Room Spray (Holiday Berry)

Ming Fai Enterprise International Co., Ltd

Safety Data Sheet according to OSHA HazCom Standard (2024) requirements

SDS No.: HKGH0325528606
Issue Date: 11/06/2025
Print Date: 11/06/2025

SECTION 1 Identification

Product Identifier

Product name	Room Spray (Holiday Berry) Contains: lemongrass oil
Synonyms	Not Available
Other means of identification	Not Available

Recommended use of the chemical and restrictions on use

Relevant identified uses	AROMATHERAPY

Name, address, and telephone number of the chemical manufacturer, importer, or other responsible party

Registered company name	**Ming Fai Enterprise International Co., Ltd**
Address	Unit D3, 8/F, TML Tower, No. 3 Hoi Shing Road, Tsuen Wan, New Territories, Hong Kong
Telephone	852 2455 4888
Fax	Not Available
Website	Not Available
Email	scarlett.chen@mingfaigroup.com

Emergency phone number

Association / Organisation	**ALDI, BATAVIA, IL 60510**
Emergency telephone number(s)	Not Available
Other emergency telephone number(s)	Not Available

SECTION 2 Hazard(s) identification

Classification of the substance or mixture

Classification	Sensitisation (Skin) Category 1

Continued...

Label elements

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

Hazard statement(s)

H317	May cause an allergic skin reaction.

Hazard(s) not otherwise classified

Not Applicable

Precautionary statement(s) General

P101	If medical advice is needed, have product container or label at hand.
P102	Keep out of reach of children.
P103	Read label before use.

Precautionary statement(s) Prevention

P261	Avoid breathing mist/vapours/spray.
P280	Wear protective gloves.
P272	Contaminated work clothing must not be allowed out of the workplace.

Precautionary statement(s) Response

P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P321	Specific treatment (see instructions on this label)
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
7732-18-5	80-90	water
61788-85-0	5-10	castor oil, hydrogenated, ethoxylated
6132-04-3	1-5	sodium citrate
Not Applicable	1-5	Fragrance – BT478271
122-99-6	0.5-0.9	ethylene glycol phenyl ether
9038-95-3	0.5-0.9	butyl alcohol propoxylated

Continued...

25265-71-8	0.1-0.5	dipropylene glycol (as part of fragrance BT478271)
5949-29-1	0.1-0.5	citric acid, monohydrate
10339-55-6	0.1-0.5	ethyl linalool (as part of fragrance BT478271)
101-86-0	0.1-0.5	alpha-hexylcinnamaldehyde (as part of fragrance BT478271)
8008-57-9	0.1-0.5	orange oil (as part of fragrance BT478271)
8007-02-1	0.1-0.5	lemongrass oil
70445-33-9	0.05-0.0999	ethylhexylglycerin
84929-31-7	0.05-0.0999	lemon oil, extract (as part of fragrance BT478271)
78-70-6	0.05-0.0999	linalool (as part of fragrance BT478271 and Cinnamon Oil)
140-11-4	0.05-0.0999	benzyl acetate (as part of fragrance BT478271)
1205-17-0	0.05-0.0999	piperonyl propanal (as part of fragrance BT478271)
18479-58-8	0.05-0.0999	dihydromyrcenol (as part of fragrance BT478271)
118-58-1	0.01-0.05	benzyl salicylate (as part of fragrance BT478271)
8000-41-7	0.01-0.05	alpha-terpineol (as part of fragrance BT478271)
1948-33-0	0.01-0.05	tert-butylhydroquinone (as part of fragrance BT478271)
Not Applicable	0.005-0.01	Fragrance – Sweet Orange Oil
Not Applicable	0.005-0.01	Fragrance – Cinnamon Oil
138-86-3	0.005-0.01	dipentene (as part of fragrance Sweet Orange Oil)
104-55-2	0.001-0.005	cinnamaldehyde (as part of fragrance Cinnamon Oil)
97-53-0	0.001-0.005	Eugenol (as part of fragrance Cinnamon Oil)
87-44-5	0.0005-0.001	beta-caryophyllene (as part of fragrance Cinnamon Oil)
123-35-3	0.0001-0.0005	Myrcene (as part of fragrance Sweet Orange Oil)
93-28-7	0.0001-0.0005	eugenyl acetate (as part of fragrance Cinnamon Oil)
5989-27-5, 5989-54-8	0.0001-0.0005	Limonene (as part of fragrance Cinnamon Oil)
124-13-0	0.0001-0.0005	octyl aldehyde (as part of fragrance Sweet Orange Oil)
10191-41-0	0.00005-0.0001	alpha-tocopherol
112-31-2	0.00005-0.0001	decyl aldehyde (as part of fragrance Sweet Orange Oil)
94-59-7	0.00005-0.0001	safrole (as part of fragrance Cinnamon Oil)
97-54-1	0.00005-0.0001	isoeugenol (as part of fragrance Cinnamon Oil)
127-91-3	0.00005-0.0001	beta-pinene (as part of fragrance Cinnamon Oil)
80-56-8	0.00005-0.0001	alpha-pinene (as part of fragrance Cinnamon Oil)
5392-40-5	0.00001-0.00005	citral (as part of fragrance Sweet Orange Oil)

SECTION 4 First-aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with eyes: ‣ Wash out immediately with water. ‣ If irritation continues, seek medical attention. ‣ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	‣ Wash hands after use.
Inhalation	‣ Other measures are usually unnecessary.
Ingestion	‣ Immediately give a glass of water. ‣ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

Most important symptoms and effects, both acute and delayed

See Section 11

Indication of any immediate medical attention and special treatment needed

Treat symptomatically

Continued...

SECTION 5 Fire-fighting measures

Extinguishing media

- There is no restriction on the type of extinguisher which may be used.
- Use extinguishing media suitable for surrounding area.

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

Special protective equipment and precautions for fire-fighters

Fire Fighting	‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear breathing apparatus plus protective gloves in the event of a fire. ‣ Prevent, by any means available, spillage from entering drains or water courses. ‣ Use fire fighting procedures suitable for surrounding area. ‣ **DO NOT** approach containers suspected to be hot. ‣ Cool fire exposed containers with water spray from a protected location. ‣ If safe to do so, remove containers from path of fire. ‣ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	carbon dioxide (CO_2) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.

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	‣ 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. ‣ Stop leak if safe to do so. ‣ Contain spill with sand, earth or vermiculite. ‣ Collect recoverable product into labelled containers for recycling. ‣ Neutralise/decontaminate residue (see Section 13 for specific agent). ‣ Collect solid residues and seal in labelled drums for disposal. ‣ Wash area and prevent runoff into drains. ‣ After clean up operations, decontaminate and launder all protective clothing and equipment before storing

Continued...

	and re-using. ▸ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ Avoid contact with incompatible materials. ▸ When handling, DO NOT eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Always wash hands with soap and water after handling. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.
Fire and explosion protection	See section 5
Other information	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer’s storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Packaging as recommended by manufacturer. ▸ Check that containers are clearly labelled. ▸ Tamper-proof containers. ▸ Polyethylene or polypropylene containers. ▸ Metal drum with sealed plastic liner. ▸ Glass container is suitable for laboratory quantities
Storage incompatibility	d-Limonene: ▸ forms unstable peroxides in storage, unless inhibited; may polymerise ▸ reacts with strong oxidisers and may explode or combust ▸ is incompatible with strong acids, including acidic clays, peroxides, halogens, vinyl chloride and iodine pentafluoride ▸ flow or agitation may generate electrostatic charges due to low conductivity Terpenoids and terpenes, are generally unsaturated, are thermolabile, are often volatile and may be easily oxidised or hydrolysed depending on their respective structure. Terpenoids are subject to autoxidation. Autoxidation is any oxidation that occurs in open air or in presence of oxygen (and sometimes UV radiation) and forms peroxides and hydroperoxides. Though autoxidation has been particularly investigated in the field of fatty oils, it also plays a most crucial part for terpenoid deterioration. Although virtually all types of organic materials can undergo air oxidation, certain types are particularly prone to autoxidation, including unsaturated compounds that have allylic or benzylic hydrogen atoms (C6H5CH2-); these materials are converted to hydroperoxides by autoxidation. Promoted by heat, catalytic quantities of redox-reactive metals, and exposure to light, autoxidation may result in the formation of explosive peroxides which may become explosive upon concentration. As a rule, however, primary autoxidation products such as hydroperoxides eventually break down during advanced stages of oxidation depending on their individual stability. Thereby they give rise to a range of stable oxidised secondary products such as mono- to polyvalent alcohols, aldehydes, ketones, epoxides, peroxides,

Continued...

or acids as well as highly viscous, often oxygen-bearing polymers. Light, heat, or increasing acidity often promote this breakdown.
Compounds rich in allylic hydrogen atoms (2HC=CHCH2-R), found in most terpenoids, make up the most probable targets for autoxidation.
Several terpenoids (typically oxygen containing derivatives) are saturated and do not react in a similar fashion to their unsaturated congeners.
Thermolabile terpenoids, especially mere terpenes and aldehydes, are susceptible to rearrangement processes at elevated temperatures. Terpenic conversion reactions, upon heating, have been reported both for isolated compounds as well as for essential oils.(which tend to be rich in mono-, and sesqui-terpenes.
Mono-, bi-, or tricyclic mono- terpenoids (those containing two isoprene units, dienes) and sesquiterpenoids (with three isoprene units, trienes) of different chemical classes, such as hydrocarbons, ketones, alcohols, oxides, aldehydes, phenols, or esters, make up the major part in essential oils.
Electron-donating groups and increasing alkyl substitution contribute to a stronger carbon-peroxide bond through a hyperconjugative effect, thus leading to more stable and subsequently built-up hydroperoxides.
Some oxygen-bearing terpenoids such as menthol, eucalyptol (1,8-cineol), and menthone do not form hydroperoxides upon oxidation but are directly converted into ketones, acids, and aldehydes.None of these are unsaturated compounds.
Due to their low volatility, diterpenes (with four isoprenes, tetraenes) are barely encountered in genuine essential oils obtained by distillation, while tri- and higher terpenoids such as sterols or carotenoids are only present in the nonvolatile fractions such as plant resins or gums and will remain in the residue
Aging processes generally come along with a more or less pronounced quality loss In addition to the frequent development of unpleasant and often pungent flavours, shifting colors such as the formation of a yellow staining or changes in consistency up to resinification have been reported both upon degradation of single terpenoids as well as of essential oils.

- Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
sodium citrate	9.3 mg/m3	100 mg/m3	610 mg/m3
butyl alcohol propoxylated	27 mg/m3	300 mg/m3	1,800 mg/m3
ethylene glycol phenyl ether	1.5 ppm	16 ppm	97 ppm
benzyl acetate	30 ppm	330 ppm	2,000 ppm
alpha-terpineol	59 mg/m3	650 mg/m3	1,000 mg/m3
octyl aldehyde	17 mg/m3	190 mg/m3	1,100 mg/m3
decyl aldehyde	1.8 ppm	19 ppm	120 ppm
d-limonene	15 ppm	67 ppm	170 ppm
safrole	5.9 mg/m3	64 mg/m3	390 mg/m3
alpha-pinene	60 ppm	120 ppm	1,500 ppm

Ingredient	Original IDLH	Revised IDLH
water	Not Available	Not Available
castor oil, hydrogenated, ethoxylated	Not Available	Not Available
sodium citrate	Not Available	Not Available

Continued...

Ingredient	Original IDLH	Revised IDLH
butyl alcohol propoxylated	Not Available	Not Available
ethylene glycol phenyl ether	Not Available	Not Available
citric acid, monohydrate	Not Available	Not Available
ethylhexylglycerin	Not Available	Not Available
alpha-tocopherol	Not Available	Not Available
lemongrass oil	Not Available	Not Available
tert-butylhydroquinone	Not Available	Not Available
benzyl acetate	Not Available	Not Available
alpha-terpineol	Not Available	Not Available
piperonyl propanal	Not Available	Not Available
benzyl salicylate	Not Available	Not Available
dihydromyrcenol	Not Available	Not Available
linalool	Not Available	Not Available
lemon oil, extract	Not Available	Not Available
orange oil	Not Available	Not Available
ethyl linalool	Not Available	Not Available
alpha-hexylcinnamaldehyde	Not Available	Not Available
dipropylene glycol	Not Available	Not Available
dipentene	Not Available	Not Available
myrcene	Not Available	Not Available
octyl aldehyde	Not Available	Not Available
decyl aldehyde	Not Available	Not Available
citral	Not Available	Not Available
cinnamaldehyde	Not Available	Not Available
eugenol	Not Available	Not Available
beta-caryophyllene	Not Available	Not Available
d-limonene	Not Available	Not Available
beta-pinene	Not Available	Not Available
eugenyl acetate	Not Available	Not Available
safrole	Not Available	Not Available
alpha-pinene	Not Available	Not Available
isoeugenol	Not Available	Not Available

Exposure controls

Appropriate engineering controls	Enclosed local exhaust ventilation is required at points of dust, fume or vapour generation. 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.

Continued...

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

See below

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

Continued...

	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	**NOTE:** ▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · 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.

Continued...

	‣ 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	‣ For quantities up to 500 grams a laboratory coat may be suitable. ‣ For quantities up to 1 kilogram a disposable laboratory coat or coverall of low permeability is recommended. Coveralls should be buttoned at collar and cuffs. ‣ For quantities over 1 kilogram and manufacturing operations, wear disposable coverall of low permeability and disposable shoe covers. ‣ For manufacturing operations, air-supplied full body suits may be required for the provision of advanced respiratory protection. ‣ Eye wash unit. ‣ Ensure there is ready access to an emergency shower. ‣ For Emergencies: Vinyl suit

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

'Forsberg Clothing Performance Index'.
Not Available

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.

Continued...

- 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	self-color

Physical state	Liquid	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	5.9	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	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
Nanoform Solubility	Not Available	**Nanoform Particle Characteristics**	Not Available
Particle Size	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.

Continued...

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 is not thought to produce adverse health effects or irritation of the respiratory tract. Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.
Ingestion	Nonionic surfactants may produce localised irritation of the oral or gastrointestinal lining and induce vomiting and mild diarrhoea. Exposure to the piperidines may result in increased blood pressure and heart rate, nausea, vomiting, salivation, laboured breathing, muscular weakness, paralysis and convulsions. It may also excite the senses of hearing and touch. High oral doses of salicylates, such as aspirin, may cause a mild burning pain in the throat and stomach, causing vomiting. This is followed (within hours) by deep, rapid breathing, tiredness, nausea and further vomiting, thirst and diarrhoea. The material has **NOT** been classified by other classification systems as 'harmful by ingestion'. This is because of the lack of corroborating animal or human evidence.
Skin Contact	Skin contact is not thought to have harmful health effects; the material may still produce health damage following entry through wounds, lesions or abrasions. There is some evidence to suggest that this material can cause inflammation of the skin on contact in some persons. Non-ionic surfactants cause less irritation than other surfactants as they have less ability to denature protein in the skin. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream, through, for example, cuts, abrasions or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Although the liquid is not thought to be an irritant, direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn). Non-ionic surfactants can cause numbing of the cornea, which masks discomfort normally caused by other agents and leads to corneal injury. Irritation varies depending on the duration of contact, the nature and concentration of the surfactant.

Continued...

Chronic	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. Chronic exposure to salicylates produce problems with metabolism, central nervous system disturbances, or kidney damage. Those with pre-existing damage to the eye, skin or kidney are especially at risk. Prolonged or repeated skin contact may cause degreasing, followed by drying, cracking and skin inflammation. 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. In one study with citrus oils, the authors concluded that a common component was capable of promoting skin tumour development in previously initiated mice. Cinnamaldehyde reacts to form an acid even when stored in tightly closed bottles with the head space flushed with nitrogen. The formation of conjugated dienals is responsible for the yellowing of the substance with time.

Room Spray (Holiday Berry)

TOXICITY	IRRITATION
Not Available	Not Available

water

TOXICITY	IRRITATION
Oral (Rat) LD50: >90000 mg/kg	Not Available

castor oil, hydrogenated, ethoxylated

TOXICITY	IRRITATION
Oral (Rat) LD50: >2000 mg/kg	Eye: no adverse effect observed (not irritating)
	Skin: no adverse effect observed (not irritating)

sodium citrate

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg	Eye: no adverse effect observed (not irritating)
Oral (Mouse) LD50; 5000-6000 mg/kg	Skin: no adverse effect observed (not irritating)

butyl alcohol propoxylated

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 13340 mg/kg	Eye (Rodent - rabbit): 20mg/24H - Moderate
Inhalation (Rat) LC50: 0.147 mg/L4h	Eye (Rodent - rabbit): 500mg
Oral (Rabbit) LD50; 1770 mg/kg	Eye (Rodent - rabbit): 500mg
	Eye (Rodent - rabbit): 500mg/24H - Mild
	Eye (Rodent - rabbit): 500mg/24H - Mild
	Eye (Rodent - rabbit): 50mg - Severe
	Eye (Rodent - rabbit): 50mg - Severe
	Eye: adverse effect observed (irritating)
	Eye: no adverse effect observed (not irritating)

Continued...

	Skin (Rodent - rabbit): 10mg/24H - Mild
	Skin (Rodent - rabbit): 500mg - Mild
	Skin (Rodent - rabbit): 500mg - Mild
	Skin (Rodent - rabbit): 500mg - Mild
	Skin (Rodent - rabbit): 500mg - Mild
	Skin (Rodent - rabbit): 500mg - Mild
	Skin (Rodent - rabbit): 500mg - Mild
	Skin (Rodent - rabbit): 500mg - Mild
	Skin (Rodent - rabbit): 500mg - Mild
	Skin (Rodent - rabbit): 500mg - Mild
	Skin (Rodent - rabbit): 500mg - Mild
	Skin (Rodent - rabbit): 500mg - Mild
	Skin (Rodent - rabbit): 500mg - Mild
	Skin (Rodent - rabbit): 500mg/24H - Mild
	Skin (Rodent - rabbit): 500mg/24H - Mild
	Skin (Rodent - rabbit): 500mg/24H - Mild
	Skin (Rodent - rabbit): 80mg/4H
	Skin (Rodent - rabbit): 80mg/4H - Mild
	Skin: adverse effect observed (irritating)
	Skin: no adverse effect observed (not irritating)

ethylene glycol phenyl ether

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg	Eye (Rodent - rabbit): 250ug/24H - Severe
Oral (Rat) LD50: 1260 mg/kg	Eye (Rodent - rabbit): 6mg - Moderate
	Eye: adverse effect observed (irreversible damage)
	Eye: adverse effect observed (irritating)
	Skin (Rodent - rabbit): 500mg/24H - Mild
	Skin: adverse effect observed (irritating)
	Skin: no adverse effect observed (not irritating)

citric acid, monohydrate

TOXICITY	IRRITATION
Oral (Mouse) LD50; 5790 mg/kg	Eye (Rodent - rabbit): 5mg/30S - Mild
	Eye (Rodent - rabbit): 750ug/24H - Severe
	Skin (Rodent - rabbit): 0.5mL - Moderate
	Skin (Rodent - rabbit): 500mg/24H - Mild

ethylhexylglycerin

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg	Skin (Human - woman): 5%/2D
Inhalation (Rat) LC50: 2.83 mg/l4h	Skin (Human - woman): 5%/2D (intermittent)
Oral (Rat) LD50: >2000 mg/kg	

alpha-tocopherol

TOXICITY	IRRITATION
dermal (rat) LD50: >3000 mg/kg	Eye: no adverse effect observed (not irritating)

Continued...

	Oral (Mouse) LD50; >5000 mg/kg	Skin: no adverse effect observed (not irritating)

lemongrass oil

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg	Eye: adverse effect observed (irritating)
Oral (Rat) LD50: >5000 mg/kg	Skin (Human - woman): 2%
	Skin (Mammal - pig): 100% - Mild
	Skin (Rodent - mouse): 100% - Mild
	Skin (Rodent - rabbit): 500mg/24H - Moderate
	Skin: adverse effect observed (irritating)

tert-butylhydroquinone

TOXICITY	IRRITATION
Dermal (Guinea Pig) LD50: >500<1000 mg/kg	Eye: adverse effect observed (irritating)
Oral (Rat) LD50: 700 mg/kg	Skin: adverse effect observed (irritating)

benzyl acetate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg	Eye: no adverse effect observed (not irritating)
Oral (Rat) LD50: 2490 mg/kg	Skin (Rodent - rabbit): 100mg/24H - Moderate
	Skin: no adverse effect observed (not irritating)

alpha-terpineol

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg	Eye (Mammal - species unspecified): 12.5% - Mild
Inhalation (Rat) LC50: >4.76 mg/l4h	Eye (Mammal - species unspecified): 12.5% - Mild
Oral (Mouse) LD50; 12.8 mg/kg	Eye: adverse effect observed (irreversible damage)
	Eye: adverse effect observed (irritating)
	Skin (Rodent - mouse): 50% - Severe
	Skin (Rodent - rabbit): 100%/4H - Mild
	Skin (Rodent - rabbit): 500mg/24H - Moderate
	Skin (Rodent - rabbit): 500mg/24H - Moderate
	Skin: adverse effect observed (irritating)
	Skin: no adverse effect observed (not irritating)

piperonyl propanal

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg	Eye: no adverse effect observed (not irritating)
Oral (Rat) LD50: 3362 mg/kg	Skin: no adverse effect observed (not irritating)

benzyl salicylate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg	Eye: adverse effect observed (irritating)
Oral (Rat) LD50: 2227 mg/kg	Skin (Human): 2%/2D
	Skin: no adverse effect observed (not irritating)

dihydromyrcenol

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg	Eye (Rodent - rabbit): 0.05% - Mild

Continued...

	Oral (Rat) LD50: 3600 mg/kg	Eye (Rodent - rabbit): 0.1mL
		Eye (Rodent - rabbit): 0.1mL
		Eye (Rodent - rabbit): 5%
		Eye (Rodent - rabbit): 7.5%
		Eye (Rodent - rabbit): 7.5% - Mild
		Eye: adverse effect observed (irritating)
		Skin (Rodent - rabbit): 0.5mL/4H
		Skin (Rodent - rabbit): 0.5mL/4H - Mild
		Skin (Rodent - rabbit): 500mg/24H - Mild
		Skin: adverse effect observed (irritating)

	TOXICITY	IRRITATION
linalool	dermal (rat) LD50: 5610 mg/kg	Eye (Rodent - rabbit): 0.1mL/1H - Moderate
	Oral (Rat) LD50: 2790 mg/kg	Eye (Rodent - rabbit): 100uL - Moderate
		Eye: adverse effect observed (irritating)
		Skin (Human - man): 16mg/48H - Mild
		Skin (Human): 10%/2D
		Skin (Human): 32%/72H - Mild
		Skin (Rodent - guinea pig): 100mg/24H - Moderate
		Skin (Rodent - rabbit): 100mg/24H - Severe
		Skin (Rodent - rabbit): 500mg/24H - Mild
		Skin: adverse effect observed (irritating)

	TOXICITY	IRRITATION
lemon oil, extract	Dermal (rabbit) LD50: >10000 mg/kg	Eye: no adverse effect observed (not irritating)
	Oral (Rat) LD50: >5000 mg/kg	Skin: adverse effect observed (irritating)

	TOXICITY	IRRITATION
orange oil	Dermal (rabbit) LD50: >5000 mg/kg	Skin (Rodent - rabbit): 500mg/24H - Moderate
	Oral (Rat) LD50: >5000 mg/kg	

	TOXICITY	IRRITATION
ethyl linalool	Dermal (rabbit) LD50: >5000 mg/kg	Eye (Rodent - rabbit): 0.05% - Mild
	Oral (Rat) LD50: >5000 mg/kg	Eye (Rodent - rabbit): 0.1mL - Moderate
		Eye: adverse effect observed (irritating)
		Skin (Rodent - rabbit): 0.05%/24H - Mild
		Skin (Rodent - rabbit): 0.5mL/4H - Moderate
		Skin (Rodent - rabbit): 1%/24H - Moderate
		Skin (Rodent - rabbit): 10gm - Moderate
		Skin (Rodent - rabbit): 5% - Mild
		Skin (Rodent - rabbit): 500mg/24H - Moderate
		Skin: adverse effect observed (irritating)

Continued...

alpha-hexylcinnamaldehyde

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >3000 mg/kg	Eye: no adverse effect observed (not irritating)
Oral (Mouse) LD50; 2300 mg/kg	Skin (Human): 5%/2D
	Skin (Rodent - guinea pig): 100mg/24H - Severe
	Skin (Rodent - mouse): 30%/3D(intermittent)
	Skin (Rodent - rabbit): 100mg/24H - Severe
	Skin (Rodent - rabbit): 500mg/24H - Moderate
	Skin: adverse effect observed (irritating)

dipropylene glycol

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5010 mg/kg	Eye (Rodent - rabbit): 500mg - Mild
Inhalation (Rat) LC50: >2.34 mg/l4h	Eye: no adverse effect observed (not irritating)
Oral (Rat) LD50: >5000 mg/kg	Skin (Rodent - rabbit): 500uL/24H - Moderate
	Skin: no adverse effect observed (not irritating)

dipentene

TOXICITY	IRRITATION
Oral (Mouse) LD50; 4773 mg/kg	Skin (Rodent - rabbit): 500mg/24H - Moderate
	Skin: adverse effect observed (irritating)
	Skin: no adverse effect observed (not irritating)

myrcene

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg	Eye: adverse effect observed (irritating)
Oral (Rat) LD50: >5000 mg/kg	Skin (Rodent - rabbit): 500mg/24H - Moderate
	Skin: adverse effect observed (irritating)

octyl aldehyde

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 6350 mg/kg	Eye (Rodent - rabbit): 0.01mL - Mild
Oral (Rat) LD50: 5630 mg/kg	Eye (Rodent - rabbit): 0.5mL - Severe
	Eye (Rodent - rabbit): 100mg - Mild
	Eye: adverse effect observed (irritating)
	Skin (Human): 128240ppm - Mild
	Skin (Rodent - rabbit): 0.5mL - Moderate
	Skin (Rodent - rabbit): 14mg/24H - Mild
	Skin (Rodent - rabbit): 500mg/24H - Mild
	Skin: adverse effect observed (irritating)

decyl aldehyde

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 5040 mg/kg	Eye: adverse effect observed (irritating)
Oral (Rat) LD50: 3730 mg/kg	Skin (Rodent - rabbit): 14.4ug/24H - Severe
	Skin (Rodent - rabbit): 500mg/24H - Mild
	Skin: no adverse effect observed (not irritating)

Continued...

citral

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg	Skin (Human - man): 16mg/48H - Severe
Oral (Rat) LD50: 4960 mg/kg	Skin (Human - woman): 2%
	Skin (Human): 2%/2D
	Skin (Human): 40mg/24H - Mild
	Skin (Mammal - pig): 50mg/48H - Severe
	Skin (Rodent - guinea pig): 1%/48H - Moderate
	Skin (Rodent - guinea pig): 100mg/24H - Severe
	Skin (Rodent - rabbit): 100mg/24H - Severe
	Skin (Rodent - rabbit): 500mg/24H - Moderate

cinnamaldehyde

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg	Eye: adverse effect observed (irritating)
Inhalation (Rat) LC50: 68.889 ppm4h	Skin (Human): 1%/2D
Oral (Rat) LD50: 2220 mg/kg	Skin (Human): 40mg/48H - Severe
	Skin: adverse effect observed (irritating)

eugenol

TOXICITY	IRRITATION
Oral (Rat) LD50: 1930 mg/kg	Eye: adverse effect observed (irritating)
	Skin (Human - man): 16mg/48H - Moderate
	Skin (Human): 1%/2D
	Skin (Human): 40mg/48H - Mild
	Skin (Mammal - pig): 50mg/48H - Mild
	Skin (Rodent - guinea pig): 100mg/24H - Moderate
	Skin (Rodent - rabbit): 100mg/24H - Severe
	Skin: no adverse effect observed (not irritating)

beta-caryophyllene

TOXICITY	IRRITATION
Oral (Mouse) LD50; >5000 mg/kg	Eye: no adverse effect observed (not irritating)
	Skin (Rodent - rabbit): 500mg/24H
	Skin: no adverse effect observed (not irritating)

d-limonene

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg	Eye: no adverse effect observed (not irritating)
Oral (Rat) LD50: >2000 mg/kg	Skin (Rodent - mouse): 700mg/7D (intermittent) - Severe
	Skin (Rodent - rabbit): 10%/24H - Mild
	Skin (Rodent - rabbit): 500mg/24H - Moderate
	Skin (Rodent - rat): 100%/1H
	Skin: no adverse effect observed (not irritating)

beta-pinene

TOXICITY	IRRITATION
Oral (Rabbit) LD50; 4700 mg/kg	Eye: no adverse effect observed (not irritating)

Continued...

	Skin (Rodent - rabbit): 500mg/24H - Moderate
	Skin: adverse effect observed (irritating)

eugenyl acetate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg	Eye: no adverse effect observed (not irritating)
Oral (Rat) LD50: 1670 mg/kg	Skin (Rodent - rabbit): 500mg/24H - Moderate
	Skin: no adverse effect observed (not irritating)

safrole

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg	Skin (Rodent - rabbit): 500mg/24H - Moderate
Oral (Rat) LD50: 1950 mg/kg	

alpha-pinene

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg	Eye: no adverse effect observed (not irritating)
Oral (Rat) LD50: >500 mg/kg	Skin (Human - man): 100% - Severe
	Skin (Rodent - rabbit): 500mg/24H - Moderate
	Skin: adverse effect observed (irritating)

isoeugenol

TOXICITY	IRRITATION
Oral (Guinea) LD50; 1410 mg/kg	Skin (Human - man): 16mg/48H - Moderate
	Skin (Human): 1%/2D
	Skin (Rodent - guinea pig): 100mg/24H - Severe
	Skin (Rodent - rabbit): 100mg/24H - Severe

CASTOR OIL, HYDROGENATED, ETHOXYLATED

Inhalation-risk test (IRT): No mortality within 8 hours as shown in animal studies. The inhalation of a highly saturated vapor-air mixture represents no acute hazard. Skin irritation: rabbit: non-irritant (OECD Guideline 404) Eye irritation : rabbit: non-irritant (BASF-Test) Sensitization: Guinea pig maximization test/guinea pig: Non-sensitizing. Chronic toxicity Genetic toxicity: In the majority of studies performed with microorganisms and in mammalian cell culture, a mutagenic effect was not found. A mutagenic effect was also not observed in in vivo tests. Developmental toxicity/teratogenicity: No indications of a developmental toxic / teratogenic effect were seen in animal studies. * BASF MSDS Cremaphor RH Surfactant
This product contains partially hydrogenated fatty acids and/ or trans fatty acids.
The consumption of trans fats increases the risk of coronary heart disease by raising levels of LDL cholesterol and lowering levels of 'good' HDL cholesterol. There is an ongoing debate about a possible differentiation between trans fats of natural origin and trans fats of man-made origin but so far no scientific consensus has been found. Two Canadian studies have shown that the natural trans fat vaccenic acid, found in beef and dairy products, may have an opposite health effect and could actually be beneficial compared to hydrogenated vegetable shortening, or a mixture of pork lard and soy fat, by lowering total and LDL cholesterol and triglyceride levels. In lack of recognized evidence and scientific agreement, nutritional authorities consider all trans fats as equally harmful for health and recommend that consumption of trans fats be reduced to trace amounts.
The use of hydrogenated oils in foods has never been completely satisfactory. Because the center arm of the triglyceride is shielded somewhat by the end fatty acids, most of the hydrogenation occurs on the end fatty acids,

Continued...

	While full hydrogenation produces largely saturated fatty acids, partial hydrogenation results in the transformation of unsaturated cis fatty acids to trans fatty acids in the oil mixture due to the heat used in hydrogenation. Partially hydrogenated oils and their trans fats have increasingly been viewed as 'unhealthy'. Trans fat is the common name for unsaturated fat with trans-isomer (E-isomer) fatty acid(s). Because the term refers to the configuration of a double carbon-carbon bond, trans fats are sometimes monounsaturated or polyunsaturated, but never saturated. Trans fats do exist in nature but also occur during the processing of polyunsaturated fatty acids in food production. Trans fats occur naturally in a limited number of cases: vaccenyl and conjugated linoleyl (CLA) containing trans fats occur naturally in trace amounts in meat and dairy products from ruminants. The exact biochemical methods by which trans fats produce specific health problems are a topic of continuing research. One theory is that the human lipase enzyme works only on the cis configuration and cannot metabolise a trans fat. A lipase is a water-soluble enzyme that helps digest, transport, and process dietary lipids such as triglycerides, fats, and oils in most - if not all - living organisms. While the mechanisms through which trans fats contribute to coronary heart disease are fairly well understood, the mechanism for trans fat's effect on diabetes is still under investigation. Trans fatty acids may impair the metabolism of long-chain polyunsaturated fatty acids (LCPUFAs), but maternal pregnancy trans fatty acid intake has been inversely associated with LCPUFAs levels in infants at birth thought to underlie the positive association between breastfeeding and intelligence. There are suggestions that the negative consequences of trans fat consumption go beyond the cardiovascular risk. In general, there is much less scientific consensus asserting that eating trans fat specifically increases the risk of other chronic health problems: It has been suggested that the intake of both trans fats and saturated fats promote the development of Alzheimer disease, although not confirmed in an animal model. It has been found that trans fats impaired memory and learning in middle-age rats. The rats' brains of trans-fat eaters had fewer proteins critical to healthy neurological function. Inflammation in and around the hippocampus, the part of the brain responsible for learning and memory. These are the exact types of changes normally seen at the onset of Alzheimer's, but seen after six weeks, even though the rats were still young. There is a growing concern that the risk of type 2 diabetes increases with trans fat consumption.[52] However, consensus has not been reached. For example, one study found that risk is higher for those in the highest quartile of trans fat consumption.Another study has found no diabetes risk once other factors such as total fat intake and BMI were accounted for. Research indicates that trans fat may increase weight gain and abdominal fat, despite a similar caloric intake. A 6-year experiment revealed that monkeys fed a trans fat diet gained 7.2% of their body weight, as compared to 1.8% for monkeys on a mono-unsaturated fat diet. Although obesity is frequently linked to trans fat in the popular media, this is generally in the context of eating too many calories; there is not a strong scientific consensus connecting trans fat and obesity, although the 6-year experiment did find such a link, concluding that 'under controlled feeding conditions, long-term TFA consumption was an independent factor in weight gain. TFAs enhanced intra-abdominal deposition of fat, even in the absence of caloric excess, and were associated with insulin resistance, with evidence that there is impaired post-insulin receptor binding signal transduction. Liver Dysfunction: Trans fats are metabolised differently by the liver than other fats and interfere with delta 6 desaturase. Delta 6 desaturase is an enzyme involved in converting essential fatty acids to arachidonic acid and prostaglandins, both of which are important to the functioning of cells. Infertility in women: One 2007 study found, 'Each 2% increase in the intake of energy from trans unsaturated fats, as opposed to that from carbohydrates, was associated with a 73% greater risk of ovulatory infertility...'. Major depressive disorder: Spanish researchers analysed the diets of 12,059 people over six years and found those who ate the most trans fats had a 48 per cent higher risk of depression than those who did not eat trans fats. One mechanism may be trans-fats' substitution for docosahexaenoic acid (DHA) levels in the orbitofrontal cortex (OFC). Very high intake of trans-fatty acids (43% of total fat) in mice from 2 to 16 months of age was associated with lowered DHA levels in the brain (p=0.001) When the brains of 15 major depressive subjects who had committed suicide were examined post-mortem and compared against 27 age-matched controls, the suicidal brains were found to have 16% less (male average) to 32% (female average) less DHA in the OFC. The OFC is known to control reward, reward expectation and empathy, which are all negatively impacted in depressive mood disorders, as well as regulating the limbic system>
SODIUM CITRATE	For citric acid (and its inorganic citrate salts) Based on extensive animal testing data and on human experience, citric acid has low acute toxicity. Citric acid is not suspected of causing cancer, birth defects or reproductive toxicity. Further, it does not cause

Continued...

	mutations. Also, the sensitizing potential is considered low. In contrast, irritation, particularly of the eyes but also the airways and the skin, is the main hazard presented by citric acid.
BUTYL ALCOHOL PROPOXYLATED	In general, the toxicity of the PPGs Butyl Ether decreased with increasing molecular weight; for example, PPG-40 Butyl Ether was less toxic than PPG-2 Butyl Ether. Mutagenicity data were not found on the PPGs Butyl Ether. However, an ether of molecular weight 800 Da (~PPG-13 Butyl Ether) was non-carcinogenic when fed to rats for 2 years. Because the PPGs Butyl Ethers undergo metabolic degradation; i.e., the butyl group are removed and oxidized, the PPG chains are split into random length fragments, the genotoxicity of the component chemicals, propylene glycol (PG) and n- Butyl Alcohol, were also considered. Both PG and n-Butyl Alcohol were non-mutagenic in mammalian and microbial assays. PG was non-carcinogenic in a 2-year feeding study using rats and in a lifetime dermal study using mice. These studies effectively eliminated the need for genotoxicity data on the PPG Butyl Ethers. There was concern about the irritancy potential of PPG-2 Butyl Ether. In animal irritation studies, the ingredient caused minor, transient erythema and desquamation; in addition, erythema, edema, ecchymosis, necrosis, and other changes were observed during an acute percutaneous study. PPG-2 Butyl Ether also caused minor to moderate conjunctival irritation and minor corneal injury. It was concluded that the PPG Butyl Ethers were safe for use in cosmetics when formulated to avoid irritation. The dermal LD50 of PPG-3 Butyl Ether was 2 g/kg in rats and rabbits, and the dermal LD50 of Buteth-3 in rats was 3.5 g/kg. The oral LD50 of PPG-3 Butyl Ether and of Buteth-3 in rats was 2 g/kg and 6.6 g/kg, respectively. Polypropyleneglycol butyl ethers (not defined) had a dermal and an oral LD50 of 2 g/kg and 0.3-2 g/kg bw, respectively,in mice.Buteth-3 (1000 mg/kg/day) was not toxic to rabbits in a 21-day dermal study; erythema, desquamation,and fissuring were observed In short-term oral toxicity studies in rats, PPG-3 Butyl Ether had a NOAEL of 1000 mg/kg bw; polypropylene glycol butyl ethers had a NOEL of 100 mg/kg bw/day for clinical observations, higher absolute and relative liver weights, and an increased incidence of liver and thyroid gland hypertrophy; and 1-(2-butoxy-1-methylethoxy)propan-2-ol had a NOAEL of 100 mg/kg/day based on very slight to slight hepatocellular hypertrophy with no corresponding increases in liver weights in low-dose males. In a 90-day oral toxicity study, administration of up to 1000 mg/kg bw/day PPG-3 Butyl Ether to rats in drinking water produced treatment-related increases in absolute and relative liver and kidney weights. The NOAELs in rats and mice exposed to=3000 ppm methoxyisopropanol via inhalation for 2 yrs were 1000 ppm (based on slight body wt decreases in males and females) and 300 ppm (based on altered hepatocellular foci in males), respectively.Dermal application of propylene glycol butyl ether was not embryotoxic or teratogenic to rabbits (=100 mg/kg bw/day applied on days 7-18 of. gestation) or rats (=1.0 ml/kg bw/day applied on days 6-16 of gestation). 1-(2-Butoxy-1-methyl-ethoxy)propan-2-ol (applied on days 6-16 or 6-15 of gestation) also was not embryotoxic or teratogenic in rats. No test-article related adverse developmental or reproductive effects were observed in rats dosed by gavage with up to 1000 mg/kg Buteth-3 or 1-(2-butoxy-1-methylethoxy)propan-2-ol or up to 500 mg/kg bw/day polypropylene glycol butyl ethers. In inhalation studies, exposure of rats to =1.0 mg/l air PPG-3 Methyl Ether did not have any teratogenic or reproductive effects. Exposure to 1000 and 3000 ppm methoxyisopropanol produced some adverse effects in a two-generation study in rats; adverse effects were not observed with 300 ppm. PPG-3 Butyl Ether was not genotoxic in vitro in the Ames test or in vivo in a mouse micronucleus assay. Propylene glycol butyl ether was not genotoxic in an Ames test or a mammalian chromosomal aberration assay in rat lymphocytes, andneither propylene glycol butyl ether or 1-(2-butoxy-1-methylethoxy)propan-2-ol were genotoxic in a mammalian cellmutation assay in CHO cell. In inhalation carcinogenicity studies, mice and rats were exposed by whole body exposure to =3000 ppm methoxyisopropan-ol for 2 yrs. An increase in S-phase DNA synthesis and in MFO activity in the liver was observed in high-dose male mice and rats. Renal epithelial tumors were not observed, and the NOEL for carcinogenicity was 3000 ppm for mice and rats. Undiluted PPG-3 Butyl Ether was not irritating to rabbit skin or eyes, and it was not an irritant or sensitizer in guinea pigs. Polypropylene glycol butyl ethers were classified as non-corrosive in an EpiDermTM study Humans have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents and other cleaning products. Exposure to these chemicals can occur through swallowing, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that relatively high volumes would have to occur to produce any toxic response. No death due to poisoning with alcohol ethoxylates has ever been reported. Studies show that alcohol ethoxylates have low toxicity through swallowing and skin contact. Animal studies show these chemicals may produce gastrointestinal irritation, stomach ulcers, hair standing up, diarrhea and lethargy. Slight to severe irritation occurred when undiluted alcohol ethyoxylates were applied to the skin and eyes of animals. These chemicals show no indication of genetic toxicity or potential to cause mutations and cancers. Toxicity is thought to be substantially lower than that of nonylphenol ethoxylates.

Continued...

	Some of the oxidation products of this group of substances may have sensitizing properties. As they cause less irritation, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their tendency to auto-oxidise also increases their irritation. Due to their irritating effect it is difficult to diagnose allergic contact dermatitis (ACD) by patch testing. Both laboratory and animal testing has shown that there is no evidence for alcohol ethoxylates (AEs) causing genetic damage, mutations or cancer. No adverse reproductive or developmental effects were observed. Tri-ethylene glycol ethers undergo enzymatic oxidation to toxic alkoxy acids. They may irritate the skin and the eyes. At high oral doses, they may cause depressed reflexes, flaccid muscle tone, breathing difficulty and coma. Death may result in experimental animal. However, repeated exposure may cause dose dependent damage to the kidneys as well as reproductive and developmental defects.
ETHYLENE GLYCOL PHENYL ETHER	Bacterial cell mutagen The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
ETHYLHEXYLGLYCERIN	Oral (-) LD50: >2000 mg/kg OECD 401 Skin: non-irritant OECD 404 Dermal (-) LD50: >2000 mg/kg OECD 402 Eye: irritant OECD 405 Non-sensitising (OECD 406) The no toxic effect level for oral application to rats over 28 days is 100 mg/kg/day. A NOEL cannot be determined. OECD 407 No experimental information on genotoxicity in vitro or in vivo available. * Schulke Alkyl glyceryl ethers (AGEs) often act as surfactants or skin conditioning agents in cosmetics. These substances show minimal dermal penetration. Furthermore, a review of the available data on toxicity revealed: an absence of genotoxicity in studies using ethylhexylglycerin, chimyl alcohol, batyl alcohol, and glyceryl allyl ether; an absence of reproductive and developmental toxicity in oral studies using ethylhexylglycerin; negative skin irritation/sensitization data in studies using ethylhexylglycerin and chimyl alcohol; and negative phototoxicity/photoallergenicity data in studies using ethylhexylglycerin. Overall, the available toxicity data, coupled with the limited dermal penetration, suggested that these ingredients could be used safely in the present practices of use and concentration. **Oral toxicity:** Using chimyl alcohol a a surrogate of this group approximately 95% is absorbed following oral administration with 40% recovered (as metabolites) in the urine after 12 hours. The lymph shows significant absorption (50%) whilst triglycerides, phospholipids and free fatty acids also seem to incorporate the absorbed substance. No mortalities or exposure-related toxicological findings were observed in rats dosed orally with undiluted ethylhexylglycerin or chimyl alcohol. Ethylhexylglycerin administered orally to rats, at doses up to 800 mg/kg/day, in a 13-week study did not result in any treatment-related deaths, macroscopic observations,or neurotoxicity. A statistically significant increase in absolute and relative-to-body weight liver weights was observed in males of all dose groups and females of the highest dose group. A dose of 50 mg/kg/day (lowest dose) was considered the lowest observed adverse effect level (LOAEL) in one study and no observed adverse effect level (NOAEL) in another. There were no treatment-related mortalities in rats dosed orally with ethylhexylglycerin at doses up to 1,500 mg/kg for 28 days. Increased liver and adrenal weights were observed in the highest dose group, and microscopic findings included slight to moderate liver hypertrophy in rats of all 3 dose groups. The 100 mg/kg dose was defined as the no-observed-adverse effect-level (NOAEL). **Dermal toxicity:** Mean absorption of another surrogate, ethylhexylglycerin through the skin of rabbits is insignificant (0.2% at approximately 2 hours post application) and there were no signs of skin irritation. The quantity of ethylhexylglycerin in the plasma was below the detection limit at the end of the 4 h application period. Over a range of 3 concentrations (44.65, 47.15, and 54.94%) applied to human skin in vitro, mean penetration rates of 2.38, 8.19, and 20.38 ug/cm2/h were reported. Chimyl alcohol was classified as a mild skin irritant in rabbits after a single application, but was a non-irritating to the skin of rabbits in a cumulative skin irritation study. Skin sensitisation was not observed in guinea pigs tested with 0.5% ethylhexylglycerin during induction and challenged with a higher concentration (50%) in the maximization test. Local lymph node assay results for ethylhexylglycerin at concentrations up to 50% were also negative. Products containing ethylhexylglycerin at concentrations ranging from 0.4% to ~1% were neither skin irritants nor sensitisers. Ethylhexylglycerin was not phototoxic or photoallergenic in guinea pigs when tested at concentrations up to 100% in the presence of UVA/UVB light. Chimyl alcohol suppressed the production of chemical mediators of UVB-irradiated keratinocytes in vitro and substantially suppressed UV-induced tanning in human skin. Based on these findings, a new concept for skin whitening via controlling keratinocyte function was proposed

Continued...

	No mortalities or signs of skin irritation or abnormal necropsy findings were observed after undiluted ethylhexylglycerin was applied to the skin of rats.Necropsy findings were unremarkable. there were no treatment-related mortalities in rats dosed orally with ethylhexylglycerin at doses up to 1,500 mg/kg for 28 days. Increased liver and adrenal weights were observed in the highest dose group, and microscopic findings included slight to moderate liver hypertrophy in rats of all 3 dose groups. The 100 mg/kg dose was defined as the no-observed-adverse effect-level (NOAEL). **Ocular toxicity:** Undiluted ethylhexylglycerin was severely irritating , but 5% ethylhexylglycerin was mildly irritating, to the eyes of rabbits **Inhalation toxicity:** In an acute inhalation toxicity study using groups of rats exposed to ethylhexylglycerin (nose-only, mean achieved concentrations of 1.89, 2.96, and 4.98 mg/l), a concentration-related increase in mortality was observed. The lung was described as a target organ, based on rapid deaths, severe respiratory changes, and abnormal colouration and enlargement of the lungs. **Parenteral toxicity**: Batyl alcohol stimulated haematopoiesis (both red and white blood cells, following subcutaneous injection) in repeated dose studies involving rats and guinea pigs. **Developmental toxicity:** The results of visceral and skeletal examinations in litters of female rats given oral doses of ethylhexylglycerin (up to 800 mg/kg/day) were negative. In the one-generation developmental toxicity study (same doses) involving male and female rats, oestrous cycles were comparable between groups, but the fertility index for rats of the highest dose group was lower when compared to controls. There were no treatment-related effects on implantation. Necropsy findings in dosed rats found dead or killed did not indicate any treatment-related changes. The no-observed-effect-level (NOEL) for developmental toxicity in both sexes was 50/mg/kg/day **Genotoxicity:** Ethylhexylglycerin, chimyl alcohol, batyl alcohol, glyceryl allyl ether were all non-genotoxic in the Ames test under a variety of conditions. No genotoxicity or clastogenic was exhibited in any of the AGEs using the micronucleus, chromosomal aberration assays assays, Studies on the carcinogenicity of the AGEs were not found in the published literature
ALPHA-TOCOPHEROL	[ROCHE] * Bronson and Jacobs SDS (for similar products) Use in foodstuffs is consistent with low order of toxicity. Based on laboratory and animal testing, exposure to the material may result in irreversible effects and mutations in humans. alpha-Tocopherol was non-mutagenic and non-carcinogenic, and the results of reproduction/ teratology studies did not indicate that alpha-tocopherol had adverse effects on reproductive function. However, in a long-term study in rats, a no-effect level could not be established with respect to effects on blood clotting and liver histology, and there was evidence from human studies that excessive intakes of alpha-tocopherol could cause haemorrhage. Other adverse effects noted in clinical studies at doses of > 720 mg alpha-tocopherol/day included weakness, fatigue, creatinuria and effects on steroid hormone metabolism. Clinical studies indicate that, generally, intakes of below about 720 mg/day are without adverse effects in man, but one investigation in elderly patients showed an increase in serum cholesterol at doses of 300 mg alpha-tocopherol daily. Incidences of allergic reactions seem to be very rare. alpha-Tocopherol may be an essential nutrient. The U.S. National Academy of Sciences/National Research Council has recommended a dietary allowance of 0.15 mg/kg b.w./day. However, excessive intakes of alpha-tocopherol produce adverse clinical and biochemical effects, and self-medication with large doses of vitamin E preparations could present a hazard. The previously-allocated ADI was amended to include a lower value, which reflects the fact that alpha-tocopherol may be an essential nutrient. The upper value, which represents the maximum value for the AID, is based on clinical experience in man. IPCS Inchem: https://www.inchem.org/documents/jecfa/jecmono/v21je05.htm
TERT-BUTYLHYDROQUINONE	Tremor and muscle weakness recorded. * [Eastman] The European Food Safety Authority (EFSA) and the United States Food and Drug Administration (FDA) have evaluated TBHQ and determined that it is safe to consume at the concentration allowed in foods. The FDA and European Union both set an upper limit of 0.02% (200mg/kg) of the oil or fat content in foods. At very high doses, it has some negative health effects on lab animals, such as producing precursors to stomach tumors and damage to DNA. A number of studies have shown that prolonged exposure to very high doses of TBHQ may be carcinogenic, especially for stomach tumors. Other studies, however, have shown opposite effects including inhibition against HCA-induced carcinogenesis (by depression of metabolic activation) for TBHQ and other phenolic antioxidants (TBHQ was one of several, and not the most potent). The EFSA considers TBHQ to be noncarcinogenic.. A 1986 review of scientific literature concerning the toxicity of TBHQ determined that a wide margin of safety exists between the levels of intake by humans and the doses that produce adverse

Continued...

	effects in animal studies. There have been reports of vision disturbances in individuals exposed to this chemical. In addition, TBHQ has been identified by high-throughput screening as having potential immunotoxic effects Data show that acute toxicity following oral and topical use of hindered phenols is low. They are not proven to cause mutations. However, long term use may affect the liver, thyroid, kidney and lymph nodes. Liver tumours have been reported.
BENZYL ACETATE	Neoplastic by RTECS criteria This is a member or analogue of a group of benzyl derivatives generally regarded as safe (GRAS), based partly on their self-limiting properties as flavouring substances in food. In humans and other animals, they are rapidly absorbed, broken down and excreted, with a wide safety margin. They also lack significant potential to cause genetic toxicity and mutations. The intake of benzyl derivatives as natural components of traditional foods is actually higher than the intake as intentionally added flavouring substances. 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.
PIPERONYL PROPANAL	* IFF MSDS
BENZYL SALICYLATE	A member or analogue of a group of hydroxy and alkoxy-substituted benzyl derivatives generally regarded as safe (GRAS) based in part on their self-limiting properties as flavouring substances in food; their rapid absorption. metabolic detoxification, and excretion in humans and other animals, their low level of flavour use, the wide margin of safety between the conservative estimates of intake and the no-observed-adverse effect levels determined from chronic and subchronic studies and the lack of significant genotoxic and mutagenic potential. This evidence of safety is supported by the fact that the intake of benzyl derivatives as natural components of traditional foods is greater than the intake as intentionally added flavouring substances. All members of this group are aromatic primary alcohols, aldehydes, carboxylic acids or their corresponding esters or acetals. The structural features common to all members of the group is a primary oxygenated functional group bonded directly to a benzene ring. The ring also contains hydroxy or alkoxy substituents. The hydroxy- and alkoxy- substituted benzyl derivatives are raidly absorbed by the gastrointestinal tract, metabolised in the liver to yield benzoic acid derivatives and excreted primarily in the urine either unchanged or conjugated. It is expected than aromatic esters and acetals will be hydrolysed in vivo through the catalytic activity of carboxylesterases, (A-esterases), Acetals hydrolyse uncatalysed in gastric juices and intestinal fluids to yield acetaldehydes. Substituted benzyl esters and benzaldehyde acetals are hydrolysed to the corresponding alcoholic alcohols and carboxylic acid. In general hydroxy- and alkoxy- derivatives of benzaldehyde and benzyl alcohol are oxidised to the corresponding benzoic aid derivatives and, to a lesser extent reduced to corresponding benzyl alcohol derivatives. Following conjugation these are excreted in the urine. Benzyl alcohol derivatives may also be reduced in gut microflora to toluene derivatives. Flavor and Extract Manufacturers Association (FEMA)
LINALOOL	Opinion holds that there are no safety concerns for linalool and the linalyl esters, as fragrance ingredients, under the present declared levels of use and exposure for the following reasons: · Linalool and the linalyl esters have a low order of acute toxicity. · No significant toxicity was observed in subchronic tests; it is concluded that these materials have dermal and oral NOAELS of 50 mg/kg/day or greater. · Based on a critical review of all available mutagenicity and genotoxicity studies,it has been determined that these materials are negative in short-term tests and therefore would have no significant potential to produce genotoxic effects. · The metabolic fate of linalool and the linalyl esters is either known or assumed from analogies with structurally related substances that indicate no production of toxic or persistent metabolites and the structural analogies indicate no concern. · Human dermatological studies show that these materials are not irritating, phototoxic or sensitizing. · These materials are used at low levels of exposure relative to doses that elicit adverse effects. The estimate for maximum systemic exposure by humans using cosmetic products is 0.3

Continued...

	mg/kg/ day for linalool and linalyl acetate and 0.1 mg/ kg/day or lower for the other linalyl esters. Using the NOAELs (50 mg/kg/day or greater) and the maximum exposure estimates and assuming 100% absorption,a margin of safety for the exposure of humans to linalool and the linalyl esters may conservatively be calculated as 167 times the maximum daily exposure for linalool and linalyl acetate (50 mg/kg/day 0.3 mg/kg/day for linalool or linalyl acetate=167) and 500 times the maximum daily exposure for the other individual linalyl esters (50 mg/kg/day / 0.1 mg/kg/day for the other individual linalyl esters=500). In general, linalool esters are hydrolyzed to their corresponding alcohol (linalool) and carboxylic acid. Hydrolysis is catalyzed by carboxylesterases or esterases . Tertiary alcohols such as linalool are metabolized primarily through conjugation with glucuronic acid and are excreted in the urine and to a lesser extent faeces. Alkyl or alkenyl substituents may undergo oxidation to form polar metabolites that may also be excreted free or in the conjugated form. Oxidation is mediated by cytochrome P-450 dependant mono-oxygenases. The carboxylic acids formed by hydrolysis of the linalyl esters included in this summary are all known to be easily and rapidly metabolized. The linear saturated carboxylic acids are metabolized normally as fatty acids that undergo beta-oxidation. The branched-chain carboxylic acids from linalyl isovalerate and isobutyrate are similarly oxidized,but the end product is acetone. The carboxylic acids from linalyl benzoate and phenylacetate are conjugated and excreted. The cinnamic acid from linalyl cinnamate is conjugated and excreted,or metabolized to benzoic acid. No sensitization was observed with linalool in guinea pig sensitization studies at concentrations up to 20%. With linalyl acetate at a concentration of 10%,weak to moderate sensitization effects were observed in guinea pig sensitization studies. Linalyl acetate was non-sensitizing when tested at 5% in these same guinea pig sensitization studies. No sensitization reactions were observed with linalyl isobutyrate and linalyl propionate (data were not available for the other linalyl esters) when tested at 8% in open epicutaneous tests in guinea pigs The Research Institute for Fragrance Materials (RIFM) Expert Panel
ETHYL LINALOOL	* The Good Scents Company - Givaudin
DIPROPYLENE GLYCOL	For dipropylene glycol (DPG) and its isomers: Acute toxicity: Animal testing shows dipropylene glycol is not acutely toxic by mouth, skin contact or inhalation. DPG is slightly irritating to the skin and eyes of rabbits. Based on human data, DPG does not cause skin sensitization. Repeat dose toxicity: Animal testing shows DPG did not cause adverse effects on repeated exposure at low doses. Higher doses may cause kidney damage. Reproductive and developmental toxicity: Animal testing has not shown DPG to cause foetal toxicity or birth defects, at levels which did not cause toxicity to the mother. Genetic toxicity: Studies show that DPG does not cause genetic toxicity.
MYRCENE	NOTE: beta-Myrcene above 0.25 g/kg was found to be detrimental to the fertility and progeny number and development in the rat when given during pregnancy by gavage
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 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

Continued...

	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 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
EUGENOL	Equivocal tumorigen by RTECS criteria 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

Continued...

	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
BETA-CARYOPHYLLENE	-Caryophyllene acts as a full agonist of the Cannabinoid receptor type 2 (CB2 receptor) in rats.[7] ß-Caryophyllene has a binding affinity of Ki = 155nM at the CB2 receptors in mice.[8] ß-Caryophyllene has been shown to have anti-inflammatory action linked to its CB2 receptor activity in a study comparing the pain killing effects in mice with and without CB2 receptors with the group of mice without CB2 receptors seeing little benefit compared to the mice with functional CB2 receptors.[7] ß-Caryophyllene has the highest cannabinoid activity compared to the ring opened isomer a-caryophyllene Humulene which may modulate CB2 activity.[9] To compare binding, Cannabinol (CBN) binds to the CB2 receptors as a partial agonist with an affinity of CB2 Ki = 126.4 nM[10] while Delta-9-Tetrahydrocannabinol binds to the CB2 receptors as a partial agonist with an affinity of Ki = 36nM.[11] Caryophyllene helps to improve cold tolerance at low ambient temperatures. Wild giant pandas frequently roll in horse manure, which contains beta-caryophyllene/caryophyllene oxide, to inhibit transient receptor potential melastatin 8 (TRPM8), an archetypical cold-activated ion channel of mammal Caryophyllene has been given GRAS (generally regarded as safe) designation by the FDA and is approved by the FDA for use as a food additive, typically for flavoring] Rats given up to 700mg/kg daily for 90 days did not produce any significant toxic effects [15] Caryophyllene has an LD50 of 5,000mg/kg in mice In animal testing, cannabinoids (found in marijuana) caused changes in behaviour, hyperactivity, and seizures. High doses has caused delayed death. Long-term effects included central nervous system depression, showing as inco-ordination, decreased activity and generalized depression, with prolonged exposure leading to development of tolerance and symptoms of central nervous system stimulation, characterized by irritability, hypersensitivity, excessive activity, aggression, tremor and convulsions. Animals showed impaired specific motor and learning skills. Monkeys exposed chronically to marijuana showed changes in emotion, endocrine function and memory. It has been thought that THC (tetrahydrocannabinol) may be selectively lethal to female embryos. Animal studies have shown that marijuana extracts and THC cause birth defects. Marijuana and THC appear to

Continued...

	affect all phases of reproduction in human men and women, by altering sex hormone levels, acting directly on the reproductive organs, or both. Chronic marijuana smokers have lower sperm counts and poorer sperm motility than the general population. In women, THC and marijuana has been shown to block ovulation and disrupt the menstrual cycle. In humans, exposure before birth is reportedly associated with voice anomalies, short stature, low body weight, decreased head size and decreased verbal and memory scores on intelligence tests in infants and children. Regular use of marijuana has reportedly been associated with cancer of the upper airway, lung and tongue in patients under 40 years old. An increased incidence of leukaemia in offspring of mothers who smoked marijuana before or during pregnancy has also been reported. Animal testing has shown an increase in benign tumours of the thyroid.
D-LIMONENE	Tumorigenic by RTECS criteria
EUGENYL ACETATE	Some phenol-based naturally occurring substances (eg phenol, guaiacol, tannic acid and eugenol) undergo conversion to produce derivatives which sensitise the skin and possibly the respiratory tract. Each of these compounds has phenolic hydroxyl groups which are readily oxidized to produce reactive quinone-like compounds.
SAFROLE	1,3-Benzodioxole derivatives are found widely in nature. Certain such derivatives such as as safrole bind to and inhibit cytochrome P450. Cytochrome P450 enzymes function to metabolize potentially toxic compounds, A lipophilic substituent placed para to the methylenedioxy group increases this activity, e.g. piperonyl butoxide . Safrole is of further interest since it produces liver tumors in mice This toxicity is probably mediated via the propenyl side chain, either by metabolism to 1-hydroxysafrole and the subsequent formation of a reactive ester derivative or by the direct formation of a reactive epoxide. A particularly interesting example of the separation of two toxicities by structural modification is provided by estragole which is a mouse liver carcinogen but does not inhibit hepatic cytochrome P450 Equally dramatic is the fact that substitution of safrole with a methoxy group yields myristicin a reported hallucinogen derived from the nutmeg tree For safrole: Safrole belongs to the class of alk-2-enylbenzenes comprising, among others, estragole, methyleugenol, eugenol and myristicin. Several studies involving estragole have clearly established that the profiles of metabolism, metabolic activation, and covalent binding are dose dependent and that the relative importance diminishes markedly at low levels of exposure (i.e. these events are not linear with respect to dose). In particular, rodent studies show that these events are minimal probably in the dose range of 1-10 mg/kg body weight, which is approximately 100-1000 times the anticipated human exposure to this substance. For these reasons it is concluded that the present exposure to estragole resulting from consumption of herbal medicinal products (short time use in adults at recommended doses) does not pose a significant cancer risk. In the meantime exposure of estragole to sensitive groups such as young children, pregnant and breastfeeding women should be minimised. The Scientific Committee on Food from the Health & Consumer Protection Directorate-General took a more concerned position and concluded that 'Estragole has been demonstrated to be genotoxic and carcinogenic. Therefore the existence of a threshold cannot be assumed and the Committee could not establish a safe exposure limit. Consequently, reductions in exposure and restrictions in use levels are indicated. Toxicological studies have shown that safrole is a weak hepatocarcinogen at higher doses in rats and mice. Safrole requires metabolic activation before exhibiting toxicological effects. Metabolic conversion of the allyl group in safrole is able to produce intermediates which are directly capable of binding covalently with DNA and proteins. Metabolism of the methylenedioxy group to a carbene allows the molecule to form ligand complexes with cytochrome P450 and P448. The formation of this complex leads to lower amounts of available free cytochrome P450. Safrole can also directly bind to cytochrome P450, leading to competitive inhibition. These two mechanisms result in lowered mixed function oxidase activity. Furthermore, because of the altered structural and functional properties of cytochrome P450, loss of ribosomes which are attached to the endoplasmatic reticulum through cytochrome P450 may occur. The allyl group thus directly contributes to mutagenicity, while the methylenedioxy group is associated with changes in the cytochrome P450 system and epigenetic aspects of carcinogenicity. In rats, safrole and related compounds produced both benign and malignant tumours after intake through the mouth. Changes in the liver are also observed through the enlargement of liver cells and cell death. Studies in the 1960s suggested that safrole was carcinogenic, causing permanent liver damage in animals. Consequently, the US Food and Drug Administration (FDA) banned sassafras and safrole for

Continued...

	human consumption. Safrole is still regarded by FDA to be a weak carcinogen in rats. However, according to a 1977 study of the metabolites of safrole in both rats and humans, two carcinogenic metabolites of safrole found in the urine of rats, 1'-hydroxysafrole and 3'-hydroxyisosafrole, were not found in human urine. The European Commission on Health and consumer protection assumes safrole to be genotoxic and carcinogenic. It occurs naturally in a variety of spices, such as cinnamon, nutmeg, and black pepper, and herbs such as basil. In that role, safrole, like many naturally occurring compounds, may have a small but measurable ability to induce cancer in rodents. Despite this, the effects in humans were estimated by the Lawrence Berkeley National Laboratory to be similar to risks posed by breathing indoor air or drinking municipally supplied water. In the United States, it was once widely used as a food additive in root beer, sassafras tea, and other common goods, but was banned by the FDA after its carcinogenicity in rats was discovered. Today, safrole is also banned for use in soap and perfumes by the International Fragrance Association. Adverse effects Besides being a hepatocarcinogen, safrole exhibits further adverse effects in that it will induce the formation of hepatic lipid hydroperoxides. Safrole also inhibits the defensive function of neutrophils against bacteria. In addition to the inhibition of the defensive function of neutrophils, it has also been discovered that safrole interferes with the formation of superoxides by neutrophils. Furthermore, safrole oxide, a metabolite of safrole, has a negative effect on the central nervous system. Safrole oxide inhibits the expression of integrin beta4/SOD, which leads to apoptosis of the nerve cells. **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [*National Toxicology Program: U.S. Dep. of Health & Human Services 2002*]
ISOEUGENOL	Somnolence, coma recorded. ADI: 0.2 mg/kg/day NOEL: 500 mg/kg/day For isoeugenol: **Acute toxicity:** Studies on animals and humans demonstrate that isoeugenol is a skin sensitiser of moderate allergenic potency.. This is substantiated by clinical data that show widespread underlying allergy to isoeugenol although very few cases of allergy are clearly attributable to the presence of isoeugenol in any specific consumer products. Isoeugenol is rapidly metabolised and eliminated. Oral toxicokinetic studies show no signs of metabolic saturation. Skin penetration studies *in vitro* and *in vivo* show isoeugenol rapidly penetrates the skin. Isoeugenol has a moderate acute toxicity by dermal and oral routes (LD50 values > 1500 mg/kg). Inhalation is not considered a significant route of exposure. Systemic toxicity studies have shown that levels of 800 mg/kg/day are well tolerated by rats although these studies do not meet modern testing requirements. Isoeugenol shows moderate skin and eye irritancy but shows no significant phototoxicity or photoallergenic potential. *Critical end-points and threshold levels*: Skin sensitisation and systemic toxicity were considered to be the critical end-points. A No Expected Sensitization Level (NESL) of 250 ug/cm2 has been determined using a “weight of evidence” approach from a large number of predictive tests carried out on animals and studies in human subjects. There is evidence to show that although the threshold for eliciting allergic responses from non-occlusive exposure, to prior-sensitised individuals, may be as low as 80 ug/cm2, these “thresholds”cannot be used in risk assessment as they are neither reliable nor unique determinants of elicitation. **Reproductive and developmental toxicity**: Evidence from multi-generation reproduction toxicity studies in rats, shows that no adverse systemic effects occur at levels of 70 mg/kg bw/day . Developmental toxicity studies in single and multiple generations of rats have shown that the developmental NOAEL is 500 mg/kg bw/day which is about twice the level of maternal toxicity. **Genotoxicity and carcinogenicity: :** Isoeugenol is negative in bacterial and mammalian genotoxicity screens except in some studies where there is evidence that the results are the results of procedural artefacts. There are no data on the carcinogenic potential of isoeugenol. In the absence of a NOAEL from conventional systemic toxicity studies, two measures were taken as a basis for risk assessment. One was a NOAEL of 70 mg/kg bw/day from multiple generation developmental toxicity studies. The other was the Threshold of Toxicological Concern (TTC) of 30 ug/kg bw/day based on a large data set NOAELs of substances that have been similarly classified chemical structures

Continued...

WATER & LEMONGRASS OIL & PIPERONYL PROPANAL & LEMON OIL, EXTRACT & ORANGE OIL & CITRAL	No significant acute toxicological data identified in literature search.
CASTOR OIL, HYDROGENATED, ETHOXYLATED & BUTYL ALCOHOL PROPOXYLATED	Polyethers (such as ethoxylated surfactants and polyethylene glycols) are highly susceptible to being oxidized in the air. They then form complex mixtures of oxidation products. Animal testing reveals that whole the pure, non-oxidised surfactant is non-sensitizing, many of the oxidation products are sensitisers. The oxidization products also cause irritation.
ETHYLENE GLYCOL PHENYL ETHER & BENZYL ACETATE & DIHYDROMYRCENOL & LEMON OIL, EXTRACT & ETHYL LINALOOL & DIPENTENE & MYRCENE & OCTYL ALDEHYDE & SAFROLE	The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.
ETHYLENE GLYCOL PHENYL ETHER & BENZYL ACETATE	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.
CITRIC ACID, MONOHYDRATE & LEMONGRASS OIL & TERT-BUTYLHYDROQUINONE & BENZYL ACETATE & ALPHA-TERPINEOL & PIPERONYL PROPANAL & BENZYL SALICYLATE & MYRCENE & DECYL ALDEHYDE & CITRAL & CINNAMALDEHYDE & EUGENOL & BETA-PINENE & ALPHA-PINENE & ISOEUGENOL	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.
CITRIC ACID, MONOHYDRATE & OCTYL ALDEHYDE	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

Continued...

LEMONGRASS OIL & CITRAL	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.
LEMONGRASS OIL & LINALOOL & ORANGE OIL & MYRCENE & 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.
LEMONGRASS OIL & LINALOOL & ALPHA-HEXYLCINNAMALDEHYDE & DECYL ALDEHYDE & CITRAL & CINNAMALDEHYDE & EUGENOL & ALPHA-PINENE & ISOEUGENOL	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.
TERT-BUTYLHYDROQUINONE & SAFROLE	Laboratory (in vitro) and animal studies show, exposure to the material may result in a possible risk of irreversible effects, with the possibility of producing mutation.

Continued...

BENZYL ACETATE & BENZYL SALICYLATE	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.
BENZYL ACETATE & EUGENOL & D-LIMONENE	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.
ALPHA-TERPINEOL & DIHYDROMYRCENOL & LINALOOL & ETHYL LINALOOL	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.
ALPHA-TERPINEOL & LINALOOL	A member or analogue of a group of aliphatic and alicyclic terpenoid tertiary alcohols and structurally related substances generally regarded as safe. Animal testing suggests that the acute toxicity of tertiary alcohols and related esters is extremely low. Genetic toxicity: Tests on bacterial and animal cells showed no evidence of genetic toxicity or potential to cause mutations. 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
BENZYL SALICYLATE & ALPHA-HEXYLCINNAMALDEHYDE & DECYL ALDEHYDE & CINNAMALDEHYDE & EUGENOL & EUGENYL ACETATE & ISOEUGENOL	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.

Continued...

DIHYDROMYRCENOL & LINALOOL & ETHYL LINALOOL	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.
LINALOOL & ETHYL LINALOOL	Inhalational exposure of mice and man to linalool caused slight sedative effects but a dose dependent response characteristic could not be determined. It may irritate the digestive tract, skin, nose and the eyes but is not considered to be a sensitiser. It is equally shown to cause kidneys and liver damage but no genetic or reproductive defect was observed.
LEMON OIL, EXTRACT & ORANGE OIL	The essential oils, oleoresins (solvent-free), and natural extractives (including distillates) derived from citrus fruits are generally recognized as safe (GRAS) for their intended use in foods for human consumption. Botanicals such as citrus are comprised of hundreds of ingredients, some of which have the potential to cause toxic effects; for example, bergapten (5-methoxypsoralen; 5-MOP) is a naturally occurring furocoumarin (psoralen) in bergamot oil that causes light-mediated toxicity. Acute toxicity: Animal testing shows that the acute toxicity of these substances is generally low via skin contact. Skin irritation: In animal testing, undiluted citrus essential oils caused varying degrees of irritation. In humans, no irritation was observed after applying a variety of these oils to skin. Eye irritation: There appeared to be no significant eye irritation in testing with these substances. Sensitisation: Testing in humans have shown that these substances generally do not cause sensitisation. However, among professional food handlers, some proportion (under 10%) had positive reactions to orange and lemon peel. Light-mediated toxicity and sensitization: Testing for this group of substances has yielded mixed results. Light-mediated toxicity and sensitization have been seen in several people exposed to bergamot oil or limes/lime juice. Cancer-causing potential: Animal testing showed that essential oils of citrus fruits promoted tumours. However, most were benign.
ALPHA-HEXYLCINNAMALDEHYDE & CINNAMALDEHYDE	Animal testing suggests that the toxicity through swallowing cinnamyl aldehyde derivatives is very low. The potential for toxicity through skin exposure is similarly low. Cinnamaldehyde and its alkyl-substituted derivatives do not directly cause mutations or genetic damage. However, animal testing suggests that they may result in poor development of the skull and kidney in the foetus. 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.
MYRCENE & CITRAL & BETA-CARYOPHYLLENE & D-LIMONENE	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.

Continued...

MYRCENE & BETA-CARYOPHYLLENE & D-LIMONENE & BETA-PINENE & ALPHA-PINENE	Monomethyltin chloride, thioglycolate esters, and tall oil ester reaction product: Monomethyltin trichloride (MMTC, CAS RN: 993-16-8), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA), CAS RN: 57583-34-3), monomethyltin tris[isooctylmercaptoacetate (MMT(IOTG), CAS RN: 54849-38-6) and methyltin reverse ester tallate reaction product (TERP, CAS RNs: 201687-58-3, 201687-57-2, 68442-12-6, 151436-98-5) are considered one category of compounds for mammalian studies via the oral route. The justification for this category is based on structural similarities and the demonstrated rapid conversion of all of the esters to the MMTC when placed in simulated mammalian gastric contents [0.07M HCl] under physiological conditions. For the MMT(EHTG) >90% conversion to MMTC occurred within 0.5 hours. For TERP, 68% of the monomethyltin portion of the compound was converted to MMTC within 1 hour. Thus, MMTC is the appropriate surrogate for mammalian toxicology studies via the oral route. TERP is a reaction product of MMTC and dimethyltin dichloride (DMTC), Na2S, and tall oil fatty acid [a mixture of carboxylic acids, predominantly C-18]. The reaction product is a mixture of carboxylic esters and includes short oligomers of mono/dimethyltins bridged by sulfide groups. Although the tall oil component of TERP is not structurally similar to EHTG, TERP s conversion to MMTC justifies its inclusion. While the contribution of the various ligands to the overall toxicity may vary, the contribution is expected to be small relative to that of the MMTC. Further, the EHTG ligand from MMT(EHTG) is likely to be more toxic than the oleic or linoleic acid from TERP so inclusion of TERP in the category is a rather conservative approach. The other possible degradate of tall oil and EHTG is 2-mercaptoethanol (2-ME), and it is common to both ligands. Data for MMT(EHTG) and MMT(IOTG) are used interchangeably because they are isomers differing only slightly in the structure of the C-8 alcohol of the mercaptoester ligand. In addition, the breakdown products of MMT(EHTG) and MMT(IOTG) are the thioglycolate esters (EHTG and IOTG), which have the common degradates, thioglycolic acid and C-8 alcohols (either 2-ethylhexanol or isooctanol). EHTG and IOTG also have similar physicochemical and toxicological properties. The chemistry of the alkyl organotins has been well studied. For organotins, like MMT(EHTG), the alkyl groups are strongly bound to tin and remain bound to tin under most reaction conditions. However, other ligands, such as carboxylates or sulfur based ligands (EHTG), are more labile and are readily replaced under mild reaction conditions. To assess the reactivity of MMT(EHTG) under physiological conditions simulating the mammalian stomach, an in-vitro hydrolysis test was performed. This in vitro test provides chemical information that strongly suggests both the probable in vivo metabolic pathway and the toxicokinetics of the MMT(EHTG) substance. This result verifies that under physiological conditions

Continued...

MMT(EHTG) is rapidly and essentially completely converted to the corresponding monomethyltin chloride, MMTC.
Acute toxicity:
The majority of toxicology studies were conducted with commercial mixtures having high monoalkyltin to dialkyltin ratios.
Gastric hydrolysis studies were conducted with TERP and MMT(EHTG) in which simulated gastric fluid [0.07M HCl under physiological conditions] converted these substances to methyltin chloride and the respective organic acids. Based on data for DMTC and DMT esters the dermal penetration of MMTC and its esters is expected to be low.
Oral:
Acute oral LD50 values for MMTC, MMT(EHTG), MMT(IOTG), and TERP indicated low to moderate toxicity; the most reliable data place the LD50s in the range of 1000 mg/kg.
The acute oral LD50 of MMT(2-EHMA) was 880 mg/kg in rats. Clinical observations included depression, comatose, piloerection, eye squinting, hunched posture, laboured breathing, ataxia, faecal/urine stains, and masticatory movement. No gross pathological changes were reported in surviving animals.
Dermal
Acute dermal LD50 values were =1000 mg/kg bw, and inhalation LC50 was >200 mg/L. MMTC was corrosive to skin and assumed corrosive to eyes.
The acute dermal LD50 of MMT(2-EHMA) in rabbits was 1000 (460 to 2020) mg/kg for females and 2150 (1000 to 4620) mg/kg for males. There were no deaths at 215 and 464 mg/kg, 0/2 males and 1/2 females died at 1000 mg/kg and 1/2 males and 2/2 females died at 2150 mg/kg. All animals died at 4640 and 10 000 mg/kg. A variety of clinical abnormalities were observed and disappeared in surviving animals by the end of the exposure period. Clinical signs included death, uncoordinated movements, shaking, and hypersensitivity to external stimuli.
Gross necropsy results for animals that died during the study included irritated intestines; blanched stomach; reddened lungs; pale or congested kidneys; and oral, ocular and/or nasal discharges
Inhalation:
The acute inhalation LC50 of MMT(2-EHMA) was 240 mg/L.
The study reported an acute inhalation LC50 of 240 (212 to 271) mg/L in a 1-hr aerosol exposure to male and female rats. The mortality rate was 2/10, 6/10, 9/10 and 10/10 animals at dose levels of 200, 250, 300 and 250 mg/L/hr, respectively. Gross findings included blood in lungs, dark spleen, pale kidneys, fluid in the chest cavity, and heart failure. The slope of the dose-response curve was 1.22 (1.04 to 1.43).
Irritation:
MMT(IOTG)/(EHTG) are irritating to skin, but not to eyes.
Sensitisation:
No data on sensitization are available on MMT(EHTG/(IOTG), but the hydrolysis products EHTG or IOTG are sensitizers. No primary irritation data were available for TERP, but it was a sensitizer in the mouse Local Lymph Node Assay.
Topical application with 5, 25 and 50 % v/v MMT(2-EHMA) elicited a stimulation index (SI) of 2.13, 7.25 and 9.05, respectively in a local lymph node assay (OECD 429), thus the material is a sensitiser.
Repeat dose toxicity:
There are no repeated-dose studies for the category members via the dermal or inhalation routes.
In a 90-day repeated dose oral study of MMTC, treatment-related changes were limited to the high dose group (750 ppm in diet; 50 mg/kg bw/d with some gender-related variation). Organ weight changes (adrenal, kidney, thymus, spleen, brain, epididymides), haematology, clinical chemistry, and urinalysis changes were noted, but histopathology only confirmed effects in the thymus and brain. The critical toxic effects were neurotoxicity and thymic atrophy. Both sexes had decreased cortex/medulla ratios in the thymus. In the brain there was loss of perikarya of neuronal cells in the pyramidal layer of the Hippocampus CA1/2 in both sexes, and in males there was loss of perikarya in the piriform cortex. The NOAEL was 150 ppm (10 mg/kg bw/d). Another 90-day dietary study using MMTC showed increased relative kidney weights and slight to moderate epithelial hyperplasia of the bladder in females at the lowest dose (NOAEL <20 ppm in diet [<1-3.6 mg/kg bw/d]) and additional effects including increased relative thymus weights in females and urinalysis results in both sexes at higher doses.
A 90-day dietary study with dose levels of 30, 100, 300, and 1000 ppm TERP in the diet resulted in slightly decreased food intake, body and organ weight changes, and decreased specific gravity of the urine at the highest dose. The NOAEL was 300 ppm in diet (equivalent to 15 mg/kg bw/d). A 28-day gavage study using TERP showed changes in clinical chemistry and slight differences in haematology at 150 mg/kg bw/d and higher. The NOAEL was 50 mg/kg bw/d.

Continued...

	The effects of MMT(IOTG) were evaluated in a 90-day dietary study using doses of 100, 500, and 1500 ppm (decreased from 2500 ppm) in the diet. Based on clinical chemistry effects at 500 ppm and other effects at higher doses, the NOAEL was 100 ppm in diet (approximately 6-21 mg/kg bw/d). Neurotoxicity: In a guideline 90-day subchronic dietary study conducted in Wistar rats, effects occurred at the high dose of 750 ppm MMT(2-EHMA, (equivalent to 49.7 mg/kg bw/day in males and 53.6 mg/kg bw/day in females), which consisted of changes in neurobehavioral parameters and associated brain histopathology. The NOAEL was the next lower dose of 150 ppm (equivalent to 9.8 mg/kg bw/day in males and 10.2 mg/kg bw/day in females Immunotoxicity: Immune function was assessed in male Sprague-Dawley rats exposed to the mixture of organotins used in PVC pipe production. Adult male rats were given drinking water for 28 d containing a mixture of dibutyltin dichloride (DBTC), dimethyltin dichloride (DMTC), monobutyltin trichloride (MBT), and monomethyltin trichloride (MMT) in a 2:2:1:1 ratio, respectively, at 3 different concentrations (5:5:2.5:2.5, 10:10:5:5, or 20:20:10:10 mg organotin/L). Rats were also exposed to MMT alone (20 or 40 mg MMT/L) or plain water as a control. Delayed-type hypersensitivity, antibody synthesis, and natural killer cell cytotoxicity were evaluated in separate endpoint groups immediately after exposure ended. The evaluated immune functions were not affected by the mixture or by MMT alone. The data suggest that immunotoxicity is unlikely to result from the concentration of organotins present in drinking water delivered via PVC pipes, as the concentrations used were several orders of magnitude higher than those expected to leach from PVC pipes Genotoxicity: In a guideline 90-day subchronic dietary study in rats,with MMT(2-EHMA), based on the changes in neurobehavioral parameters and associated brain histopathology that occurred at the high dose of 750 ppm (equivalent to 49.7 mg/kg bw/day in males and 53.6 mg/kg bw/day in females), as well as changes in haematology, clinical chemistry, urinalysis, organ weights, and pathology of the thymus at the same dose, the NOAEL was the next lower dose of 150 ppm (equivalent to 9.8 mg/kg bw/day in males and 10.2 mg/kg bw/day in females). The monomethyltin compounds as a class are not mutagenic in the Ames test. TERP was positive in a human lymphocyte assay. MMTC was equivocal for induction of micronucleated polychromatic erythrocytes (MPEs) in an in vivo rat micronucleus test (OECD 474). In this study a statistically significant increase in MPE was observed only at 24 h and not at 48 h after treatment and there was no dose-response. Based on these observations the overall conclusion is that MMTC does not have genotoxic potential. From the results obtained in a micronucleus test with MMT(2-EHMA), it was demonstrated that the substance was weakly genotoxic to bone marrow cells of rats and that the substance has the potential to induce damage to the mitotic spindle apparatus of the bone marrow target cells. Carcinogenicity: In a limited carcinogenicity study, MMT(EHTG) produced no compound-related macroscopic or microscopic changes in rats fed 100 ppm in the diet for two years. Toxicity to reproduction: In the reproductive satellite portion of the 90-day study using MMTC (with dose levels of 30, 150, and 750 ppm in the diet), post-implantation loss, decreased litter size and increased neonatal mortality occurred at 750 ppm (26-46 mg/kg bw/d for females). Maternal gestational body weights were transiently suppressed and other maternal toxicity was inferred from the repeated dose results at this dose. There were no malformations observed at any dose. The NOAEL for maternal toxicity, and reproductive, and foetotoxic effects was 150 ppm in the diet (6-12 mg/kg bw/d). SIDS Inital Assessment Profile (SIAM 23 2006) ECHA Registration Dossier for MMT(2-EHMA) (ethylhexyl 10-ethyl-4-[[2-[(2-ethylhexyl)oxy]-2-oxoethyl]thio]-4-methyl-7-oxo-8-oxa-3,5-dithia-4-stannatetradecanoate)
OCTYL ALDEHYDE & DECYL 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

Continued...

	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.
BETA-PINENE & ALPHA-PINENE	Bicyclic terpenes are very low in acute toxicity. However, repeated dosing may have deleterious effects on the liver and kidney. Members of this category show no significant reproductive or developmental toxicity and may have a little, if any, potential to alter genetic material.

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

Legend: ✗ *– Data either not available or does not fill the criteria for classification*
✓ *– No data available for the final mixture, but the level of individual ingredients are considered in the overall property.*

SECTION 12 Ecological information

Toxicity

Room Spray (Holiday Berry)

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

water

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

castor oil, hydrogenated, ethoxylated

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

sodium citrate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>50mg/l	2
EC50	96h	Algae or other aquatic plants	>18000-32000mg/l	1
EC50(ECx)	48h	Crustacea	>50mg/l	2

butyl alcohol propoxylated

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>500mg/l	1
EC50	72h	Algae or other aquatic plants	>500mg/l	1
EC50	96h	Algae or other aquatic plants	744.74mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	62.5mg/l	2
LC50	96h	Fish	1350mg/l	1

Continued...

LC50	96h	Fish	564mg/l	2
EC50	48h	Crustacea	>100mg/l	2
EC50	72h	Algae or other aquatic plants	445mg/l	2
NOEC(ECx)	96h	Algae or other aquatic plants	<15.9mg/l	2
EC50	96h	Algae or other aquatic plants	315mg/l	2
EC50	48h	Crustacea	89-101mg/L	4
EC50(ECx)	48h	Crustacea	89-101mg/L	4
LC50	96h	Fish	48-52mg/L	4

ethylene glycol phenyl ether

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	460mg/l	2
EC50	72h	Algae or other aquatic plants	>100mg/l	2
NOEC(ECx)	24h	Fish	5mg/l	2
LC50	96h	Fish	154mg/l	2

citric acid, monohydrate

Endpoint	Test Duration (hr)	Species	Value	Source
EC10(ECx)	24h	Algae or other aquatic plants	>1000mg/l	4

ethylhexylglycerin

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	78.3mg/l	2
NOEC(ECx)	72h	Fish	<1.5mg/l	2
EC50	72h	Algae or other aquatic plants	48.28mg/l	2
LC50	96h	Fish	60.2mg/l	2

alpha-tocopherol

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	>10mg/l	2
EC50	48h	Crustacea	>23.53mg/l	2
EC50	72h	Algae or other aquatic plants	>25.8mg/l	2
NOEC(ECx)	384h	Fish	1mg/l	4

lemongrass oil

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

tert-butylhydroquinone

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	0.57mg/l	2
NOEC(ECx)	672h	Fish	0.004mg/l	2
LC50	96h	Fish	0.6mg/l	Not Available

benzyl acetate

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

alpha-terpineol

Endpoint	Test Duration (hr)	Species	Value	Source

Continued...

	EC50	48h	Crustacea	73mg/l	2
	EC50	72h	Algae or other aquatic plants	~17mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	~3.9mg/l	2
	LC50	96h	Fish	6.3 mg/l	4

piperonyl propanal

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	8.3mg/l	2
EC50	72h	Algae or other aquatic plants	14mg/l	2
NOEC(ECx)	96h	Fish	2.4mg/l	2
LC50	96h	Fish	5.3mg/l	2

benzyl salicylate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	1.16mg/l	2
EC50	72h	Algae or other aquatic plants	0.691mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	0.502mg/l	2
LC50	96h	Fish	1.03mg/l	2

dihydromyrcenol

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

linalool

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

lemon oil, extract

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	7.2mg/l	2
NOEC(ECx)	48h	Crustacea	3.8mg/l	2

orange oil

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

ethyl linalool

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	23mg/l	2
EC50	72h	Algae or other aquatic plants	13.3mg/l	2
NOEC(ECx)	48h	Crustacea	3.2mg/l	2
LC50	96h	Fish	24mg/l	2

alpha-hexylcinnamaldehyde

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>0.36<0.59mg/l	2
EC50	72h	Algae or other aquatic plants	>0.065mg/l	2
NOEC(ECx)	504h	Crustacea	0.063mg/L	2

Continued...

	LC50	96h	Fish	~1.7mg/l	2

dipropylene glycol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>100mg/l	2
EC50	72h	Algae or other aquatic plants	>100mg/l	2
EC50	96h	Algae or other aquatic plants	968mg/l	2
EC0(ECx)	48h	Crustacea	>100mg/l	2
LC50	96h	Fish	>1000mg/l	2

dipentene

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	28.2mg/l	4
EC50(ECx)	24h	Fish	~17.2mg/L	4
LC50	96h	Fish	35.4-41.8mg/l	4

myrcene

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	1.47mg/l	2
EC50	72h	Algae or other aquatic plants	0.31mg/l	2
EC50(ECx)	72h	Algae or other aquatic plants	0.31mg/l	2

octyl aldehyde

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	1.54mg/l	2
EC50	72h	Algae or other aquatic plants	0.42mg/l	2
EC50(ECx)	72h	Algae or other aquatic plants	0.42mg/l	2

decyl aldehyde

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	1.17mg/l	2
EC50	72h	Algae or other aquatic plants	1.79mg/l	2
NOEC(ECx)	48h	Crustacea	0.588mg/l	2
LC50	96h	Fish	1.45mg/l	2

citral

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

cinnamaldehyde

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	3.21mg/l	2
EC50	72h	Algae or other aquatic plants	4.07mg/l	2
EC50(ECx)	504h	Crustacea	0.402mg/l	2
LC50	96h	Fish	1.313-2.352mg/L	4

eugenol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	1.05mg/l	2

Continued...

	EC50	72h	Algae or other aquatic plants	23mg/l	2
	EC0(ECx)	48h	Crustacea	0.36mg/l	2
	LC50	96h	Fish	13mg/l	2

beta-caryophyllene

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>0.17mg/l	2
EC50	72h	Algae or other aquatic plants	>0.033mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	>=0.033mg/l	2

d-limonene

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	0.46mg/l	2
EC50	48h	Crustacea	0.307mg/l	2
EC50	72h	Algae or other aquatic plants	0.214mg/l	2
NOEC(ECx)	0h	Algae or other aquatic plants	<0.05-1.5mg/L	4

beta-pinene

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	1440h	Fish	0.058mg/L	4
EC50	48h	Crustacea	1.09mg/l	2
EC50	72h	Algae or other aquatic plants	0.7mg/l	2
NOEC(ECx)	96h	Algae or other aquatic plants	<0.02-0.65mg/L	4
LC50	96h	Fish	0.402-0.625mg/L	4

eugenyl acetate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	33mg/l	2
EC50	72h	Algae or other aquatic plants	26mg/l	2
EC50	96h	Algae or other aquatic plants	6.727mg/l	2
EC50(ECx)	96h	Algae or other aquatic plants	6.727mg/l	2

safrole

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

alpha-pinene

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	0.475mg/l	2
EC50	72h	Algae or other aquatic plants	0.31mg/l	2
NOEC(ECx)	48h	Algae or other aquatic plants	0.131mg/l	2
LC50	96h	Fish	0.303mg/l	2

isoeugenol

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

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
water	LOW	LOW
butyl alcohol propoxylated	LOW	LOW

Continued...

ethylene glycol phenyl ether	LOW	LOW
citric acid, monohydrate	LOW	LOW
alpha-tocopherol	HIGH	HIGH
tert-butylhydroquinone	HIGH	HIGH
benzyl acetate	LOW	LOW
alpha-terpineol	HIGH	HIGH
piperonyl propanal	HIGH	HIGH
benzyl salicylate	HIGH	HIGH
dihydromyrcenol	HIGH	HIGH
linalool	HIGH	HIGH
ethyl linalool	HIGH	HIGH
alpha-hexylcinnamaldehyde	LOW	LOW
dipropylene glycol	LOW	LOW
dipentene	HIGH	HIGH
myrcene	HIGH	HIGH
octyl aldehyde	LOW	LOW
decyl aldehyde	LOW	LOW
citral	LOW	LOW
cinnamaldehyde	LOW	LOW
eugenol	HIGH	HIGH
beta-caryophyllene	HIGH	HIGH
d-limonene	HIGH	HIGH
beta-pinene	HIGH	HIGH
eugenyl acetate	LOW	LOW
safrole	LOW (Half-life = 56 days)	LOW (Half-life = 0.25 days)
alpha-pinene	HIGH	HIGH
isoeugenol	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
water	LOW (LogKOW = -1.38)
sodium citrate	LOW (LogKOW = -0.28)
butyl alcohol propoxylated	LOW (LogKOW = 1.2706)
ethylene glycol phenyl ether	LOW (LogKOW = 1.16)
citric acid, monohydrate	LOW (LogKOW = -1.64)
alpha-tocopherol	LOW (LogKOW = 12.18)
lemongrass oil	LOW (LogKOW = 3.45)
tert-butylhydroquinone	LOW (LogKOW = 2.94)
benzyl acetate	LOW (LogKOW = 1.96)
alpha-terpineol	LOW (LogKOW = 3.28)
piperonyl propanal	LOW (LogKOW = 2.51)
benzyl salicylate	MEDIUM (LogKOW = 4.31)
dihydromyrcenol	LOW (LogKOW = 3.47)

Continued...

linalool	LOW (LogKOW = 2.97)
orange oil	MEDIUM (LogKOW = 4.38)
ethyl linalool	MEDIUM (LogKOW = 3.87)
alpha-hexylcinnamaldehyde	HIGH (LogKOW = 4.82)
dipropylene glycol	LOW (BCF = 4.6)
dipentene	MEDIUM (LogKOW = 4.38)
myrcene	MEDIUM (LogKOW = 4.17)
octyl aldehyde	LOW (LogKOW = 2.78)
decyl aldehyde	LOW (LogKOW = 3.76)
citral	LOW (LogKOW = 3.45)
cinnamaldehyde	LOW (BCF = 10)
eugenol	LOW (LogKOW = 2.27)
beta-caryophyllene	HIGH (LogKOW = 6.3)
d-limonene	HIGH (LogKOW = 4.8275)
beta-pinene	MEDIUM (LogKOW = 4.16)
eugenyl acetate	LOW (LogKOW = 3.06)
safrole	LOW (LogKOW = 3.45)
alpha-pinene	MEDIUM (LogKOW = 4.44)
isoeugenol	LOW (LogKOW = 3.04)

Mobility in soil

Ingredient	Mobility
butyl alcohol propoxylated	LOW (Log KOC = 10)
ethylene glycol phenyl ether	LOW (Log KOC = 12.12)
citric acid, monohydrate	LOW (Log KOC = 10)
alpha-tocopherol	LOW (Log KOC = 51280000)
tert-butylhydroquinone	LOW (Log KOC = 3162)
benzyl acetate	LOW (Log KOC = 133.7)
alpha-terpineol	LOW (Log KOC = 57.85)
piperonyl propanal	LOW (Log KOC = 56.07)
benzyl salicylate	LOW (Log KOC = 5156)
dihydromyrcenol	LOW (Log KOC = 54.78)
linalool	LOW (Log KOC = 56.32)
ethyl linalool	LOW (Log KOC = 182.1)
alpha-hexylcinnamaldehyde	LOW (Log KOC = 4025)
dipropylene glycol	HIGH (Log KOC = 1)
dipentene	LOW (Log KOC = 1324)
myrcene	LOW (Log KOC = 1269)
octyl aldehyde	LOW (Log KOC = 58.97)
decyl aldehyde	LOW (Log KOC = 200.6)
citral	LOW (Log KOC = 147.7)
cinnamaldehyde	LOW (Log KOC = 102.4)
eugenol	LOW (Log KOC = 1124)

Continued...

beta-caryophyllene	LOW (Log KOC = 22290)
d-limonene	LOW (Log KOC = 1324)
beta-pinene	LOW (Log KOC = 1204)
eugenyl acetate	LOW (Log KOC = 304.1)
safrole	LOW (Log KOC = 297.5)
alpha-pinene	LOW (Log KOC = 1204)
isoeugenol	LOW (Log KOC = 1124)

Other adverse effects

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

SECTION 13 Disposal considerations

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. Valuable substance, hold all residues for recovery. Disposal of the material must be carried out in accordance with the requirements of the relevant Federal/State Act(s) or Code(s) regulating the disposal of Drugs of Addiction. ‣ Consult manufacturer/supplier for recycling options. ‣ Decontaminate empty containers with water; incinerate plastic bags. ‣ **DO NOT** reuse containers. Bury empty containers in an authorised landfill. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ‣ Reduction ‣ Reuse ‣ Recycling ‣ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ‣ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ‣ It may be necessary to collect all wash water for treatment before disposal. ‣ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ‣ Where in doubt contact the responsible authority. ‣ Recycle wherever possible. ‣ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ‣ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible material). ‣ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO

Continued...

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

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

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

14.7. Maritime transport in bulk according to IMO instruments

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

Not Applicable

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

Product name	Group
water	Not Available
castor oil, hydrogenated, ethoxylated	Not Available
sodium citrate	Not Available
butyl alcohol propoxylated	Not Available
ethylene glycol phenyl ether	Not Available
citric acid, monohydrate	Not Available
ethylhexylglycerin	Not Available
alpha-tocopherol	Not Available
lemongrass oil	Not Available
tert-butylhydroquinone	Not Available
benzyl acetate	Not Available
alpha-terpineol	Not Available
piperonyl propanal	Not Available
benzyl salicylate	Not Available
dihydromyrcenol	Not Available
linalool	Not Available
lemon oil, extract	Not Available
orange oil	Not Available
ethyl linalool	Not Available
alpha-hexylcinnamaldehyde	Not Available
dipropylene glycol	Not Available
dipentene	Not Available
myrcene	Not Available
octyl aldehyde	Not Available
decyl aldehyde	Not Available
citral	Not Available
cinnamaldehyde	Not Available
eugenol	Not Available
beta-caryophyllene	Not Available
d-limonene	Not Available
beta-pinene	Not Available
eugenyl acetate	Not Available
safrole	Not Available

Continued...

alpha-pinene	Not Available
isoeugenol	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
water	Not Available
castor oil, hydrogenated, ethoxylated	Not Available
sodium citrate	Not Available
butyl alcohol propoxylated	Not Available
ethylene glycol phenyl ether	Not Available
citric acid, monohydrate	Not Available
ethylhexylglycerin	Not Available
alpha-tocopherol	Not Available
lemongrass oil	Not Available
tert-butylhydroquinone	Not Available
benzyl acetate	Not Available
alpha-terpineol	Not Available
piperonyl propanal	Not Available
benzyl salicylate	Not Available
dihydromyrcenol	Not Available
linalool	Not Available
lemon oil, extract	Not Available
orange oil	Not Available
ethyl linalool	Not Available
alpha-hexylcinnamaldehyde	Not Available
dipropylene glycol	Not Available
dipentene	Not Available
myrcene	Not Available
octyl aldehyde	Not Available
decyl aldehyde	Not Available
citral	Not Available
cinnamaldehyde	Not Available
eugenol	Not Available
beta-caryophyllene	Not Available
d-limonene	Not Available
beta-pinene	Not Available
eugenyl acetate	Not Available
safrole	Not Available
alpha-pinene	Not Available
isoeugenol	Not Available

Continued...

SECTION 15 Regulatory information

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

water is found on the following regulatory lists

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

castor oil, hydrogenated, ethoxylated is found on the following regulatory lists

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

sodium citrate is found on the following regulatory lists

US DOE Temporary Emergency Exposure Limits (TEELs)

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

butyl alcohol propoxylated is found on the following regulatory lists

US - California Hazardous Air Pollutants Identified as Toxic Air Contaminants

US - Pennsylvania - Hazardous Substance List

Continued...

US DOE Temporary Emergency Exposure Limits (TEELs)
US EPA Integrated Risk Information System (IRIS)
US EPCRA Section 313 Chemical List
US New York City Community Right-to-Know: List of Hazardous Substances
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Section 4/12 (b) - Sunset Dates/Status

ethylene glycol phenyl ether is found on the following regulatory lists

US - California Hazardous Air Pollutants Identified as Toxic Air Contaminants
US - Pennsylvania - Hazardous Substance List
US DOE Temporary Emergency Exposure Limits (TEELs)
US EPCRA Section 313 Chemical List
US New York City Community Right-to-Know: List of Hazardous Substances
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

citric acid, monohydrate is found on the following regulatory lists

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

ethylhexylglycerin is found on the following regulatory lists

Not Applicable

alpha-tocopherol is found on the following regulatory lists

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

lemongrass oil is found on the following regulatory lists

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

tert-butylhydroquinone is found on the following regulatory lists

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

benzyl acetate is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
US - New Jersey Right to Know Hazardous Substances
US DOE Temporary Emergency Exposure Limits (TEELs)
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

alpha-terpineol is found on the following regulatory lists

US DOE Temporary Emergency Exposure Limits (TEELs)
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

piperonyl propanal is found on the following regulatory lists

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

benzyl salicylate is found on the following regulatory lists

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

dihydromyrcenol is found on the following regulatory lists

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

linalool is found on the following regulatory lists

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

lemon oil, extract is found on the following regulatory lists

Not Applicable

orange oil is found on the following regulatory lists

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

ethyl linalool is found on the following regulatory lists

Continued...

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

alpha-hexylcinnamaldehyde is found on the following regulatory lists

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

dipropylene glycol is found on the following regulatory lists

US - Pennsylvania - Hazardous Substance List

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

dipentene is found on the following regulatory lists

US - New Jersey Right to Know Hazardous Substances

US New York City Community Right-to-Know: List of Hazardous Substances

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

US Toxicology Excellence for Risk Assessment (TERA) Workplace Environmental Exposure Levels (WEEL)

myrcene is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

US - California Proposition 65 - Carcinogens

US - California Safe Drinking Water and Toxic Enforcement Act of 1986 - Proposition 65 List

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

octyl aldehyde is found on the following regulatory lists

US - Massachusetts - Right To Know Listed Chemicals

US DOE Temporary Emergency Exposure Limits (TEELs)

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

decyl aldehyde is found on the following regulatory lists

US DOE Temporary Emergency Exposure Limits (TEELs)

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

citral is found on the following regulatory lists

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

cinnamaldehyde is found on the following regulatory lists

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

eugenol is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

beta-caryophyllene is found on the following regulatory lists

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

d-limonene is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

US - New Jersey Right to Know Hazardous Substances

US AIHA Workplace Environmental Exposure Levels (WEELs)

US DOE Temporary Emergency Exposure Limits (TEELs)

US EPA Integrated Risk Information System (IRIS)

US New York City Community Right-to-Know: List of Hazardous Substances

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

US Toxicology Excellence for Risk Assessment (TERA) Workplace Environmental Exposure Levels (WEEL)

beta-pinene is found on the following regulatory lists

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

Continued...

eugenyl acetate is found on the following regulatory lists

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

safrole is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
US - California Proposition 65 - Carcinogens
US - California Proposition 65 - No Significant Risk Levels (NSRLs) for Carcinogens
US - California Safe Drinking Water and Toxic Enforcement Act of 1986 - Proposition 65 List
US - Massachusetts - Right To Know Listed Chemicals
US - New Jersey Right to Know - Special Health Hazard Substance List (SHHSL): Carcinogens
US - New Jersey Right to Know Hazardous Substances
US - Pennsylvania - Hazardous Substance List
US Clean Air Act - Hazardous Air Pollutants
US DOE Temporary Emergency Exposure Limits (TEELs)
US Drug Enforcement Administration (DEA) List I and II Regulated Chemicals
US EPCRA Section 313 Chemical List
US National Toxicology Program (NTP) 15th Report Part B. Reasonably Anticipated to be a Human Carcinogen
US New York City Community Right-to-Know: List of Hazardous Substances
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

alpha-pinene is found on the following regulatory lists

US - Massachusetts - Right To Know Listed Chemicals
US - New Jersey Right to Know - Special Health Hazard Substance List (SHHSL): Flammables
US - New Jersey Right to Know Hazardous Substances
US - Pennsylvania - Hazardous Substance List
US DOE Temporary Emergency Exposure Limits (TEELs)
US New York City Community Right-to-Know: List of Hazardous Substances
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

isoeugenol is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

Additional Regulatory Information

Not Applicable

Continued...

SECTION 16 Other information

Other information

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.

End of SDS