMDG Subsidiary Hazard: Not Applicable
14.4. **Packing group**	III
14.5 **Environmental hazard**	Marine Pollutant
14.6. **Special precautions for user**	EMS Number: F-A , S-F Special provisions: 274 335 969 Limited Quantities: 5 L

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

Not Applicable

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

Product name	Group
linalool	Not Available
linalyl acetate	Not Available
geranyl acetate	Not Available
coumarin	Not Available
4-tert-butylcyclohexyl acetate	Not Available
dihydromyrcenol	Not Available
terpinyl acetate	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
linalool	Not Available
linalyl acetate	Not Available
geranyl acetate	Not Available
coumarin	Not Available
4-tert-butylcyclohexyl acetate	Not Available
dihydromyrcenol	Not Available
terpinyl acetate	Not Available

SECTION 15 Regulatory information

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

linalool is found on the following regulatory lists

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

linalyl acetate is found on the following regulatory lists

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

geranyl acetate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

Continued...

coumarin is found on the following regulatory lists

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

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

Australian Inventory of Industrial Chemicals (AIIC)

dihydromyrcenol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

terpinyl acetate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (linalool; geranyl acetate; coumarin; 4-tert-butylcyclohexyl acetate; dihydromyrcenol; terpinyl acetate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (terpinyl acetate)
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	***Yes = All CAS declared ingredients are on the inventory*** ***No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.***

SECTION 16 Other information

Revision Date	10/03/2023
Initial Date	13/09/2021

SDS Version Summary

Version	Date of Update	Sections Updated
3.1	10/12/2021	Classification change due to full database hazard calculation/update.
4.1	10/03/2023	Classification change due to full database hazard calculation/update.

Other information

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

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

Definitions and abbreviations

Continued...

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

- AIIC: Australian Inventory of Industrial Chemicals
- DSL: Domestic Substances List
- NDSL: Non-Domestic Substances List
- IECSC: Inventory of Existing Chemical Substance in China
- EINECS: European INventory of Existing Commercial chemical Substances
- ELINCS: European List of Notified Chemical Substances
- NLP: No-Longer Polymers
- ENCS: Existing and New Chemical Substances Inventory
- KECI: Korea Existing Chemicals Inventory
- NZIoC: New Zealand Inventory of Chemicals
- PICCS: Philippine Inventory of Chemicals and Chemical Substances
- TSCA: Toxic Substances Control Act
- TCSI: Taiwan Chemical Substance Inventory
- INSQ: Inventario Nacional de Sustancias Químicas
- NCI: National Chemical Inventory
- FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances