Tork Constant Air Freshener Blossom

Essity Australasia

Chemwatch Hazard Alert Code: **2**

Chemwatch: **5689-92**
Version No: **3.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Issue Date: **03/09/2024**
Print Date: **03/10/2024**
S.GHS.AUS.EN

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

Product Identifier

Product name	Tork Constant Air Freshener Blossom
Chemical Name	Not Applicable
Synonyms	Not Available
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Air freshener. Use according to manufacturer's directions. SDS are intended for use in the workplace ONLY. For domestic-use products, refer to consumer labels.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**Essity Australasia**	**Essity Australasia**
Address	30-32 Westall Road SPRINGVALE VIC 3171 Australia	Level 2, 103 Carlton Gore Road Newmarket Auckland 1023 New Zealand
Telephone	(03) 9550 2999	0800 523 565
Fax	1800 630 234	Not Available
Website	https://www.tork.com.au/	https://www.tork.co.nz/
Email	customerservice.anz@essity.com	customerservice.anz@essity.com

Emergency telephone number

Association / Organisation	**Essity Australasia**	**Essity Australasia**	**CHEMWATCH EMERGENCY RESPONSE (24/7)**
Emergency telephone numbers	1800 643 634	0800 523 565	+61 1800 951 288
Other emergency telephone numbers	Not Available	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

COMBUSTIBLE LIQUID, regulated for storage purposes only

Poisons Schedule	Not Applicable
Classification [1]	Flammable Liquids Category 4, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Carcinogenicity Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
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	**Danger**

Hazard statement(s)

H227	Combustible liquid.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H318	Causes serious eye damage.

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H335	May cause respiratory irritation.
H351	Suspected of causing cancer.
H412	Harmful to aquatic life with long lasting effects.
AUH019	May form explosive peroxides.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
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

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.
P302+P352	IF ON SKIN: Wash with plenty of water.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
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
140-11-4	20-<25	benzyl acetate
18479-58-8	5-<10	dihydromyrcenol
18479-51-1	5-<10	1,2-dihydrolinalool
470-82-6	1-<5	eucalyptol
5392-40-5	1-<5	citral
106-30-9	0.25-<1	ethyl heptanoate
67634-14-4	0.25-<1	p-ethyl-alpha,alpha-dimethyldihydrocinnamaldehyde
67634-15-5	0.25-<1	p-ethyl-alpha,alpha-dimethylhydrocinnamaldehyde
68039-49-6	0.25-<1	dimethylcyclohex-3-ene-1-carbaldehyde
106-72-9	0.1-<1	2,6-dimethyl-5-heptenal
6485-40-1	0.1-<1	carvone
97-53-0	0.1-<1	eugenol
112-54-9	0.1-<1	dodecyl aldehyde
112-45-8	0.1-<0.25	10-undecenal
1655500-83-6	0.1-<0.25	rosyfolia
23696-85-7	0.025-<0.1	rose ketones
23726-93-4	0.025-<0.1	alpha-damascone
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: ▸ Immediately hold eyelids apart and flush the eye continuously with running water.

Continued...

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

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.
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 full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ Avoid spraying water onto liquid pools. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	▸ Combustible. ▸ Slight fire hazard when exposed to heat or flame. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke. ▸ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO_2) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes. **WARNING**: Long standing in contact with air and light may result in the formation of potentially explosive peroxides.
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

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

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ **DO NOT** allow clothing wet with material to stay in contact with skin ▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ Avoid smoking, naked lights or ignition sources. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	▸ Store in 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	▸ Glass container is suitable for laboratory quantities

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	▸ Metal can or drum ▸ Packaging as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Ingredient	Original IDLH	Revised IDLH
benzyl acetate	Not Available	Not Available
dihydromyrcenol	Not Available	Not Available
1,2-dihydrolinalool	Not Available	Not Available
eucalyptol	Not Available	Not Available
citral	Not Available	Not Available
ethyl heptanoate	Not Available	Not Available
p-ethyl-alpha,alpha-dimethyldihydrocinnamaldehyde	Not Available	Not Available
p-ethyl-alpha,alpha-dimethylhydrocinnamaldehyde	Not Available	Not Available
dimethylcyclohex-3-ene-1-carbaldehyde	Not Available	Not Available
2,6-dimethyl-5-heptenal	Not Available	Not Available
carvone	Not Available	Not Available
eugenol	Not Available	Not Available
dodecyl aldehyde	Not Available	Not Available
10-undecenal	Not Available	Not Available
rosyfolia	Not Available	Not Available
rose ketones	Not Available	Not Available
alpha-damascone	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
benzyl acetate	E	≤ 0.1 ppm
dihydromyrcenol	E	≤ 0.1 ppm
1,2-dihydrolinalool	D	> 0.1 to ≤ 1 ppm
eucalyptol	E	≤ 0.1 ppm
citral	E	≤ 0.1 ppm
ethyl heptanoate	E	≤ 0.1 ppm
p-ethyl-alpha,alpha-dimethyldihydrocinnamaldehyde	E	≤ 0.1 ppm
p-ethyl-alpha,alpha-dimethylhydrocinnamaldehyde	E	≤ 0.1 ppm
dimethylcyclohex-3-ene-1-carbaldehyde	E	≤ 0.1 ppm
carvone	E	≤ 0.1 ppm
eugenol	E	≤ 0.1 ppm
dodecyl aldehyde	E	≤ 0.1 ppm
10-undecenal	E	≤ 0.1 ppm
rosyfolia	E	≤ 0.1 ppm
rose ketones	E	≤ 0.1 ppm
alpha-damascone	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.***	

Exposure controls

Appropriate engineering controls	**For potent pharmacological agents:** **Solutions Handling:** ▸ Solutions can be handled outside a containment system or without local exhaust ventilation during procedures with no potential for aerosolisation. If the procedures have a potential for aerosolisation, an air-purifying respirator is to be worn by all personnel in the immediate area. ▸ Solutions used for procedures where aerosolisation may occur (e.g., vortexing, pumping) are to be handled within a containment system or with local exhaust ventilation. ▸ In situations where this is not feasible (may include animal dosing), an air-purifying respirator is to be worn by all personnel in the immediate area. If using a ventilated enclosure that has not been validated, wear a half-mask respirator equipped with HEPA cartridges until the enclosure is validated for use. ▸ Ensure gloves are protective against solvents in use. Enclosed local exhaust ventilation is required at points of dust, fume or vapour generation.

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HEPA terminated local exhaust ventilation should be considered at point of generation of dust, fumes or vapours.
Barrier protection or laminar flow cabinets should be considered for laboratory scale handling.
A fume hood or vented balance enclosure is recommended for weighing/ transferring quantities exceeding 500 mg.
When handling quantities up to 500 gram in either a standard laboratory with general dilution ventilation (e.g. 6-12 air changes per hour) is preferred. Quantities up to 1 kilogram may require a designated laboratory using fume hood, biological safety cabinet, or approved vented enclosures. Quantities exceeding 1 kilogram should be handled in a designated laboratory or containment laboratory using appropriate barrier/ containment technology.
Manufacturing and pilot plant operations require barrier/ containment and direct coupling technologies.
Barrier/ containment technology and direct coupling (totally enclosed processes that create a barrier between the equipment and the room) typically use double or split butterfly valves and hybrid unidirectional airflow/ local exhaust ventilation solutions (e.g. powder containment booths). Glove bags, isolator glove box systems are optional. HEPA filtration of exhaust from dry product handling areas is required.
Fume-hoods and other open-face containment devices are acceptable when face velocities of at least 1 m/s (200 feet/minute) are achieved.
Partitions, barriers, and other partial containment technologies are required to prevent migration of the material to uncontrolled areas. For non-routine emergencies maximum local and general exhaust are necessary. 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, 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 (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, 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.)

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 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 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.
The need for respiratory protection should also be assessed where incidental or accidental exposure is anticipated: Dependent on levels of contamination, PAPR, full face air purifying devices with P2 or P3 filters or air supplied respirators should be evaluated.
The following protective devices are recommended where exposures exceed the recommended exposure control guidelines by factors of:
10; high efficiency particulate (HEPA) filters or cartridges
10-25; loose-fitting (Tyvek or helmet type) HEPA powered-air purifying respirator.
25-50; a full face-piece negative pressure respirator with HEPA filters
50-100; tight-fitting, full face-piece HEPA PAPR
100-1000; a hood-shroud HEPA PAPR or full face-piece supplied air respirator operated in pressure demand or other positive pressure mode.

Individual protection measures, such as personal protective equipment

Eye and face protection

When handling very small quantities of the material eye protection may not be required.
For laboratory, larger scale or bulk handling or where regular exposure in an occupational setting occurs:
- Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]
- Face shield. Full face shield may be required for supplementary but never for primary protection of eyes.
- 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

- Elbow length PVC gloves

NOTE:
- The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
· frequency and duration of contact,
· chemical resistance of glove material,
· glove thickness and
· dexterity
Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
· Contaminated gloves should be replaced.

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	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. ▸ Rubber gloves (nitrile or low-protein, powder-free latex, latex/ nitrile). Employees allergic to latex gloves should use nitrile gloves in preference. ▸ Double gloving should be considered. ▸ PVC gloves. ▸ Change gloves frequently and when contaminated, punctured or torn. ▸ Wash hands immediately after removing gloves. ▸ Protective shoe covers. [AS/NZS 2210] ▸ Head covering.
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ P.V.C apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
Tork Constant Air Freshener Blossom

Material	CPI
NATURAL RUBBER	C
NITRILE	C

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

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	Light yellow liquid with fruity smell.

Physical state	Liquid	**Relative density (Water = 1)**	0.998 @20C
Odour	fruity	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available

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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)	67	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Combustible.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	0.054 @20C	**Gas group**	Not Available
Solubility in water	Not Available	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available
Heat of Combustion (kJ/g)	Not Available	**Ignition Distance (cm)**	Not Available
Flame Height (cm)	Not Available	**Flame Duration (s)**	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	**Enclosed Space Ignition Deflagration Density (g/m3)**	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	The material can cause respiratory irritation in some persons. The body's response to such irritation can cause further lung damage. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by sleepiness, reduced alertness, loss of reflexes, lack of co-ordination, 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. Benzyl acetate produces respiratory tract irritation and depressed consciousness. Continued exposure results in kidney damage. It has been reported to cause increased heart rate, low blood pressure and shallow breathing amongst workers. Aliphatic alcohols with more than 3-carbons cause headache, dizziness, drowsiness, muscle weakness and delirium, central depression, coma, seizures and behavioural changes. Secondary respiratory depression and failure, as well as low blood pressure and irregular heart rhythms, may follow. Inhalation of high concentrations of gas/vapour causes lung irritation with coughing and nausea, central nervous depression with headache and dizziness, slowing of reflexes, fatigue and inco-ordination.
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. Overexposure to non-ring alcohols causes nervous system symptoms. These include headache, muscle weakness and inco-ordination, giddiness, confusion, delirium and coma. Long term oral intake of benzyl acetate may cause stunted growth. Brain damage with tremors and kidney damage may occur, according to results of animal testing. Death may occur at very high concentrations.
Skin Contact	The material may accentuate any pre-existing dermatitis condition Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream, through, for example, cuts, abrasions or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man. The material may cause moderate inflammation of the skin either following direct contact or after a delay of some time. Repeated exposure can cause contact dermatitis which is characterised by redness, swelling and blistering.
Eye	If applied to the eyes, this material causes severe eye damage.
Chronic	There has been concern that this material can cause cancer or mutations, but there is not enough data to make an assessment. Long-term exposure to respiratory irritants may result in airways disease, involving difficulty breathing and related whole-body problems. Inhaling this product is more likely to cause a sensitisation reaction in some persons compared to the general population. Skin contact with the material is more likely to cause a sensitisation reaction in some persons compared to the general population. Harmful: danger of serious damage to health by prolonged exposure through inhalation. This material can cause serious damage if one is exposed to it for long periods. It can be assumed that it contains a substance which can produce severe defects. Based on experience with similar materials, there is a possibility that exposure to the material may reduce fertility in humans at levels which do not cause other toxic effects. Based on experience with animal studies, there is a possibility that exposure to the material may result in toxic effects to the development of the foetus, at levels which do not cause significant toxic effects to the mother. Substance accumulation, in the human body, may occur and may cause some concern following repeated or long-term occupational exposure. Cyclic ethers can cause cancers, especially of the liver. Certain substances, commonly found in perfumes or perfumed products, produce hypersensitivity. Contact allergy to perfumes occurs with a relatively high incidence, only exceeded by nickel allergy. There is no cure for perfume allergy. One sensitized, exposure to even extremely small amounts of the perfume gives rise to eruptions and eczema. These symptoms may be treated with steroid creams, although frequent use of steroids produces unwanted side effects.

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Exposure to Aliphatic aldehydes can cause irritation of the skin.
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.
d-Limonene may cause damage to and growths in the kidney. These growths can progress to cancer.
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. This should be less than 10 millimoles of peroxide per litre. This is because peroxides may have sensitizing properties.

Tork Constant Air Freshener Blossom

TOXICITY	IRRITATION
Not Available	Not Available

benzyl acetate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
Oral (Rat) LD50: 2490 mg/kg[2]	Skin (rabbit): 100mg/24h-moderate
	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]

1,2-dihydrolinalool

TOXICITY	IRRITATION
Oral (Rat) LD50: >2000 mg/kg[1]	Not Available

eucalyptol

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
Oral (Rat) LD50: 2480 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]

citral

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Skin (guinea pig): 1%/48h - mod
Oral (Rat) LD50: 4960 mg/kg[2]	Skin (guinea pig):100mg/24hSEVERE
	Skin (human): 40 mg/24h - mild
	Skin (man): 16 mg/48h - SEVERE
	Skin (pig): 50 mg/24h - SEVERE
	Skin (rabbit): 100 mg/24h-SEVERE
	Skin (rabbit): 500 mg/24h - mod

ethyl heptanoate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
Oral (Rat) LD50: >34640 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]

p-ethyl-alpha,alpha-dimethyldihydrocinnamaldehyde

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg[2]	Not Available

p-ethyl-alpha,alpha-dimethylhydrocinnamaldehyde

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg[2]	Not Available

dimethylcyclohex-3-ene-1-carbaldehyde

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 5000 mg/kg[2]	Not Available
Oral (Rat) LD50: 3600 mg/kg[2]	

2,6-dimethyl-5-heptenal

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >=3000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
Oral (Rat) LD50: >5000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

carvone

TOXICITY	IRRITATION
Oral (Guinea) LD50; 766 mg/kg[2]	Not Available

eugenol

TOXICITY	IRRITATION
Oral (Rat) LD50: 1930 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
	Skin (human) 40 mg/24h - mild
	Skin (man): 16 mg/48h - moderate
	Skin (rabbit): 100 mg/24h-SEVERE

Continued...

		Skin: no adverse effect observed (not irritating)[1]
dodecyl aldehyde	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
	Oral (Rat) LD50: 23000 mg/kg[2]	Skin (human): 5 mg/48h - mild
		Skin (rabbit): 500 mg/24h - mod
		Skin: adverse effect observed (irritating)[1]
10-undecenal	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >5000 mg/kg[2]	Skin (rabbit): 500 mg/24h - mild
		Skin: adverse effect observed (irritating)[1]
rosyfolia	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: ~1000~2000 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin : Mild*
		Skin: adverse effect observed (irritating)[1]
rose ketones	**TOXICITY**	**IRRITATION**
	Oral (Mouse) LD50; 1821 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
alpha-damascone	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >1000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Mouse) LD50; 1821 mg/kg[2]	Skin: adverse effect observed (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***

BENZYL ACETATE	Neoplastic by RTECS criteria For certain benzyl derivatives: The members of this group are rapidly absorbed through the gastrointestinal tract, metabolised primarily in the liver, and excreted primarily in the urine either unchanged or as conjugates of benzoic acid derivatives. At high dose levels, gut micro-organisms may act to produce minor amounts of breakdown products. However, no adverse effects have been reported even at repeated high doses. Similarly, no effects were observed on reproduction, foetal development and tumour potential. This is a member or analogue of a group of benzyl derivatives generally regarded as safe (GRAS), based partly on their self-limiting properties as flavouring substances in food. In humans and other animals, they are rapidly absorbed, broken down and excreted, with a wide safety margin. They also lack significant potential to cause genetic toxicity and mutations. The intake of benzyl derivatives as natural components of traditional foods is actually higher than the intake as intentionally added flavouring substances. Aryl alkyl alcohol simple acid ester derivates (AAASAE) have a low level of acute toxicity. Repeat-dose toxicity tests did not show significant toxicity. Testing did not show any evidence of AAASAE to have potential to cause cancer, mutations or genetic toxicity. At expected exposure levels, there is no evidence that AAASAE causes adverse effects on reproduction or development. In general there are currently no safety concerns regarding AAASAE at current levels of use and exposure. The aryl alkyl alcohol (AAA) fragrance ingredients have diverse chemical structures, with similar metabolic and toxicity profiles. The AAA fragrances demonstrate low acute and subchronic toxicity by skin contact and swallowing. At concentrations likely to be encountered by consumers, AAA fragrance ingredients are non-irritating to the skin. The potential for eye irritation is minimal. With the exception of benzyl alcohol, phenethyl and 2-phenoxyethyl AAA alcohols, testing in humans indicate that AAA fragrance ingredients generally have no or low sensitization potential. Available data indicate that the potential for photosensitization is low. Testing suggests that at current human exposure levels, this group of chemicals does not cause maternal or developmental toxicity. Animal testing shows no cancer-causing evidence, with little or no genetic toxicity. It has been concluded that these materials would not present a safety concern at current levels of use, as fragrance ingredients.
1,2-DIHYDROLINALOOL	With few exceptions* (see below), there are no safety concerns regarding certain cyclic and non-cyclic terpene alcohols **, as fragrance ingredients, under present declared levels of use and exposure, because - They have low acute toxicity - No significant toxicity was observed in repeat dose toxicity tests - They were not found to cause mutations or genetic toxicity - Substances in this group are processed similarly in the body - There is no indication of persistent breakdown products causing severe toxicity - They practically do not irritate the skin - They have a generally low potential for sensitization - The margin of safety is more than 100 times the maximum daily exposure. *Safety concerns exist for the following substances for the following reasons: - 6,7-dihydrogeraniol, hydroabietyl alcohol and 2-isopropyl-2-decahydronapthalenol are potent skin sensitisers. - Farnesol is a weak sensitizer. - Scalerol and linalool may contain impurities and/or oxidation products that are strong sensitisers. - 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
EUCALYPTOL	d-Limonene is readily absorbed by inhalation and swallowing. Absorption through the skin is reported to the lower than by inhalation. It is rapidly distributed to different tissues in the body, readily metabolized and eliminated, primary through the urine.

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	Limonene shows low acute toxicity by all three routes in animals. Limonene is a skin irritant in both experimental animals and humans. Limited data is available on the potential to cause eye and airway irritation. Autooxidised products of d-limonene have the potential to sensitise the skin. Limited data is available on the potential to cause respiratory sensitization in humans. Limonene will automatically oxidize in the presence of light in air, forming a variety of oxygenated monocyclic terpenes. When contact with these oxidation products occurs, the risk of skin sensitization is high. Limonene does not cause genetic toxicity of birth defects, and it is not toxic to the reproductive system.
CITRAL	Produces maternal effects (oogenesis, ovaries, fallopian tube changes) and effects live-birth index. A member or analogue of a group of aliphatic, linear alpha,beta-unsaturated aldehydes and structurally related substances These substances are generally regarded as safe. They are found in flavouring substances in food and are rapidly absorbed and broken down in the body. for citral Citral is rapidly absorbed from the gastrointestinal tract. Much of an applied dermal dose is lost due to its extreme volatility, but the citral remaining on the skin was fairly well absorbed. Citral is rapidly metabolised and excreted as metabolites.Urine is the major route of elimination. **Acute toxicity** of this chemical is low in rodents because the oral or dermal LD50 values were more than 1000 mg/kg. This chemical is irritating to skin and not irritating to eyes in rabbits. There is some evidence that this chemical is a human skin sensitiser. **Repeat dose toxicity:** Several repeated dose oral studies show no adverse effect of citral at less than 1,000 mg/kg/day exposure and some histological changes in the nasal cavity or forestomach, the first exposure sites, probably due to irritation, at more than 1,000 mg/kg/day. Male and female F344/N rats received microencapsulated citral in feed at concentrations of 0, 0.63, 1.25, 2.5, 5 and 10% (resultant doses: 0, 142, 285, 570, 1,140 and 2,280 mg/kg/day) for 14 days. Minimal to mild hyperplasia and/or squamous metaplasia of the respiratory epithelium was observed in nasal cavity without inflammatory response at 1,140 and 2,280 mg/kg/day of both sexes. The NOAEL was established at 570 mg/kg/day. In an OECD preliminary reproduction toxicity screening test [TG 421], citral was administered to Crj:CD (SD) rats by gavage at doses of 0, 40, 200 and 1,000 mg/kg/day in males for 46 days and in females for 39-50 days including before and through mating and gestation periods and until day 3 of lactation. Squamous hyperplasia, ulcer and granulation in lamina propria were observed in the forestomach at 1,000 mg/kg/day of both sexes. Therefore, the NOAEL for repeated dose toxicity was 200 mg/kg/day for both sexes. **Developmental toxicity**: in the above preliminary reproductive study, no effects were detected in reproductive ability, organ weights or histopathology of the reproductive organs of both sexes, and delivery or maternal behavior. However, body weights of male and female pups were reduced in the 1000 mg/kg group. Therefore, an oral NOAEL for developmental toxicity was 200 mg/kg/day. In a teratogenicity study, SD pregnant rats were exposed to citral by inhalation for 6 hr/day on gestation days 6-15 at mean concentration of 0, 10 or 34 ppm as vapour, or 68 ppm as an aerosol/vapour mixture. Even in the presence of the maternal effects, no significant teratogenicity was noted at 68 ppm. An inhalation NOAEL of teratogenicity was established at 68 ppm (423 mg/m3). **Genotoxicity:** Seven bacterial reverse mutation studies indicate negative results with and without metabolic activation. As for non-bacterial in vitro study, two chromosomal aberration results in Chinese hamster cells are negative however one positive result in sister chromatid exchange is given in the same cells. Additionally, two in vivo micronucleus tests in rodents indicate negative results. Based on the above information, the genotoxic potential of citral can be considered to be negative. **Carcinogenicity:** A NTP study shows that there was no evidence of carcinogenic activity in male/female rats and male mice but some evidence of malignant lymphoma in female mice (up to 4,000 ppm in feed in rats and up to 2,000 ppm in feed in mice). Dermal application of citral induces prostate hyperplasia with low severity only in some strains of rats. However, the NTP oral carcinogenicity studies in rats and mice found no evidence of lesions (neoplastic or non-neoplastic) in any male reproductive organ, including the prostate. The health significance of the effects seen in the dermal studies in rats is uncertain due to dramatic strain differences and it is noted that the work has primarily been performed in a single laboratory. For dienaldehydes: Dienaldehydes are by-products of peroxidation of polyunsaturated lipids and commonly found in many foods or food-products. Both National Cancer Institute (NCI) and NTP have expressed great concern on the potential genotoxicity and carcinogenicity of dienaldehydes. 2,4-Decadienal and 2,4-hexadienal are autooxidation products of polyunsaturated fatty acids Several researchers have implied there could be a link between exposures to lipid peroxidation products in the diet and the development of human cancers. Lipid hydroperoxides have been shown to give rise to low intracellular levels of 2,4-decadienal and other alpha-beta-unsaturated aldehydes that are known to be reactive with DNA. Ingested lipid oxidation products and oxidized fats have been reported to cause increased excretion of mutagens, cellular injury to liver and kidneys, increased cell proliferation in the gastrointestinal tract, and other nonspecific tissue injury and irritation effects resulting from induced oxidative stress. Treatment related changes following gastric lavage administration for up to 3 months were similar for 2,4-hexadienal and 2,4-decadienal, and in both cases the forestomach and nose were identified as target organs, In two week studies of 2,4-hexadienal and 2,3 decadienal in rats and mice, forestomach lesions included necrosis and ulceration;epithelial hyperplasia was observed in rats and mice in the 2,4-hexadienal studies. In the 3-month studies of 2,4-hexadienal and 2,4-decadienal, forestomach epithelial hyperplasia and degeneration with or without chronic active inflammation occurred i addition to nasal olfactory epithelia atrophy and necrosis. Carcinogenicity and mutagenicity data from testing of dienals are limited. In the two year carcinogenicity studies, 2,4-hexadienal induced significantly increased incidences of forestomach neoplasms in rats and mice. NTP Technical Report 2,4-decadienal Trans, trans-2,4-decadienal (tt-DDE or 2,4-De), a specific type of dienaldehyde, is abundant in heated oils and has been associated with lung adenocarcinoma development in women due to their exposure to oil fumes during cooking. Cultured human bronchial epithelial cells (BEAS-2B cells) were exposed to 0.1 or 1.0 uM tt-DDE for 45 days, and oxidative stress, reactive oxygen species (ROS) production, GSH/GSSG ratio, cell proliferation, and expression of TNFalpha and IL-1beta were measured. The results show that tt-DDE induced oxidative stress, an increase in ROS production, and a decrease in GSH/GSSG ratio (glutathione status) in a dose-dependent manner. Treatment of BEAS-2B cells with 1.0 uM tt-DDE for 45 days increased cell proliferation and the expression and release of pro-inflammatory cytokines TNFalpha and IL-1beta. Cotreatment of BEAS-2B cells with antioxidant N-acetylcysteine prevented tt-DDE-induced cell proliferation and release of cytokines. Therefore, these results suggest that tt-DDE-induced changes may be due to increased ROS production and enhanced oxidative stress. Since increased cell proliferation and the release of TNF-alpha and IL-1beta are believed to be involved in tumor promotion, these results suggest that tt-DDE may play a role in cancer promotion. Previous studies on dienaldehydes have focused on their genotoxic or carcinogenic effects in the gastrointestinal tract; the present study suggests a potential new role of tt-DDE as a tumor promoter in human lung epithelial cells. Trans, Trans-2,4-Decadienal, a Product Found in Cooking Oil Fumes, Induces Cell Proliferation and Cytokine Production Due to Reactive Oxygen Species in Human Bronchial Epithelial Cells Louis W. Chang Wai-Sze Lo Pinpin Lin Toxicological Sciences, Volume 87, Issue 2, 1 October 2005, Pages 337–343, https://doi.org/10.1093/toxsci/kfi258 2,4-Decadienal is produced by the oxidation of linoleic acid. 2,4-Decadienal is found as a contaminant in water.It is generated from polyunsaturated fatty acids by the action of plant lipoxygenases and is produced in mammalian tissues in certain physiological and pathophysiological processes such as lipid peroxidation, arachidonic acid oxidation, and reactions with reactive oxygen species Alpha,beta-unsaturated aldehydes and ketones are potentially genotoxic. It is believed that nucleophilic sites in DNA react through a 1,4-nucleophilic addition (Michael reaction) with alpha,beta-unsaturated carbonyl compounds. The flavour industry provided genotoxicity studies for the substance 4,5-epoxydec-2(trans)-enal. Based on these data, a European Food Safety Authority (EFSA Panel concluded that 4,5-epoxydec-2(trans)-enal did not induce gene mutations in bacterial cells but was positive in an in vitro micronucleus assay, so, 4,5-epoxydec-2(trans)-enal is considered an in vitro genotoxic agent. The negative results obtained in an in vivo micronucleus assay cannot overrule the positive results of the in vitro micronucleus assay with and without S9-mix due to the lack of demonstration of bone marrow exposure. Following this, the flavour industry has provided plasma analysis of a satellite group of rats treated with 4,5-epoxydec-2(trans)-enal in order to

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	investigate the systemic exposure of animals in the in vivo micronucleus assay. However, the plasma analysis did not provide enough evidence of target tissue exposure. An in vivo Comet assay in rodents was recommended in order to investigate possible genotoxic effects at the first site of contact (e.g. stomach/duodenum cells) and in the liver. An in vivo Comet assay in liver and duodenum was provided that suggests that 4,5-epoxydec-2(trans)-enal did not induce DNA damage in the duodenum of rats. However, the genotoxic effect observed in vitro was confirmed in the in vivo Comet assay in the liver of rats. The Panel concluded that 4,5-epoxydec-2(trans)-enal does raise a safety concern with respect to genotoxicity **Scientific Opinion on Flavouring Group Evaluation 226 Revision 1 (FGE.226Rev1): consideration of genotoxicity data on one alpha,beta-unsaturated aldehyde from chemical subgroup 1.1.1(b) of FGE.19; May 2017** https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2017.4847
P-ETHYL-ALPHA,ALPHA-DIMETHYLDIHYDROCINNAMALDEHYDE	* The Good Scents Company After reviewing all available data on the related esters and alcohols of cinnamic acid and cinnamyl alcohol, and on their parent materials, cinnamyl alcohol, cinnamaldehyde and cinnamic acid, it was found that there are unlikely to be safety concerns regarding these materials as fragrance ingredients, under present conditions of use and exposure for the following reasons: Acute toxicity: Studies show that these materials have low to moderate toxicity if given by mouth, a low toxicity by skin contact. Subchronic toxicity: Studies show that toxicity is very unlikely at levels absorbed by humans from their use as fragrance ingredients. Genetic toxicity: Available evidence shows that these substances are unlikely to cause genetic toxicity. Irritation and sensitization: In human studies, allyl cinnamate has the potential to produce irritation; with the remaining cinnamyl materials, no irritation was observed at concentrations of under 10%. It is not expected that these materials have potential to cause light-mediated toxicity or allergy. These substances are generally regarded as safe. Cinnamyl derivatives are natural components of certain foods, and are found in greater amounts there than in flavouring substances. They are rapidly absorbed, broken down and eliminated in the human body, and do not have significant potential to cause genetic toxicity and mutations.
CARVONE	for (+/-)-carvone for (R)-(-)-carvone [RTECS No.: OS 8650000] Carvone substances have been reported to occur naturally in foods, including fruits, spices, and berries. Currently there are no safety concerns for any of the carvones based on current levels of intake. Following short term intake, increases in serum cholesterol and lipid concentrations have been reported in rats, as well as decreases in food consumption and body weight. Repeated intake may also cause shrinking of the testes. They appear to be protective against liver, lung, stomach and intestine tumours in rats.
EUGENOL	Equivocal tumorigen by RTECS criteria Allergic reactions involving the respiratory tract are usually due to interactions between IgE antibodies and allergens and occur rapidly. Allergic potential of the allergen and period of exposure often determine the severity of symptoms. Some people may be genetically more prone than others, and exposure to other irritants may aggravate symptoms. Allergy causing activity is due to interactions with proteins. Attention should be paid to atopic diathesis, characterised by increased susceptibility to nasal inflammation, asthma and eczema. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure. Reported side effects of leukotriene modifiers (inhibitors and receptor antagonists) include · agitation · aggression · anxiety · dream abnormalities · hallucinations · depression · insomnia · irritability · restlessness · suicidal thinking and behavior · tremor Neuropsychiatric events were not commonly observed, however, in March 2020, the FDA did require that the manufacturer of montelukast include a new boxed warning to inform people of the risk of serious behavioral and mood changes. This includes increased suicidal thoughts and actions. For eugenol: The acute toxicity of eugenol is low. High doses may cause damage to the stomach lining, bleeding, inflammation of the stomach, liver discoloration and congestion in animals. Eugenol is readily absorbed through the skin; products containing eugenol or clove oil may irritate the skin and eyes. Inhalation may be a substantial route of exposure. Eugenol relaxes the blood vessels, causing low blood pressure and a slow heart rate. Animal testing shows that eugenol can cause genetic damage. It is believed that it does not cause cancer, and may even reduce the cancer-causing effect of certain other substances. Incidents of liver injury or failure among modern antifungal medicines are very low to non-existent. However, some can cause allergic reactions in people.[There are also many drug interactions. Patients must read in detail the enclosed data sheet(s) of any medicine. For example, the azole antifungals such as ketoconazole or itraconazole can be both substrates and inhibitors of the P-glycoprotein, which (among other functions) excretes toxins and drugs into the intestines.] Azole antifungals also are both substrates and inhibitors of the cytochrome P450 family CYP3A4,[] causing increased concentration when administering, for example, calcium channel blockers, immunosuppressants, chemotherapeutic drugs, benzodiazepines, tricyclic antidepressants, macrolides and SSRIs.[35] Before oral antifungal therapies are used to treat nail disease, a confirmation of the fungal infection should be made.[Approximately half of suspected cases of fungal infection in nails have a non-fungal cause.[The side effects of oral treatment are significant and people without an infection should not take these drugs.[Azoles are the group of antifungals which act on the cell membrane of fungi. They inhibit the enzyme 14-alpha-sterol demethylase, a microsomal CYP, which is required for biosynthesis of ergosterol for the cytoplasmic membrane. This leads to the accumulation of 14-alpha-methylsterols resulting in impairment of function of certain membrane-bound enzymes and disruption of close packing of acyl chains of phospholipids, thus inhibiting growth of the fungi. Some azoles directly increase permeability of the fungal cell membrane. vntifungal resistance is a subset of antimicrobial resistance, that specifically applies to fungi that have become resistant to antifungals. Resistance to antifungals can arise naturally, for example by genetic mutation or through aneuploidy. Extended use of antifungals leads to development of antifungal resistance through various mechanisms. Some fungi (e.g. Candida krusei and fluconazole) exhibit intrinsic resistance to certain antifungal drugs or classes, whereas some species develop antifungal resistance to external pressures. Antifungal resistance is a One Health concern, driven by multiple extrinsic factors, including extensive fungicidal use, overuse of clinical antifungals, environmental change and host factors.] Unlike resistance to antibacterials, antifungal resistance can be driven by antifungal use in agriculture. Currently there is no regulation on the use of similar antifungal classes in agriculture and the clinic. The emergence of Candida auris as a potential human pathogen that sometimes exhibits multi-class antifungal drug resistance is concerning and has been associated with several outbreaks globally. The WHO has released a priority fungal pathogen list, including pathogens with antifungal resistance For G-protein inhibitors:/ antagonists/ modulators. G protein-coupled receptors (GPCRs) are essential cell membrane signaling molecules and represent the most important class of drug targets. Some signaling pathways downstream of a GPCR may be responsible for drug adverse effects, while others mediate therapeutic efficacy. Biased ligands preferentially activate only a subset of all GPCR signaling pathways. They hold great potential to become next-generation GPCR drugs with less side effects due to their potential to exclusively activate desired signaling pathways.

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	GPCR ligands include odorants, tastants, and neurotransmitters, and vary in size and properties. Dramatic chemical diversity may occur even among ligands of the same receptor. Chemical variability of antagonists significantly correlates with the binding site hydrophobicity and anti-correlates with the number of hydrogen bond donors in the binding site. The number of disulfide bridges in the extracellular region of a receptor anti-correlates with the range of molecular weights of its antagonists, highlighting the role of the entrance pathway in determining the size selectivity for GPCR antagonists. The number of protein targets included in the cross-pharmacology profile of the different GPCRs changes significantly upon varying the ligand similarity and binding affinity criteria. However, with the exception of muscarinic receptors, aminergic GPCRs distinguish themselves from the rest of the members in the family by their remarkably high levels of pharmacological similarity among them. GPCRs are classified under the GRAFS system (Metabotropic Glutamate, Rhodopsin, Adhesion, Frizzled/taste2/Smoothened and Secretin), with therapies having been developed for about 30 GPCRs from the glutamate, rhodopsin and secretin families. GPCR signaling requires significant conformational changes within the trans-membrane TM domain, triggered by agonist binding, and is often coupled to interactions from the extracellular domains or loops.It is becoming clear that many binding sites and mechanisms exist for positive and negative allosteric regulation, and for biased signaling pathways, likely in greater numbers than seen in most other protein systems. When GPCRs are exposed to a neutral agonist, such as morphine on mu-opioid receptor, an occupied receptor can generate several signal waves (non-biased agonist). In GPCR signaling, the ability of a molecule to selectively activate one pathway without affecting another pathway is called biased agonism. Biased signaling occurs at different signaling proteins, including G proteins, GRKs, beta-arrestins, and even at levels of the allosteric binding site. Since GPCR activation-induced two distinct signal waves, G protein-dependent signaling followed by beta-arrestin-dependent signaling opens a new promising therapeutic future in the world of GPCRs. This is true since discovering such molecules dramatically lowers the adverse effects by turning off unwanted signals. For example, the analgesic effect of morphine (neutral agonist) through the activation of u-receptors is accompanied by several side effects, including constipation, respiratory depression, tolerance, nausea, and sedation Despite the long history and obvious desirability of developing drugs targeting GPCRs, there are several problems associated with their development. For example, the muscarinic M1 receptor is a well-validated target for agonists that could alleviate cognitive decline during neurodegeneration . Muscarinic acetylcholine receptors (MRs, or mAChRs), which are more sensitive to muscarine than to nicotine, are a group of class A GPCRs comprising five distinct subtypes, named as muscarinic M1, M2, M3, M4, and M5 receptors (M1R-M5R) M1R, M3R, and M5R are coupled to the Gq/11 family of G proteins, whereas M2R and M4R are coupled to the Gi/o family of G proteins. However, the orthosteric binding site of M1 is virtually identical to those of the related receptors M2,M3, M4, and M5 as they all bind the native ligand acetylcholine, and activation of M2 and M3 in particular gives rise to dose-limiting side effects (gastrointestinal [GI] disturbances, cardiovascular effects). Atropine and other anticholinergic agents exert their bronchodilator effects through the blockade of MRs in the airways. As a tertiary ammonium derivative, atropine is a nonselective antagonist with similar affinity for all of the MR subtypes The half-life of atropine for M3R residence is 3.5 hours. Although extensively used in the past, atropine is rarely used at the present time because it is well absorbed into the systemic circulation and penetrates the blood–brain barrier, leading to multiple systemic side effects, including tachycardia. Several long-acting muscarinic antagonists (LAMAs) are under investigation or are available for the treatment of obstructive airway diseases. LAMAs are considered to be safe drugs at recommended dosages. However, because MRs are expressed not only in the lungs, but also in the heart and the digestive and urinary tracts, the blockade of different MR subtypes in these organs by LAMA treatment can cause diverse, unwanted physiologic effects. For example, these agents can initially block prejunctional M2R on cholinergic airway nerves that normally reduce the release of the bronchoconstricting neurotransmitter acetylcholine, thus resulting in cough and paradoxical bronchoconstriction. Side effects including cardiovascular morbidity and mortality of inhaled LAMA agents in asthma need to be further studied and defined. Another potential source of side effects when targeting other receptors could arise due to signaling through multiple different pathways There are multiple signaling pathways for GPCRs, and it is sometimes possible to bias the signaling of a given GPCR through either a specific G protein or through beta arrestin which could reduce the side effects of some drugs Targeting G protein alpha-subunits has the potential for pleiotropic effects and could result in multiple side effects. Particular targets of concern include ion channels such as the G protein-activated inward rectifier K+ channel (GIRK) and the N-type voltage-gated calcium channels. Gbeta-gamma activates GIRK channels in neurons and in atria, leading to a hyperpolarization-induced decrease in action potential firing. Therefore, when considering the use of Gbeta-gamma inhibitors in cardiac or immune therapy, interfering with the regulation of action potentials would have highly undesirable side effects, such as arrhythmias. However, empirical data using prototypical Gbeta-gamma blockers indicate that these pathways are unaffected by Gbeta-gamma inhibitors, and animals treated with gallein show no signs of arrhythmias or alterations in heart rate.
DODECYL ALDEHYDE	For n-alkyl aldehydes: Acute toxicity hazard of the n-alkyl aldehyde cluster members is moderate via inhalation and low via oral and dermal routes of exposure. Cluster members have been shown to be eye and skin irritants, but not skin sensitisers. Positive results for genotoxicity were reported for cluster members with lower molecular weights (<100), while members with molecular weights > 100 were negative, with the exception of nonanal (124-19-6). Although cancer bioassay data are not available for this cluster, several members of this cluster are considered potential carcinogens due to structural analogy to their carcinogenic lower homologs, acetaldehyde and formaldehyde . The primary metabolism of linear saturated aliphatic aldehydes and acids is a fundamental part of cell biochemistry. Aldehydes are successively oxidized to their corresponding carboxylic acids. To a minor extent, aldehydes also may be reduced to alcohols or conjugated with labile sulfhydryl-containing substances, such as glutathione In general, the inhalation route is expected to be of higher concern than the oral or dermal route because of rapid oxidation of the reactive aldehyde group to the relatively innocuous acid. However, individuals with genetic deficiency of aldehyde dehydrogenase may still be susceptible via the oral route.
ROSYFOLIA	Dermal (Rat) LD50: 1000-2000 mg/kg* Eye : Mild* Mouse, skin sensitisation – local lymph node assay evidence of sensitisation (EC3 = 62.5%) Rat, repeat dose oral toxicity – 28 days NOAEL = 3000 ppm Mutagenicity – bacterial reverse mutation non mutagenic Genotoxicity – in vitro mammalian chromosome aberration test non genotoxic Toxicokinetics Based on the low molecular weight (500 Da), water solubility (7 × 10-2 mg/L at 20 ºC) and partition coefficient (log Pow = 3.5 at 35 °C) of the notified chemical, there is potential for the chemical to cross biological membranes. Acute toxicity The n chemical was found to be of low toxicity via the oral route in a study conducted in rats. The chemical was found to be harmful via the dermal route in a study conducted in rats. Three of five female animals and one of five male animals treated with 2,000 mg/kg bw died. There were no mortalities for animals treated with 1,000 mg/kg bw. As female is generally the appropriate sex of test animals for the OECD test, the LD50 is therefore considered to be 1,000 – 2,000 mg/kg bw. Irritation In an in vitro skin irritation study conducted using the reconstructed human epidermis model (EpiSkin™), the chemical was determined to be irritating to the skin. In an in vitro bovine corneal opacity and permeability (BCOP) test the notified chemical was determined to be irritating to eyes. Although no prediction on the classification was made in the eye irritation study, the notified chemical is classified as H319 - Causes serious eye irritation by the notifier. Sensitisation The notified chemical was a skin sensitiser in mice (local lymph node assay: stimulation indices of 2.2, 2.7 and 3.9 at 25%, 50% and 100%, respectively). The EC3 value was calculated to be 62.5%. Repeated dose toxicity A repeated dose oral (diet) toxicity study on the notified chemical was conducted in rats, in which the test substance was administered at 1,000 ppm (equivalent to 98 mg/kg bw/day for both sexes), 3,000 ppm (equivalent to 296 mg/kg bw/day for males and 300 mg/kg bw/day for females) and 10,000 ppm (equivalent to 1,011 mg/kg bw/day for males and 944 mg/kg bw/day for females) for 28 consecutive days, with a 14-day recovery period for high dose and control animals. The No Observed Adverse Effect Level (NOAEL) was established as 3,000 ppm (equivalent to 296 mg/kg bw/day for males and 300 mg/kg bw/day for females) in this study based on morphological changes in the kidney of males (tubular degeneration, papillary cysts, tubular dilation and hyperplasia of the pelvic urothelium) in the high dose group. Mutagenicity/Genotoxicity The notified chemical was negative in a bacterial reverse mutation assay and in an in vitro mammalian chromosome aberration test in human lymphocytes *Nicas public report LTD/1926 march 2017

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ROSE KETONES	Inhibition of NF-kB in vivo can be detrimental. NF-kB controls multiple functions in homeostasis including a functional immune response, cell cycle, and cell death. Genetic studies in mice and analysis of naturally occurring mutations in humans point to specific developmental and immune consequences due to altering NF-kB activity. The same functions that make NF-kB attractive for developing inhibitors for treating disease also play a role in homeostasis,and disruption of the NF-kB pathway during development or in adults leads to unfavorable and potentially unhealthy consequences. NF-kB plays a role in multiple homeostatic cellular processes including response to stimuli,cell proliferation, and death, regulating communication between cells, but is also tightly linked with other signaling pathways within the cell, such a p38 and JNK. In addition to mediating proinflammatory responses, NF-kB may regulate apoptotic and cell cycle changes induced by cellular stress, DNA damage or oncogenes by communication with the tumor suppressor p53. Disruption of normal cellular responses by inhibiting NF-kB can have adverse consequences such as immune suppression and tissue damage. Understanding the consequences of lack of NF-kB activity in adult humans comes from observation of naturally occurring genetic deficiencies in this pathway. Mutations have been discovered in humans in signaling molecules upstream of NF-kB resulting in defects in development or immunity. Genetic defects have also been discovered in genes that immediately affect NF-kB activation including IKK gamma (NEMO), a subunit of the IKK complex, and IkBalpha.The IKK gamma mutations result in a defective IKK complex and the IkBalpha mutation results in an IkBalpha protein that cannot be phosphorylated and degraded. Both genetic defects result in suppressed NF-kB activation and ectodermal dysplasia with immunodeficiency.In general patients with these genetic defects have multiple immunological defects including impaired innate immunity,impaired antibody production, and ultimately severe bacterial infections. Understanding the immune defects and susceptibilities in patients with genetic defects in the NF-kB pathway will help prepare for potential adverse effects of pharmacologic NF-kB inhibitors The requirement for NF-kB in the development and maintenance of the immune system is well documented. NF-kB is required for survival during fetal development and for normal lymphocyte generation in adult mice. Removal of the p65 (RelA) subunit of NF-kB or the IKKbeta gene results in death during fetal development primarily due to massive liver apoptosis Fetal liver stem cells from p65 or IKKbeta deficient mice have been transplanted into irradiated hosts revealing a specific requirement of NF-kB for T-cells, B-cells, and common lymphoid progenitor development but not for myeloid cells or stem cells. The failure to produce lymphocytes is mediated through hypersensitivity to TNF due to lack of NF-kB activity. Lymphocyte depletion with chemical or genetic inhibition of NF-kB have implications for therapeutic potential use in humans. The double-sided nature of NF-kB inhibition is clear in this instance where chemical inhibition in vivo mimics genetic experiments inducing rapid TNF-dependent apoptosis. Rapid induction of apoptosis may be an advantage for treating some forms of cancer, but at the same time cause depletion of some lymphocyte populations. In addition to controlling lymphocyte development, NF-kB plays a major role in both adaptive and innate immunity. Various signaling pathways responding to receptor recognition of immune challenge converge on NF-kB which then regulates genes that control the immune response. Both T-cell receptor and B-cell receptors activate NF-kB through phosphorylation of CARMA1 by PKC theta and PKC beta respectively, resulting in recruitment and activation of IKK and ultimately expression of genes that control cellular activation, proliferation, and survival. In addition, NF-kB plays a role in T-cell response to costimulatory signals. Cells respond to pathogenic microorganisms in part through recognition by Toll-like receptors (TLRs).TLR-family members recognize different molecular structures present in microbes and respond by activating signaling pathways including NF-kB leading to expression of anti-microbial effector molecules, as well as molecules that help in development of the adaptive immune response. Inhibition of NF-kB during TLR stimulation can lead to macrophage apoptosis, a mechanism used by some pathogens to help evade immune response. NF-kB is clearly required for normal mature B-cell and T-cell maintenance and function, including regulatory, memory, and natural killer-like T cells. Inhibition of NF-kB activation in lymphocytes results in defects in growth, survival, and cytokine production and blocks multiple steps in germinal center formation.Given the diverse roles NF-kB plays in immune response to pathogens it is not surprising to find mice genetically deficient in components of the NF-kB pathway are susceptible to parasitic and bacterial infection. The role of NF-kB in inhibition of apoptosis is one of the factors that make it a potential target for cancer therapy. NF-kB deficient mice die during embryogenesis in part due to TNF-mediated liver damage. Adult mice with impaired NF-kB targeted to the liver have normal liver function, but have severe liver damage after challenge with concanavalin A, a pan-T cell activator.Liver damage occurs due to sustained activation of JNK due to accumulation of reactive oxygen species (ROS) in the absence of normal NF-kB activation. A member or analogue of EFSA Chemical Group 10 secondary aliphatic saturated or unsaturated alcohols, ketones, ketals and esters with a secondary or tertiary oxygenated functional group used as flavourings No safety concern would arise for the consumer from the use of these compounds up to the highest proposed level in feeds. Hazards for skin and eye contact and respiratory exposure are recognised for the majority of the compounds under application. Most are classified as irritating to the respiratory system. Aliphatic acyclic and alicyclic alpha-diketones and alpha-hydroxyketones are generally used as flavouring agents up to average maximum levels of 200 ppm. In rats and mice, orally administered aliphatic alpha-diketones are rapidly absorbed from the gastrointestinal tract. It is anticipated that at low levels of exposure, humans will metabolize aliphatic acyclic alpha-diketone principally by alpha-hydroxylation and subsequent oxidation of the terminal methyl group to yield the corresponding ketocarboxylic acid. The acid may undergo oxidative decarboxylation to yield carbon dioxide and a simple aliphatic carboxylic acid, which may be completely metabolized in the fatty acid pathway and citric acid cycle. At high concentrations, another detoxification pathway is used which involves reduction to the diol and subsequent conjugation with glucuronic acid. Acyclic alpha-diketones and alpha-hydroxyketones without a terminal methyl group and alicyclic diketones and hydroxyketones are mainly metabolized by reduction to the corresponding diol, followed by glucuronic acid conjugation and excretion Compounds belonging to CG 10 are absorbed from the gastrointestinal tract and share common pathways of metabolism: (i) hydrolysis of esters by carboxylesterases, (ii) reduction of ketones to alcohols, (iii) oxidation of alcohols to acids, (iv) alpha–hydroxylation of the terminal methyl group to yield corresponding ketocarboxylic acids, (v) oxidative decarboxylation to yield carbon dioxide and an aliphatic carboxylic acid, and (vi) conjugation of alpha-hydroxyketones or their diol metabolites with glucuronic acid. Aliphatic acyclic diketones and alpha–hydroxyketones, which contain a carbonyl function at the 2-position (i.e. a methyl ketone) are expected to undergo alpha–hydroxylation and subsequent oxidation of the terminal methyl group to eventually yield corresponding ketocarboxylic acids. These compounds are intermediary metabolites (e.g.alpha-ketoacids), which may undergo oxidative decarboxylation to yield carbon dioxide and an aliphatic carboxylic acid. The acid is then metabolised via beta-oxidation and the citric acid cycle. beta-Ketoacids and derivatives readily undergo decarboxylation to yield breakdown products, which are incorporated into normal biochemical pathways. Alternatively, the methyl-substituted diketones may be successively reduced to the corresponding hydroxyketones and diols, which are excreted in the urine as glucuronic acid conjugates. This pathway is favoured at elevated in vivo concentrations, especially for longer chain length ketones. If the carbonyl function is located elsewhere on the chain, reduction is the predominant pathway. alpha-hydroxyketones or their diol metabolites may be excreted as glucuronic acid conjugates. Low concentrations of aliphatic acyclic methyl ketones are mainly metabolised by oxidation of the terminal methyl group. At higher concentrations, acyclic alpha-diketones are metabolised via a reduction pathway to the diol and subsequent conjugation with glucuronic acid In a 13-week study in rats (males/females, 15 animals/group), 3-hydroxybutan-2-one was administered with the diet at doses of 0, 85, 330 and 1,345 mg/kg bw per day. No treatment-related effects on body weight gain, haematological and urinary parameters, serum chemistry, organ weight and histopathology were seen up to 330 mg/kg bw per day. Several effects were observed at the highest dose tested, i.e. a reduction in body weight gain associated with a reduction in food and water consumption, an increase in relative liver weight and a slight anaemia. From this study, a no observed adverse effect level (NOAEL) of 330 mg/kg bw per day could be derived. A NOAEL of 90 mg/kg bw per day was derived from a 13-week study in rats (15 males/15 females each group), in which diacetyl [07.052] was administered by gavage at nominal doses of 0, 10, 30, 90 and 540 mg/kg bw per day. No adverse effects were seen at the three low doses tested on haematological and urinary parameters, serum chemistry, absolute and relative organ weight and histopathology. Several effects were observed at the highest dose tested (540 mg/kg bw), i.e. a decrease in weight gain associated with an increase in water consumption, anaemia, increased leucocyte count, increased relative weights of the liver, kidneys, adrenals and pituitary glands. At the same dose, stomach lesions seen at necropsy revealed necrosis with in filtration by inflammatory cells.

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	A trial was conducted to assess the chronic toxicity of 3-ethylcyclopentan-1,2-dione ((due to keto-enol tautomerism this substance can exist as two isomers; the keto-isomer is 3-ethylcyclopentan-1,2-dione a synonym for the keto-isomer is ethylcyclopentenolone) on reproduction and development in rats (male and female Charles River CD-COBS) following administration to three successive generations. In each generation, rats received diet containing 3-ethylcyclopentan-1,2-dione corresponding to dose levels of 0 (untreated controls), 0 (propylene glycol vehicle), 30, 80, and 200 mg/kg body weight/day. The F0 group (20 animals/sex/treatment) entered the study at weaning and were mated on day 64. Animals from the control groups and the high-dose group were maintained on trial for 12 months. The F1 generation 50 animals/sex per treatment except control, 100 animals/sex) was exposed to the test substance in utero, via milk until weaning and then through the diet for a further 23 months. The final examination of the F1 generation included ophthalmology, clinical chemistry, haematology and a full histopathology. The F1 generation was bred twice (days 99 and 155) and 20 litters/treatment group from the first mating selected to provide the F2 generation which were in turn mated at day 84. The F3 generation were killed after weaning. Survival, food consumption, growth, reproductive performance, haematological and clinical chemistry parameters were not adversely affected. Gross pathological and histopathological examination revealed no significant treatment-related effects. The incidence of benign or malignant tumours in treated animals was not significantly different to that in controls in the F0 and F1 generations. From this study, it is concluded that ethylcyclopentan-1,2-dione was not carcinogenic in rats under the study conditions and that a NOAEL of 200 mg/kg body weight (the highest dose tested) can be derived for chronic and developmental effects. A structural alert for genotoxicity is overruled for 3-ethyl-2-hydroxy-2-cyclopenten-1-one as well as for the nine structurally related substances (alpha,beta-unsaturated alicyclic ketones and their precursors) Maltol and ethyl maltol were considered separately because in contrast to the other substances in this subgroup they contain a ring-oxygen atom. Ethyl maltol induced gene mutations in bacteria Maltol induced gene mutations in bacteria and sister chromatid exchanges (SCE) in human lymphocytes In vivo, maltol induced micronuclei in mouse bone marrow after intraperitoneal application. Negative results were obtained in a sex-linked recessive lethal mutation assay in Drosophila. However, the micronucleus assay is considered more relevant than the Drosophila assay.Ethyl maltol induced gene mutations in bacteria EFSA Scientific Opinion October 2016: Safety and efficacy of secondary aliphatic saturated or unsaturated alcohols, ketones, ketals and esters with a second secondary or tertiary oxygenated functional group belonging to chemical group 10 when used as flavourings for all animal species Safety Evaluation of Aliphatic, Acyclic and Alicyclic alpha-Diketones and related Hydroxyketones; WHO Food Additive Series Joint FAO/ WHO Expert Committee on Food Additives 1999 The alpha,beta-unsaturated aldehyde and ketone structures are considered by the Panel to be structural alerts for genotoxicity. Flavouring Group Evaluation 213: alpha,beta-Unsaturated alicyclic ketones and precursors from chemical subgroup 2.7 of FGE.19: Scientific Opinion of the Panel on Food Contact Materials, Enzymes, Flavourings and Processing Aids (CEF)
BENZYL ACETATE & EUCALYPTOL & CITRAL & ETHYL HEPTANOATE & DIMETHYLCYCLOHEX-3-ENE-1-CARBALDEHYDE & EUGENOL & DODECYL ALDEHYDE	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.
BENZYL ACETATE & DIHYDROMYRCENOL & DODECYL ALDEHYDE & 10-UNDECENAL	The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.
BENZYL ACETATE & EUGENOL	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.
DIHYDROMYRCENOL & 1,2-DIHYDROLINALOOL	For terpenoid tertiary alcohols and their related esters: These substances are metabolised in the liver and excreted primarily in the urine and faeces. A portion is also excreted unchanged. They have low short term toxicity when ingested or applied on the skin. However, repeated and long term use may cause dose dependent harm to both the foetus and mother. Current opinion holds that there are no safety concerns regarding the branched chain unsaturated non-cyclic alcohols, as fragrance ingredients, at current declared levels of use and exposure; however, use of these materials at higher maximum levels of skin or whole-body exposure requires re-evaluation. At current declared levels of use, there was no evidence or only minimal evidence of skin irritation in humans. Sensitising hydroperoxides may be formed by contact with air. It should be ensured that oxidation reactions are prevented in the end product. The use of these materials under the declared levels of use and exposure will not induce sensitization. These compounds generally have low acute toxicity. The branched chain, unsaturated alcohols tested had low whole-body toxicity after repeated application. In animals, repeated exposure at high doses caused liver changes and kidney damage. There was little or no evidence of adverse effects on fertility or development. Data on cancer-causing potential is not available, but they are not of primary concern. Alkyl alcohols of chain length C6-13 are absorbed from skin, when inhaled or swallowed but show evidence of little harm. They are broken down and rapidly excreted by the body.
DIHYDROMYRCENOL & EUCALYPTOL & CITRAL & DIMETHYLCYCLOHEX-3-ENE-1-CARBALDEHYDE & CARVONE & EUGENOL & ROSE KETONES & ALPHA-DAMASCONE	Adverse reactions to fragrances in perfumes and fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, sensitivity to light, immediate contact reactions, and pigmented contact dermatitis. Airborne and connubial contact dermatitis occurs. Contact allergy is a lifelong condition, so symptoms may occur on re-exposure. Allergic contact dermatitis can be severe and widespread, with significant impairment of quality of life and potential consequences for fitness for work. If the perfume contains a sensitizing component, intolerance to perfumes by inhalation may occur. Symptoms may include general unwellness, coughing, phlegm, wheezing, chest tightness, headache, shortness of breath with exertion, acute respiratory illness, hayfever, asthma and other respiratory diseases. Perfumes can induce excess reactivity of the airway without producing allergy or airway obstruction. Breathing through a carbon filter mask had no protective effect. Occupational asthma caused by perfume substances, such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms, even though the exposure is below occupational exposure limits. Prevention of contact sensitization to fragrances is an important objective of public health risk management. Hands: Contact sensitization may be the primary cause of hand eczema or a complication of irritant or atopic hand eczema. However hand eczema is a disease involving many factors, and the clinical significance of fragrance contact allergy in severe, chronic hand eczema may not be clear. Underarm: Skin inflammation of the armpits may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a skin specialist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy. Face: An important manifestation of fragrance allergy from the use of cosmetic products is eczema of the face. In men, after-shave products can cause eczema around the beard area and the adjacent part of the neck. Men using wet shaving as opposed to dry have been shown to have an increased risk of allergic to fragrances. Irritant reactions: Some individual fragrance ingredients, such as citral, are known to be irritant. Fragrances may cause a dose-related contact urticaria (hives) which is not allergic; cinnamal, cinnamic alcohol and Myroxylon pereirae are known to cause hives, but others, including menthol, vanillin and benzaldehyde have also been reported.

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	Pigmentary anomalies: Type IV allergy is responsible for “pigmented cosmetic dermatitis”, referring to increased pigmentation on the face and neck. Testing showed a number of fragrance ingredients were associated, including jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol and geranium oil. Light reactions: Musk ambrette produced a number of allergic reactions mediated by light and was later banned from use in Europe. Furocoumarins (psoralens) in some plant-derived fragrances have caused phototoxic reactions, with redness. There are now limits for the amount of furocoumarins in fragrances. Phototoxic reactions still occur, but are rare. General/respiratory: Fragrances are volatile, and therefore, in addition to skin exposure, a perfume also exposes the eyes and the nose / airway. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma. Asthma-like symptoms can be provoked by sensory mechanisms. A significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients and hand eczema.
DIHYDROMYRCENOL & EUCALYPTOL & CITRAL & DIMETHYLCYCLOHEX-3-ENE-1-CARBALDEHYDE & CARVONE & ALPHA-DAMASCONE	Fragrance allergens act as haptens, which are small molecules that cause an immune reaction only when attached to a carrier protein. However, not all sensitizing fragrance chemicals are directly reactive, but some require previous activation. A prehapten is a chemical that itself causes little or no sensitization, but it is transformed into a hapten outside the skin by a chemical reaction (oxidation in air or reaction with light) without the requirement of an enzyme. For prehaptens, it is possible to prevent activation outside the body to a certain extent by different measures, for example, prevention of air exposure during handling and storage of the ingredients and the final product, and by the addition of suitable antioxidants. When antioxidants are used, care should be taken that they will not be activated themselves, and thereby form new sensitisers. Prehaptens: Most terpenes with oxidisable allylic positions can be expected to self-oxidise on air exposure. Depending on the stability of the oxidation products that are formed, the oxidized products will have differing levels of sensitization potential. Tests shows that air exposure of lavender oil increased the potential for sensitization. Prohaptens: Compounds that are bioactivated in the skin and thereby form haptens are referred to prohaptens. The possibility of a prohapten being activated cannot be avoided by outside measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Various enzymes play roles in both activating and deactivating prohaptens. Skin-sensitizing prohaptens can be recognized and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or studies of sensitization. QSAR prediction: Prediction of sensitization activity of these substances is complex, especially for those substances that can act both as pre- and prohaptens.
1,2-DIHYDROLINALOOL & EUCALYPTOL & CITRAL & DIMETHYLCYCLOHEX-3-ENE-1-CARBALDEHYDE & CARVONE & EUGENOL & 10-UNDECENAL & ROSYFOLIA & ROSE KETONES & ALPHA-DAMASCONE	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.
1,2-DIHYDROLINALOOL & CITRAL & 2,6-DIMETHYL-5-HEPTENAL	No significant acute toxicological data identified in literature search.
EUCALYPTOL & CITRAL	The terpenoid hydrocarbons are found in needle trees and deciduous plants. This category of chemicals shows very low acute toxicity. They are ecreted in the urine. They are unlikely to cause genetic damage, but animal testing shows that they do cause increased rates of kidney cancer. They have low potential to cause reproductive and developmental toxicity.
CITRAL & CARVONE	-
CITRAL & ROSE KETONES	Epoxidation of double bonds is a common bioactivation pathway for alkenes. The allylic epoxides formed were found to be sensitizing. Research has shown that conjugated dienes in or in conjunction with a six-membered ring are prohaptens, while related dienes containing isolated double bonds or an acrylic conjugated diene were weak or non-sensitising.
CITRAL & EUGENOL	The material may cause severe skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin. Repeated exposures may produce severe ulceration.
CARVONE & ROSE KETONES & ALPHA-DAMASCONE	A member or analogue of a group of aliphatic and alicyclic terpenoid tertiary alcohols and structurally related substances generally regarded as safe. Most alicyclic substances used as flavour ingredients are mono- and bicyclic terpenes which occur naturally in a wide variety of foods. With the exception of pulegone, alicyclic substances show very low oral acute toxicity. In most subchronic studies performed on animals, no adverse effects were observed at any dose level.
EUGENOL & ROSE KETONES	Fragrance allergens act as haptens, low molecular weight chemicals that cause an immune response only when attached to a carrier protein. However, not all sensitizing fragrance chemicals are directly reactive, but require previous activation. A prehapten is a chemical that itself causes little or no sensitization, but is transformed into a hapten in the skin (bioactivation), usually via enzyme catalysis. It is not always possible to know whether a particular allergen that is not directly reactive acts as a prehapten or a prohapten , or both. Prohaptens: Compounds that are bioactivated in the skin and thereby form haptens are referred to prohaptens. The possibility of a prohapten being activated cannot be avoided by outside measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Various enzymes play roles in both activating and deactivating prohaptens. Skin-sensitizing prohaptens can be recognized and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or studies of sensitization. QSAR prediction: Prediction of sensitization activity of these substances is complex, especially for those substances that can act both as pre- and prohaptens.
ROSE KETONES & ALPHA-DAMASCONE	For ionones and rose ketones, when used as fragrance ingredients: Ionones have low to moderate toxicity if swallowed. Acute toxicity by skin contact is low. Animal testing has not shown subchronic toxicity. Under intended conditions of use as fragrance ingredients, they do not have significant potential for genetic, reproductive or developmental toxicity. Ionones are non-irritating when used as fragrance ingredients, while the rose ketones have limited irritation potential in sensitive subjects. The ionones are considered to be without significant potential to sensitise the skin, while the rose ketones are sensitisers when present at concentrations greater than 0.2%. The safety margin is considered to be high. These should not be used as fragrance ingredients at concentrations more than 0.02%, individually or in combination with other isomers of damascone. This is based on data showing potential for sensitisation and evidence of cross-reactivity. Beta-ionone is absorbed after oral exposure. Metabolism takes place mainly in the liver, and beta-ionone is excreted via urine. It produces abnormal liver, kidney and thyroid changes, and may cause depression and tremors. It causes dose dependent eye and skin irritation but no evidence of cancer-causing effect, nerve or genetic toxicity was observed.

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

Continued...

sensitisation			
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

Tork Constant Air Freshener Blossom

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

benzyl acetate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	92mg/l	2
EC50	48h	Crustacea	17mg/l	2
LC50	96h	Fish	3.48-4.6mg/l	4
NOEC(ECx)	672h	Fish	0.92mg/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

1,2-dihydrolinalool

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	32mg/l	2
LC50	96h	Fish	42mg/l	2
EC10(ECx)	96h	Algae or other aquatic plants	3.7mg/l	2
EC50	96h	Algae or other aquatic plants	21mg/l	2

eucalyptol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>74mg/l	2
EC50	48h	Crustacea	>100mg/l	2
LC50	96h	Fish	57mg/l	2
EC50	96h	Algae or other aquatic plants	>74mg/l	2
NOEC(ECx)	96h	Algae or other aquatic plants	9.1mg/l	2

citral

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	16mg/l	1
EC50	48h	Crustacea	6.8mg/l	2
LC50	96h	Fish	4.6mg/l	1
EC50	96h	Algae or other aquatic plants	19mg/l	1
EC10(ECx)	96h	Algae or other aquatic plants	1.9mg/l	1

ethyl heptanoate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.202mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	0.101mg/l	2
LC50	96h	Fish	>1.01mg/l	2

p-ethyl-alpha,alpha-dimethyldihydrocinnamaldehyde

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

p-ethyl-alpha,alpha-dimethylhydrocinnamaldehyde

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

dimethylcyclohex-3-ene-1-carbaldehyde

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	22.2mg/l	2
EC50	48h	Crustacea	26.4mg/l	2
LC50	96h	Fish	8.61mg/l	2
EC10(ECx)	72h	Algae or other aquatic plants	10.7mg/l	2

2,6-dimethyl-5-heptenal

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	7.66mg/l	2
NOEC(ECx)	48h	Crustacea	0.39mg/l	2
EC50	48h	Crustacea	2.4mg/l	2

Continued...

	LC50	96h	Fish	2.288mg/l	2
	EC50	96h	Algae or other aquatic plants	4.3mg/l	2

carvone

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	50mg/l	2
EC50	72h	Algae or other aquatic plants	154.67mg/l	2
EC50	48h	Crustacea	249.516mg/l	2
EC50(ECx)	96h	Algae or other aquatic plants	110mg/l	2
EC50	96h	Algae or other aquatic plants	110mg/l	2
EC50	72h	Algae or other aquatic plants	19mg/l	2
EC50	48h	Crustacea	>9.59mg/l	2
NOEC(ECx)	96h	Fish	<2.91mg/l	4
LC50	96h	Fish	1.1mg/l	2
EC50	96h	Algae or other aquatic plants	26mg/l	2

eugenol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	23mg/l	2
EC50	48h	Crustacea	1.05mg/l	2
LC50	96h	Fish	13mg/l	2
EC0(ECx)	48h	Crustacea	0.36mg/l	2

dodecyl aldehyde

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.042mg/l	2
EC50	48h	Crustacea	>0.27mg/l	2
EC50(ECx)	72h	Algae or other aquatic plants	0.042mg/l	2
LC50	96h	Fish	~2.6mg/l	2

10-undecenal

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	1.3mg/l	2
LC50	96h	Fish	>18.72mg/l	2
NOEC(ECx)	504h	Crustacea	0.201mg/l	2

rosyfolia

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	3.4mg/l	2
EC50	72h	Algae or other aquatic plants	3.8mg/l	2
LC50	96h	Fish	3.2mg/l	2
NOEC(ECx)	96h	Fish	0.31mg/l	2

rose ketones

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	8.8mg/l	2
EC50	48h	Crustacea	9.5mg/l	2
EC50(ECx)	72h	Algae or other aquatic plants	8.8mg/l	2
LC50	96h	Fish	1.09mg/l	2
EC50	72h	Algae or other aquatic plants	8.3mg/l	2
EC50	48h	Crustacea	9mg/l	2
EC50(ECx)	72h	Algae or other aquatic plants	8.3mg/l	2
LC50	96h	Fish	1.09mg/l	2
EC50	72h	Algae or other aquatic plants	5mg/l	2
EC50	48h	Crustacea	2.37mg/l	2
EC50(ECx)	48h	Crustacea	2.37mg/l	2
EC50	72h	Algae or other aquatic plants	2.45mg/l	2
EC50	48h	Crustacea	2.32mg/l	2
EC10(ECx)	72h	Algae or other aquatic plants	1.14mg/l	2
EC50	72h	Algae or other aquatic plants	2.47mg/l	2
LC50	96h	Fish	0.97mg/l	2
EC10(ECx)	768h	Fish	0.074mg/l	2

alpha-damascone

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	1.09mg/l	2
EC50	72h	Algae or other aquatic plants	8.3mg/l	2
EC50	48h	Crustacea	9mg/l	2
EC50(ECx)	72h	Algae or other aquatic plants	8.3mg/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***

Continued...

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
benzyl acetate	LOW	LOW
dihydromyrcenol	HIGH	HIGH
1,2-dihydrolinalool	HIGH	HIGH
eucalyptol	HIGH	HIGH
citral	LOW	LOW
ethyl heptanoate	LOW	LOW
p-ethyl-alpha,alpha-dimethyldihydrocinnamaldehyde	HIGH	HIGH
dimethylcyclohex-3-ene-1-carbaldehyde	LOW	LOW
2,6-dimethyl-5-heptenal	LOW	LOW
carvone	HIGH	HIGH
eugenol	HIGH	HIGH
dodecyl aldehyde	LOW	LOW
10-undecenal	LOW	LOW
rose ketones	HIGH	HIGH
alpha-damascone	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
benzyl acetate	LOW (LogKOW = 1.96)
dihydromyrcenol	LOW (LogKOW = 3.4666)
1,2-dihydrolinalool	LOW (LogKOW = 3.4666)
eucalyptol	LOW (LogKOW = 2.74)
citral	LOW (LogKOW = 3.4453)
ethyl heptanoate	LOW (LogKOW = 3.3197)
p-ethyl-alpha,alpha-dimethyldihydrocinnamaldehyde	MEDIUM (LogKOW = 3.9425)
dimethylcyclohex-3-ene-1-carbaldehyde	LOW (LogKOW = 2.8536)
2,6-dimethyl-5-heptenal	LOW (LogKOW = 3.0395)
carvone	LOW (LogKOW = 2.71)
eugenol	LOW (LogKOW = 2.27)
dodecyl aldehyde	HIGH (LogKOW = 4.7451)
10-undecenal	MEDIUM (LogKOW = 4.1176)
rose ketones	MEDIUM (LogKOW = 4.4235)
alpha-damascone	MEDIUM (LogKOW = 4.2938)

Mobility in soil

Ingredient	Mobility
benzyl acetate	LOW (Log KOC = 133.7)
dihydromyrcenol	LOW (Log KOC = 54.78)
1,2-dihydrolinalool	LOW (Log KOC = 56.32)
eucalyptol	LOW (Log KOC = 106.7)
citral	LOW (Log KOC = 147.7)
ethyl heptanoate	LOW (Log KOC = 137.1)
p-ethyl-alpha,alpha-dimethyldihydrocinnamaldehyde	LOW (Log KOC = 843.8)
dimethylcyclohex-3-ene-1-carbaldehyde	LOW (Log KOC = 82.5)
2,6-dimethyl-5-heptenal	LOW (Log KOC = 80.06)
carvone	LOW (Log KOC = 123.7)
eugenol	LOW (Log KOC = 1124)
dodecyl aldehyde	LOW (Log KOC = 682.4)
10-undecenal	LOW (Log KOC = 370)
rose ketones	LOW (Log KOC = 668.6)
alpha-damascone	LOW (Log KOC = 668.6)

SECTION 13 Disposal considerations

Continued...

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible 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

COMBUSTIBLE LIQUID	COMBUSTIBLE LIQUID, regulated for storage purposes only
Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

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

Product name	Group
benzyl acetate	Not Available
dihydromyrcenol	Not Available
1,2-dihydrolinalool	Not Available
eucalyptol	Not Available
citral	Not Available
ethyl heptanoate	Not Available
p-ethyl-alpha,alpha-dimethyldihydrocinnamaldehyde	Not Available
p-ethyl-alpha,alpha-dimethylhydrocinnamaldehyde	Not Available
dimethylcyclohex-3-ene-1-carbaldehyde	Not Available
2,6-dimethyl-5-heptenal	Not Available
carvone	Not Available
eugenol	Not Available
dodecyl aldehyde	Not Available
10-undecenal	Not Available
rosyfolia	Not Available
rose ketones	Not Available
alpha-damascone	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
benzyl acetate	Not Available
dihydromyrcenol	Not Available
1,2-dihydrolinalool	Not Available
eucalyptol	Not Available
citral	Not Available
ethyl heptanoate	Not Available

Continued...

Product name	Ship Type
p-ethyl-alpha,alpha-dimethyldihydrocinnamaldehyde	Not Available
p-ethyl-alpha,alpha-dimethylhydrocinnamaldehyde	Not Available
dimethylcyclohex-3-ene-1-carbaldehyde	Not Available
2,6-dimethyl-5-heptenal	Not Available
carvone	Not Available
eugenol	Not Available
dodecyl aldehyde	Not Available
10-undecenal	Not Available
rosyfolia	Not Available
rose ketones	Not Available
alpha-damascone	Not Available

SECTION 15 Regulatory information

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

benzyl acetate is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

dihydromyrcenol is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)

1,2-dihydrolinalool is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)

eucalyptol is found on the following regulatory lists
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)

citral is found on the following regulatory lists
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australian Inventory of Industrial Chemicals (AIIC)

ethyl heptanoate is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)

p-ethyl-alpha,alpha-dimethyldihydrocinnamaldehyde is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)

p-ethyl-alpha,alpha-dimethylhydrocinnamaldehyde is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)

dimethylcyclohex-3-ene-1-carbaldehyde is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)

2,6-dimethyl-5-heptenal is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)

carvone is found on the following regulatory lists
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

eugenol is found on the following regulatory lists
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

dodecyl aldehyde is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)

10-undecenal is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)

rosyfolia is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)

rose ketones is found on the following regulatory lists
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

alpha-damascone is found on the following regulatory lists

Continued...

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
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	No (rosyfolia)
Canada - NDSL	No (benzyl acetate; dihydromyrcenol; 1,2-dihydrolinalool; eucalyptol; citral; ethyl heptanoate; p-ethyl-alpha,alpha-dimethyldihydrocinnamaldehyde; p-ethyl-alpha,alpha-dimethylhydrocinnamaldehyde; dimethylcyclohex-3-ene-1-carbaldehyde; 2,6-dimethyl-5-heptenal; carvone; eugenol; dodecyl aldehyde; 10-undecenal)
China - IECSC	No (rosyfolia)
Europe - EINEC / ELINCS / NLP	No (rosyfolia)
Japan - ENCS	No (p-ethyl-alpha,alpha-dimethyldihydrocinnamaldehyde; rosyfolia; alpha-damascone)
Korea - KECI	No (rosyfolia)
New Zealand - NZIoC	No (rosyfolia)
Philippines - PICCS	No (rosyfolia)
USA - TSCA	Yes
Taiwan - TCSI	No (rosyfolia)
Mexico - INSQ	No (p-ethyl-alpha,alpha-dimethyldihydrocinnamaldehyde; p-ethyl-alpha,alpha-dimethylhydrocinnamaldehyde; rosyfolia)
Vietnam - NCI	Yes
Russia - FBEPH	No (1,2-dihydrolinalool; p-ethyl-alpha,alpha-dimethyldihydrocinnamaldehyde; rosyfolia; alpha-damascone)
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	03/09/2024
Initial Date	30/08/2024

SDS Version Summary

Version	Date of Update	Sections Updated
3.1	03/09/2024	Toxicological information - Acute Health (inhaled), Toxicological information - Acute Health (skin), Toxicological information - Acute Health (swallowed), First Aid measures - Advice to Doctor, Physical and chemical properties - Appearance, Toxicological information - Chronic Health, Hazards identification - Classification, Ecological Information - Environmental, Exposure controls / personal protection - Exposure Standard, First Aid measures - First Aid (skin), Handling and storage - Handling Procedure, Composition / information on ingredients - Ingredients, Exposure controls / personal protection - Personal Protection (other), Accidental release measures - Spills (major), Accidental release measures - Spills (minor), Handling and storage - Storage (storage incompatibility), Handling and storage - Storage (storage requirement), Identification of the substance / mixture and of the company / undertaking - Supplier Information, Toxicological information - Toxicity and Irritation (Other), Identification of the substance / mixture and of the company / undertaking - Use, Name

Other information

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

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

Definitions and abbreviations

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

- AIIC: Australian Inventory of Industrial Chemicals
- DSL: Domestic Substances List
- NDSL: Non-Domestic Substances List
- IECSC: Inventory of Existing Chemical Substance in China
- EINECS: European INventory of Existing Commercial chemical Substances

Continued...

- 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

end of SDS