Room spray (Spiced Chestnut)

Ming Fai Enterprise International Co., Ltd

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

SDS No.: HKGH0325528608
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SECTION 1 Identification

Product Identifier

Product name	Room spray (Spiced Chestnut) Contains: cinnamaldehyde
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 1A

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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
9038-95-3	1-5	butyl alcohol propoxylated
Not Applicable	0.5-1	Fragrance – BT573552
122-99-6	0.5-0.99	ethylene glycol phenyl ether

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25265-71-8	0.5-0.99	dipropylene glycol (as part of fragrance BT573552 and BT312972)
Not Applicable	0.1-0.5	Fragrance – BT66197
5989-27-5	0.1-0.5	d-limonene (as part of fragrance BT66197, AAL10620/00 and Cinnamon Oil)
77-92-9	0.1-0.5	citric acid
Not Applicable	0.1-0.5	Fragrance – BT312972
97-53-0	0.1-0.5	eugenol (as part of fragrance BT66197, Cinnamon Oil and Clove Bud Oil)
Not Applicable	0.1-0.5	Fragrance – Clove Bud Oil
24851-98-7	0.1-0.5	methyl dihydrojasmonate (as part of fragrance BT573552)
Not Applicable	0.05-0.1	Fragrance – Cinnamon Oil
70445-33-9	0.05-0.1	ethylhexylglycerin
104-55-2	0.05-0.1	cinnamaldehyde (as part of fragrance BT66197 and Cinnamon Oil)
89-78-1	0.05-0.1	menthol (as part of fragrance BT66197)
Not Applicable	0.01-0.05	Fragrance – AAL10620/00
3100-36-5	0.01-0.05	8-cyclohexadecen-1-one (as part of fragrance BT573552)
106-02-5	0.01-0.05	omega-pentadecalactone (as part of fragrance BT573552)
8015-91-6	0.01-0.05	cinnamon oil, Ceylon (as part of fragrance BT66197)
54464-57-2	0.01-0.05	isocyclemone E (as part of fragrance BT312972)
78-70-6	0.01-0.05	linalool (as part of fragrance BT66197, BT312972, AAL10620/00 and Cinnamon Oil)
93-28-7	0.01-0.05	eugenyl acetate (as part of fragrance Cinnamon Oil and Clove Bud Oil)
1222-05-5	0.01-0.05	galaxolide (as part of fragrance BT573552 and AAL10620/00)
87-44-5	0.01-0.05	beta-caryophyllene (as part of fragrance Cinnamon Oil and Clove Bud Oil)
128-37-0	0.01-0.05	2,6-di-tert-butyl-4-methylphenol (as part of fragrance BT573552 and BT66197)
18479-58-8	0.01-0.05	dihydromyrcenol (as part of fragrance BT312972 and AAL10620/00)
28219-61-6	0.01-0.05	ethyl-4-trimethylcyclopentenyl-2-butenol (as part of fragrance BT312972 and AAL10620/00)
82356-51-2	0.005-0.01	3-methylcyclopentadecenone (mixed isomers) (as part of fragrance BT573552)
8007-80-5	0.005-0.01	cinnamon oil (as part of fragrance BT66197)
121-32-4	0.005-0.01	ethyl vanillin (as part of fragrance BT312972)
115-95-7	0.005-0.01	linalyl acetate (as part of fragrance BT312972)
8008-52-4	0.001-0.005	coriander oil (as part of fragrance BT66197)
8007-12-3	0.001-0.005	nutmeg oil, expressed (as part of fragrance BT66197)
88-41-5	0.001-0.005	2-tert-butylcyclohexyl acetate (as part of fragrance AAL10620/00)
5989-54-8	0.001-0.005	l-limonene (as part of fragrance Cinnamon Oil)
91-64-5	0.001-0.005	coumarin (as part of fragrance BT312972 and AAL10620/00)
121-33-5	0.001-0.005	vanillin (as part of fragrance BT312972)
8022-15-9	0.001-0.005	lavandin oil (as part of fragrance BT312972)
8008-79-5	0.001-0.005	spearmint oil (as part of fragrance BT66197)
101-86-0	0.001-0.005	alpha-hexylcinnamaldehyde (as part of fragrance AAL10620/00)
32210-23-4	0.001-0.005	4-tert-butylcyclohexyl acetate (as part of fragrance AAL10620/00)
1335-46-2	0.001-0.005	Ionone, methyl- (as part of fragrance AAL10620/00)
60-12-8	0.001-0.005	phenethyl alcohol (as part of fragrance AAL10620/00)
106-22-9	0.001-0.005	beta-citronellol (as part of fragrance AAL10620/00)
8007-75-8	0.001-0.005	bergamot oil (as part of fragrance BT312972)
127-51-5	0.001-0.005	methylionone, isomers (as part of fragrance BT312972)
8008-57-9	0.001-0.005	orange oil (as part of fragrance BT312972)
106-24-1	0.001-0.005	geraniol (as part of fragrance AAL10620/00)

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14901-07-6	0.001-0.005	beta-ionone (as part of fragrance AAL10620/00)
118-58-1	0.001-0.005	benzyl salicylate (as part of fragrance AAL10620/00)
104-67-6	0.001-0.005	gamma-undecalactone (as part of fragrance AAL10620/00)
127-91-3	0.0005-0.001	beta-pinene (as part of fragrance Cinnamon Oil)
94-59-7	0.0005-0.001	safrole (as part of fragrance Cinnamon Oil)
80-56-8	0.0005-0.001	alpha-pinene (as part of fragrance Cinnamon Oil)
97-54-1	0.0005-0.001	isoeugenol (as part of fragrance Cinnamon Oil)
1506-02-1	0.0001-0.0005	7-acetyl-1,1,3,4,4,6-hexamethyltetraline (as part of fragrance AAL10620/00)
2705-87-5	0.0001-0.0005	allyl cyclohexanepropionate (as part of fragrance AAL10620/00)
6485-40-1	0.0001-0.0005	carvone (as part of fragrance AAL10620/00)
112-54-9	0.0001-0.0005	dodecyl aldehyde (as part of fragrance AAL10620/00)
93-15-2	0.0001-0.0005	methyl eugenol (as part of fragrance Clove Bud Oil)
10191-41-0	<0.0001	alpha-tocopherol
123-35-3	<0.0001	myrcene (as part of fragrance AAL10620/00)
112-45-8	<0.0001	10-undecenal (as part of fragrance AAL10620/00)
103-95-7	<0.0001	p-isopropyl-alpha-methylhydrocinnamaldehyde (as part of fragrance AAL10620/00)
57378-68-4	<0.0001	rose ketones (as part of fragrance AAL10620/00)

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	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary.
Ingestion	▸ 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

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

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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 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.

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SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	‣ Avoid all personal contact, including inhalation. ‣ Wear protective clothing when risk of exposure occurs. ‣ Use in a well-ventilated area. ‣ Prevent concentration in hollows and sumps. ‣ **DO NOT** enter confined spaces until atmosphere has been checked. ‣ **DO NOT** allow material to contact humans, exposed food or food utensils. ‣ Avoid contact with incompatible materials. ‣ When handling, **DO NOT** eat, drink or smoke. ‣ Keep containers securely sealed when not in use. ‣ Avoid physical damage to containers. ‣ Always wash hands with soap and water after handling. ‣ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ‣ Use good occupational work practice. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS. ‣ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained. ‣ **DO NOT** allow clothing wet with material to stay in contact with skin
Other information	**NOTE**: Special security requirements may be mandated under Federal/State Regulation(s). ‣ Store in original containers. ‣ Store in vault fitted with warning devices or detectors recommended by various Federal/State authorities. ‣ Store in vault used only for the purpose of storage of drugs of addiction. ‣ Vault must be locked at all times except when the materials stored therein are required. ‣ Keep storage area free from debris, wastes and combustibles. ‣ Keep dry. ‣ Keep containers securely sealed. ‣ Protect containers against physical damage. ‣ Check regularly for spills and leaks.

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

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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, 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

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SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US OSHA Permissible Exposure Limits (PELs) Table Z-1	2,6-di-tert-butyl-4-methylphenol	Particulates Not Otherwise Regulated (PNOR)- Respirable fraction	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	2,6-di-tert-butyl-4-methylphenol	Particulates Not Otherwise Regulated (PNOR)- Total dust	15 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	2,6-di-tert-butyl-4-methylphenol	Inert or Nuisance Dust: Total Dust	15 mg/m3 / 50 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	2,6-di-tert-butyl-4-methylphenol	Inert or Nuisance Dust: Respirable fraction	5 mg/m3 / 15 mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	2,6-di-tert-butyl-4-methylphenol	2,6-Di-tert-butyl-p-cresol	10 mg/m3	Not Available	Not Available	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
d-limonene	15 ppm	67 ppm	170 ppm
coumarin	0.88 mg/m3	9.7 mg/m3	58 mg/m3
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
butyl alcohol propoxylated	Not Available	Not Available
ethylene glycol phenyl ether	Not Available	Not Available
citric acid	Not Available	Not Available
ethylhexylglycerin	Not Available	Not Available
alpha-tocopherol	Not Available	Not Available
2,6-di-tert-butyl-4-methylphenol	Not Available	Not Available
3-methylcyclopentadecenone (mixed isomers)	Not Available	Not Available
8-cyclohexadecen-1-one	Not Available	Not Available
omega-pentadecalactone	Not Available	Not Available

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galaxolide	Not Available	Not Available
methyl dihydrojasmonate	Not Available	Not Available
dipropylene glycol	Not Available	Not Available
coriander oil	Not Available	Not Available
nutmeg oil, expressed	Not Available	Not Available
spearmint oil	Not Available	Not Available
cinnamon oil	Not Available	Not Available
linalool	Not Available	Not Available
eugenol	Not Available	Not Available
cinnamaldehyde	Not Available	Not Available
cinnamon oil, Ceylon	Not Available	Not Available
menthol	Not Available	Not Available
d-limonene	Not Available	Not Available
orange oil	Not Available	Not Available
dihydromyrcenol	Not Available	Not Available
ethyl-4-trimethylcyclopentenyl-2-butenol	Not Available	Not Available
methylionone, isomers	Not Available	Not Available
bergamot oil	Not Available	Not Available
lavandin oil	Not Available	Not Available
vanillin	Not Available	Not Available
coumarin	Not Available	Not Available
linalyl acetate	Not Available	Not Available
ethyl vanillin	Not Available	Not Available
isocyclemone E	Not Available	Not Available
2-tert-butylcyclohexyl acetate	Not Available	Not Available
alpha-hexylcinnamaldehyde	Not Available	Not Available
4-tert-butylcyclohexyl acetate	Not Available	Not Available
Ionone, methyl-	Not Available	Not Available
phenethyl alcohol	Not Available	Not Available
beta-ionone	Not Available	Not Available
benzyl salicylate	Not Available	Not Available
geraniol	Not Available	Not Available
beta-citronellol	Not Available	Not Available
gamma-undecalactone	Not Available	Not Available
7-acetyl-1,1,3,4,4,6-hexamethyltetraline	Not Available	Not Available
allyl cyclohexanepropionate	Not Available	Not Available
myrcene	Not Available	Not Available
10-undecenal	Not Available	Not Available
p-isopropyl-alpha-methylhydrocinnamaldehyde	Not Available	Not Available
carvone	Not Available	Not Available
dodecyl aldehyde	Not Available	Not Available

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rose ketones	Not Available	Not Available
beta-caryophyllene	Not Available	Not Available
l-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
methyl eugenol	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. 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/

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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 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

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	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. ▸ Double gloving should be considered. ▸ PVC gloves.

Continued...

	‣ 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)

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

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

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

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

Continued...

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)	5.82	**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.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

Continued...

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. 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.
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. 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.

Continued...

Room spray (Spiced Chestnut)

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)
	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

Continued...

	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

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg	Eye (Rodent - rabbit): 750ug/24H - Severe
Oral (Rat) LD50: 3000 mg/kg	Eye: adverse effect observed (irritating)
	Skin (Rodent - rabbit): 0.5mL - Moderate
	Skin (Rodent - rabbit): 500mg/24H - Mild
	Skin: adverse effect observed (irritating)
	Skin: no adverse effect observed (not irritating)

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

2,6-di-tert-butyl-4-methylphenol

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg	Eye (Rodent - rabbit): 100mg/24H - Moderate
Oral (Rat) LD50: 890 mg/kg	Eye: no adverse effect observed (not irritating)
	Skin (Human): 500mg/48H - Mild
	Skin (Rodent - rabbit): 500mg/48H - Moderate
	Skin: no adverse effect observed (not irritating)

3-methylcyclopentadecenone (mixed isomers)

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg	Not Available

Continued...

	Oral (Rat) LD50: >2000 mg/kg	

8-cyclohexadecen-1-one

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >4600 mg/kg	Eye: no adverse effect observed (not irritating)
Oral (Rat) LD50: >10000 mg/kg	Skin: no adverse effect observed (not irritating)

omega-pentadecalactone

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

galaxolide

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

methyl dihydrojasmonate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg	Eye: no adverse effect observed (not irritating)
Inhalation (Rat) LC50: >4.93 mg/l4h	Skin: no adverse effect observed (not irritating)
Oral (Rat) LD50: >5000 mg/kg	

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)

coriander oil

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg	Skin (Rodent - rabbit): 500mg/24H
Oral (Rat) LD50: 4130 mg/kg	

nutmeg oil, expressed

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg	Skin (Rodent - rabbit): 500mg/24H - Moderate
Oral (Rat) LD50: 3640 mg/kg	

spearmint oil

TOXICITY	IRRITATION
Oral (Rat) LD50: 5000 mg/kg	Skin (Rodent - guinea pig): 100% - Mild
	Skin (Rodent - rabbit): 500mg/24H - Moderate

cinnamon oil

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 320 mg/kg	Eye: adverse effect observed (irreversible damage)
Oral (Mouse) LD50; 2670 mg/kg	Skin (Human): 100%
	Skin (Rodent - mouse): 100% - Mild

Continued...

	Skin (Rodent - rabbit): 500mg/24H - Severe
	Skin: adverse effect observed (corrosive)

linalool

TOXICITY	IRRITATION
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)

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)

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)

cinnamon oil, Ceylon

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg	Eye: adverse effect observed (irreversible damage)
Oral (Rat) LD50: 2650 mg/kg	Eye: adverse effect observed (irritating)
	Skin (Human): 100%
	Skin (Rodent - mouse): 100% - Mild
	Skin (Rodent - rabbit): 500mg/24H - Severe
	Skin: adverse effect observed (corrosive)
	Skin: no adverse effect observed (not irritating)

menthol

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg	Eye (Mammal - species unspecified): 0.7% - Mild
Inhalation (Rat) LC50: ~5.289 mg/L4h	Eye (Mammal - species unspecified): 100% - Severe

Continued...

	Oral (Cat) LD50; 800 mg/kg	Eye (Rodent - rabbit): 1%
		Eye (Rodent - rabbit): 1%
		Eye (Rodent - rabbit): 250ug - Severe
		Eye: adverse effect observed (irritating)
		Skin (Rodent - rabbit): 100%/24H - Mild
		Skin (Rodent - rabbit): 500mg/24H - Mild
		Skin: adverse effect observed (irritating)
		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)

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

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

ethyl-4-trimethylcyclopentenyl-2-butenol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg	Eye: adverse effect observed (irritating)
	Oral (Rat) LD50: 5000 mg/kg	Skin: no adverse effect observed (not irritating)

methylionone, isomers	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg	Eye: no adverse effect observed (not irritating)
	Oral (Rat) LD50: >5000 mg/kg	Skin (Human): 5%/2D
		Skin: adverse effect observed (irritating)

Continued...

		Skin: no adverse effect observed (not irritating)

bergamot oil

TOXICITY	IRRITATION
Oral (Rat) LD50: 11520 mg/kg	Skin (Rodent - rabbit): 500mg/24H - Mild

lavandin oil

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

vanillin

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg	Eye: adverse effect observed (irritating)
Inhalation (Rat) LC50: >0.042 mg/L4h	Skin: no adverse effect observed (not irritating)
Oral (Guinea) LD50; 1400 mg/kg	

coumarin

TOXICITY	IRRITATION
dermal (rat) LD50: 293 mg/kg	Skin (Human - man): 5%
Oral (Rat) LD50: ~290 mg/kg	Skin (Human): 5%/2D

linalyl acetate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg	Skin (Rodent - guinea pig): 100mg/24H - Moderate
Oral (Mouse) LD50; 12000 mg/kg	Skin (Rodent - rabbit): 100mg/24H - Severe

ethyl vanillin

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg	Eye: adverse effect observed (irritating)
Oral (Rat) LD50: 1590 mg/kg	Skin (Human): 10mg/48H - Mild
	Skin: no adverse effect observed (not irritating)

isocyclemone E

TOXICITY	IRRITATION
Oral (Rat) LD50: >5000 mg/kg	Not Available

2-tert-butylcyclohexyl acetate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg	Eye (Rodent - rabbit): 50% - Severe
Oral (Rat) LD50: 4600 mg/kg	Eye: no adverse effect observed (not irritating)
	Skin (Rodent - rabbit): 100%/4H - Moderate
	Skin: no adverse effect observed (not irritating)

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

Continued...

	Skin (Rodent - mouse): 30%/3D(intermittent)
	Skin (Rodent - rabbit): 100mg/24H - Severe
	Skin (Rodent - rabbit): 500mg/24H - Moderate
	Skin: adverse effect observed (irritating)

4-tert-butylcyclohexyl acetate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >4670 mg/kg	Eye: no adverse effect observed (not irritating)
Oral (Rat) LD50: >300<2000 mg/kg	Skin (Rodent - guinea pig): 3%/4H - Mild
	Skin (Rodent - rabbit): 100%/4H - Moderate
	Skin (Rodent - rabbit): 500mg/24H - Moderate
	Skin: no adverse effect observed (not irritating)

Ionone, methyl-

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg	Skin: adverse effect observed (irritating)
Oral (Rat) LD50: >5000 mg/kg	Skin: no adverse effect observed (not irritating)

phenethyl alcohol

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 790 mg/kg	Eye (Rodent - rabbit): 12gm/10M - Mild
Inhalation (Rat) LC50: >4.63 mg/l4h	Eye (Rodent - rabbit): 750ug/24H - Severe
Oral (Rat) LD50: 1603.3 mg/kg	Eye: adverse effect observed (irritating)
	Skin (Rodent - guinea pig): 100% - Mild
	Skin (Rodent - guinea pig): 100mg/24H - Moderate
	Skin (Rodent - rabbit): 100mg/24H - Moderate
	Skin: no adverse effect observed (not irritating)

beta-ionone

TOXICITY	IRRITATION
dermal (mouse) LD50: >2000-7000 mg/kg	Skin: no adverse effect observed (not irritating)
Inhalation (Rat) LC50: 176.859 ppm4h	
Oral (Rat) LD50: 4590 mg/kg	

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)

geraniol

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg	Eye: adverse effect observed (irreversible damage)
Oral (Rat) LD50: 3600 mg/kg	Skin (Human - man): 16mg/24H - Severe
	Skin (Human): 0.2mL/4H
	Skin (Human): 1%
	Skin (Human): 2%/2D
	Skin (Human): 32%/48H - Severe
	Skin (Rodent - guinea pig): 100mg/24H - Severe

Continued...

	Skin (Rodent - guinea pig): 30% - Mild
	Skin (Rodent - rabbit): 0.5mL/4H - Moderate
	Skin (Rodent - rabbit): 100mg/24H - Severe
	Skin: adverse effect observed (irritating)

beta-citronellol

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 2650 mg/kg	Eye (Rodent - rabbit): 0.42% - Moderate
Oral (Rat) LD50: 3450 mg/kg	Eye: adverse effect observed (irritating)
	Skin (Human - man): 16mg/48H - Moderate
	Skin (Human): 2%/2D
	Skin (Human): 32%/48H
	Skin (Human): 5%
	Skin (Human): 5%
	Skin (Mammal - pig): 100%/48H
	Skin (Rodent - guinea pig): 100%/48H
	Skin (Rodent - guinea pig): 100mg/24H - Severe
	Skin (Rodent - rabbit): 0.42%/4H - Moderate
	Skin (Rodent - rabbit): 0.5mL/4H - Severe
	Skin (Rodent - rabbit): 100mg/24H - Severe
	Skin: adverse effect observed (irritating)

gamma-undecalactone

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg	Eye: no adverse effect observed (not irritating)
Oral (Rat) LD50: 18500 mg/kg	Skin (Rodent - guinea pig): 100mg/24H - Moderate
	Skin (Rodent - rabbit): 100mg/24H - Severe
	Skin: no adverse effect observed (not irritating)

7-acetyl-1,1,3,4,4,6-hexamethyltetraline

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg	Eye: no adverse effect observed (not irritating)
Oral (Rat) LD50: 570 mg/kg	Skin: no adverse effect observed (not irritating)

allyl cyclohexanepropionate

TOXICITY	IRRITATION
Oral (g.pig) LD50: 380 mg/kg	Eye: no adverse effect observed (not irritating)
Oral (Rat) LD50: 585 mg/kg	Skin (Human): 2/24H
	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)

10-undecenal

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg	Eye: no adverse effect observed (not irritating)

Continued...

	Oral (Rat) LD50: >5000 mg/kg	Skin (Rodent - rabbit): 500mg/24H - Mild
		Skin: adverse effect observed (irritating)

p-isopropyl-alpha-methylhydrocinnamaldehyde

TOXICITY	IRRITATION
dermal (rat) LD50: >5000 mg/kg	Eye (Rodent - rabbit): 100mg - Mild
Oral (Rat) LD50: 3810 mg/kg	Eye: no adverse effect observed (not irritating)
	Skin (Human): 15mg/48H - Mild
	Skin: adverse effect observed (irritating)

carvone

TOXICITY	IRRITATION
Oral (Guinea) LD50; 766 mg/kg	Eye: no adverse effect observed (not irritating)
	Skin (Rodent - rabbit): 500mg/24H - Mild
	Skin: no adverse effect observed (not irritating)

dodecyl aldehyde

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg	Eye: adverse effect observed (irritating)
Oral (Rat) LD50: 23000 mg/kg	Skin (Human): 5mg/48H - Mild
	Skin (Rodent - rabbit): 500mg/24H - Moderate
	Skin: adverse effect observed (irritating)

rose ketones

TOXICITY	IRRITATION
Oral (Mouse) LD50; 1821 mg/kg	Eye: no adverse effect observed (not irritating)
	Skin: adverse effect observed (irritating)
	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)

l-limonene

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg	Eye: no adverse effect observed (not irritating)
Oral (Rat) LD50: 4400 mg/kg	Skin (Rodent - rabbit): 500mg/24H - Moderate
	Skin: no adverse effect observed (not irritating)

beta-pinene

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

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	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

methyl eugenol

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg	Eye: no adverse effect observed (not irritating)
Inhalation (Rat) LC50: >1.2 mg/l4h	Skin (Rodent - rabbit): 500mg/24H
Oral (Rat) LD50: 1179 mg/kg	Skin: no adverse effect observed (not irritating)

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

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hydrogenation occurs on the end fatty acids,
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,

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	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>
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

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	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. 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
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.

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	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 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. 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.

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	IPCS Inchem: https://www.inchem.org/documents/jecfa/jecmono/v21je05.htm
2,6-DI-TERT-BUTYL-4-METHYLPHENOL	* Degussa SDS Effects such as behavioral changes, reduction in body weight gain, and decrement in body weight have been observed after long-term administration of BHT to mice and rats. Toxic effects may be attributed more to BHT metabolites than to their parent compound, only a few studies have focused on their carcinogenicity and toxicity, and not only on that of BHT. The metabolite BHT-QM (syn: 2,6-di-tert-butyl-1,4-methylene-2,5-cyclohexadien-1-one, CAS RN: 2607-52-5) is a very reactive compound which is considered to play a significant role in hepatoxicity, pneumotoxicity, and skin tumor promotion in mice. In addition, it was reported that another quinone derivative, BHT-OH(t)QM (syn 2-tert-butyl-6-(2-hydroxy-tert-butyl-4-methylene-2,5-cyclohexadien-1-one, CAS RN: 124755-19-7), is chemically more reactive than BHT-QM, and it has been recognized as the principal metabolite responsible for lung tumor promotion activity of BHT in mice. BHT has been reported to exert prooxidant effects under certain conditions. Thus, when BHT was added in excess to a wheat seedling medium in aerobic conditions, an enhancement of the generation rate of superoxide anion was observed. This is a reactive particle that may damage cellular structures at high concentrations In addition, an increase in hepatic microsomal lipid peroxidation was observed in rats fed with diets containing 0.2% of BHT for 30 days. Due to this ability of BHT to exert prooxidant effects at high concentrations, it has been used to induce experimental models of oxidative stress in several animals and fungi in order to study the protective effects of other compounds. Quinone methide derivatives form adducts with several proteins, including enzymes that protect cells from oxidative stress; this prooxidant state can also lead to cell oxidative damage. It must be noted that relationships between chronic oxidative stress and tumor promotion are well known Some authors have reported that at high aeration rate, BHT can react with molecular oxygen rather than with the reactive oxygen species present, yielding BHT-phenoxyl radical and superoxide anion. In addition, the phenolic radical itself may undergo redox recycling which can be a critical factor depending on the reductant involved However, it has to be noted that BHT-phenoxyl radical has been reported to be relatively stable. Furthermore, the potential reactivity of BHT-derived metabolites should be taken into account; some studies reported that not only BHT but also its metabolites, such as BHT-Q and BHT-QM, can act as prooxidant. As BHT undergoes several reactions during biotransformation, a large number of intermediate metabolites have been identified. However, their nature and concentration depend on the environmental conditions and on the animal species. Although the changes undergone by BHT during in vivo digestion processes have not been studied, after submission of a fluid deep-frying fat containing BHT and BHT-QM to an in vitro gastrointestinal digestion model, both these were detected in the digested samples. These results indicate that BHT and its toxic metabolite could remain bioaccessible for intestinal absorption. Studies concerning BHT metabolism have shown that, unlike other synthetic antioxidants, BHT is a potent inducer of the microsomal monooxygenase system and its major route of degradation is oxidation catalyzed by cytochrome P450. Studies have reported potential toxicity derived from the ingestion or administration of BHT. As for acute oral toxicity, although this is considered low in animals, it must be noted that 2 clinical cases were reported in patients who suffered acute neurotoxicity and gastritis after ingesting a high dose of BHT (4 and 80 g without medical prescription) to cure recurrent genital herpes. Regarding short-term subchronic toxicity studies, it has been reported that BHT causes dose-related increase in the incidence and severity of toxic nephrosis in mice, nephrotoxicity and pneumotoxicity in rats, and in chicken a marked congestion of the liver and kidney, as well as diffuse enlargement of the liver with rounded borders and rupture with hemorrhaging . It has to be noted that the EFSA Panel (2012) pointed out certain inconsistencies in the findings obtained from the short-term and subchronic toxicity studies. Several genotoxicity studies on BHT concluded that BHT does not represent a genotoxic risk, because most of the studies carried out to that date had shown BHT was not able to induce mutations or to damage deoxyribonucleic acid (DNA). Nevertheless, it must be mentioned that other studies reported contrary results. The effect of BHT and 7 of its metabolites on in vitro DNA cleavage was studied and the metabolites BHT-Q (syn: 2,6-di-tert-butyl-2,5-cyclohexadiene-1,4-dione, CAS RN: 719-22-2), BHT-CHO (syn: 3,5-di-tert-butyl-4-hydroxybenzaldehyde, CAS RN: 1620-98-0 and BHT-OOH (syn: 2,6-di-tert-butyl-4-methyl-4-hydroperoxy-2,5-cyclohexadien-1-one, CAS RN: 6485-57-0) were able to cleave DNA.. The Panel on Food Additives and Nutrient Sources Added to Food of the European Food Safety Authority (EFSA) recognized that these positive genotoxicity results may be due to the prooxidative chemistry of BHT, which gives rise to reactive metabolites. Some studies addressed the carcinogenicity and chronic toxicity of BHT and its metabolites in rodents with

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	contradictory results. Thus, mice-fed dietary BHT for a year developed marked hyperplasia of the hepatic bile ducts with an associated subacute cholangitis Moreover, after 104 wk of administration of BHT, the formation of hepatocellular tumors in male mice was observed. After 10 months of feeding mice with a diet containing different amounts of BHT, an increased incidence of liver tumors in male, but not female, animals was also reported . However, in a similar study no evidence of the carcinogenicity of BHT administered to mice was observed. Studies performed in rats also reported dose-related increases in hepatocellular adenomas and carcinomas; nevertheless, other studies carried out with rats showed no consistent carcinogenic effects. Several studies have demonstrated the potential of BHT to act either as a tumor promotor or as a tumor suppressor, modulating the carcinogenicity of some well-known carcinogens. Barbara Nieva-Echevarria etal: Comprehensive reviews in Food Science and Food Safety, Vol 14, Dec 2014 https://onlinelibrary.wiley.com/doi/10.1111/1541-4337.12121/pdf for bridged alkyl phenols: **Acute toxicity:** Acute oral and dermal toxicity data are available for all but two of the substances in the group. The data show that acute toxicity of these substances is low. The testing for acute toxicity spans five decades **Repeat dose toxicity:** Repeat dose studies on the members of this category include both subchronic and chronic exposures. The liver is identified as the target organ in rats for all of the substances tested. NOAEL s or NOEL s in rats for 13- week studies ranged from 100 ppm (approximately 5 mg/kg/day) to 500 ppm (approximately 25 mg/kg/day) while NOAEL s or NOEL s in rats for chronic studies were the same, 25 mg/kg/day (500 ppm). **Reproductive toxicity:** Evaluation of effects on reproduction for the bridged alkyl phenols is supplemented by histopathological data on male and female reproductive organs in repeated dose studies. The data on the effects of bridged alkyl phenols on reproduction and reproductive organs span the range of structures and molecular weights. While not all of the data for reproductive effects are from reproduction studies, microscopic evaluations of reproductive organs along with other short-term tests for reproductive effects provide adequate data to evaluate the effects of these bridged alkyl phenols on reproduction It can be concluded that reproductive toxicity is low. Typically a two-year chronic feeding study provides data for 4,4'-thiobis-6-(t-butyl-m-cresol) (96-69-5). No adverse effects were noted on reproductive organs **Genotoxicity:** Data from bacterial reverse mutation assays and in vitro and in vivo chromosome aberration studies were reviewed. Adequate bacterial gene mutation assays have been conducted with all of the category chemicals except two. Chromosome aberration studies, in vitro and/or in vivo, are available for all but two substances. The mutagenicity data span the range of structures and molecular weights and data can be bridged from other members of the group to meet any outstanding requirements. The weight of evidence for mutagenic potential for this category indicates these substances are not mutagenic. **Carcinogenicity:** The mutagenicity data combined with the animal data plus the long historical use of BHT (128-37-0) indicate that the chemicals in this class are not expected to exhibit any significant potential to cause cancer. The weight of the evidence indicates that these chemicals are not genotoxic. The Bridged Alkyl Phenols Category consists of a group of chemicals in which two molecules of mono or di-substituted alkyl (C1, C4, and/or C9) phenols are “bridged” or linked by a single atom (carbon or sulfur). The carbon atom linking the alkyl phenol groups contains hydrogen, propyl, or methyl substitutions. CAS No. 128-37-0 (BHT) is included in this category for data purposes because it is an alkyl phenol with a single carbon group such as the ones that link the phenol groups 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.
3-METHYLCYCLOPENTADECENONE (MIXED ISOMERS)	Skin sensitisation: Sensitization (Guinea pig Maximization)(OECD 406): 60% dermal responses Sensitization (HRIPT): Non sensitizing @ 10% Sensitization (HRIPT): Non sensitizing @ 20% Germ cell mutagenicity: Mutagenicity (Salmonella Reversion)(OECD 471): Non mutagenic Mutagenicity (Mammalian cell gene, in vitro)(OECD 476): Non mutagenic Genotoxicity (Human lymphocytes, in vitro)(OECD 473): No effects Reproductive Toxicity (1 generation, gav., rat) (OECD 415): NOEL 1000 mg/kg/d
OMEGA-PENTADECALACTONE	Skin (rabbit): 100% slight Irritation (dermal, pig)(photoxicity study): Non irritant @ 100% Irritation (dermal)(Human max): Non irritant @ 4% Irritation (dermal, human)(single patch test): Non

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	irritant @ 10% Sensitization (Human max.): Non sensitizing @ 10% Photo-toxicity (rabbit): Effects @ 10%, no effects @ 5% Photo-toxicity (guinea pig): Effects @ 50%, no effects @ 10% Photo-toxicity (guinea pig): No effects @ 20% Photo-toxicity (mouse): No effects @ 100% Photo-toxicity (pig): No effects @ 100% Mutagenicity (OECD 471): Non mutagenic (5 tests) Genotoxicity (Micronucleus test)(OECD 474): No effects * RIFM Monograph 502 Vigon MSDS Current opinion holds that there are no safety concerns for the Macrocyclic Lactone and Lactide (MLs, natural and synthetic musks) derivatives at reported levels of use and exposure as fragrance ingredients. · The MLs had low acute toxicity and no significant toxicity in repeat dose oral or dermal toxicity studies. Effects on blood biochemistry were reversible after 2 weeks of no treatment · Human dermatological studies show MLs are generally not irritating after one application. Minor irritation was observed in a few individuals following multiple applications.For high end users, calculated maximum dermal exposures vary from 0.47% to 11.15%; systemic exposures vary from 0.0008 to 0.25 mg/kg/day. . · In animal studies, the MLs are not sensitizers at lower exposures from consumer products. Eleven ML materials were evaluated for human sensitization. Of these, only ethylene brassylate showed evidence of sensitization in 2/27 studies (sensitization frequency 4/2059 total). · At rates consistent with reported levels for current human exposure, no phototoxicity or photosensitization was observed. · No mutagenic or genotoxic activity in bacteria and mammalian cell line assays was observed. The common structural element of the ML group of fragrance ingredients is a mono- or diester-lactone group, R–C (=O) O–R' , contained within a macrocyclic ring of C14 to C16 carbon chain length. . The naturally occurring macrocyclic lactones are generally derived from various plant, rather than animal, sources The macrocyclic lactone fragrance ingredients are generally lipophilic and log Kow increases with increasing ring size. log Kow values range from 6.7 for the mono C16 saturated lactone oxacycloheptadec-10-ene-2-one (CAS RN 28645-51-4) to 3.65 for the saturated C14 diester ethylene dodecanedioate (CAS RN 54982-83.-1). As a class, the macrocyclic lactone fragrance ingredients have a low volatility and are not appreciably water soluble. The initial and primary metabolism would be hydrolysis of the lactone functionality to generate the corresponding long chain open carboxylic acid and alcohol which should undergo fatty acid type beta-oxidation. It is believed that all the materials in this group have similar metabolism and are detoxified in the same manner. Their toxicological profiles would, then, be similar The Research Institute for Fragrance Materials (RIFM) Expert Panel
GALAXOLIDE	Changes in liver weight, maternal effects, foetotoxicity reported.
METHYL DIHYDROJASMONATE	Current opinion holds that there are no safety concerns for the cyclopentanones and cyclopentenones at reported levels of use and exposure as fragrance ingredients. The cyclopentanones and cyclopentenones have low levels of toxicity and no significant toxicity in repeat dose studies. Minimal evidence of skin irritation in humans is associated with current levels of use. Some of these substances irritate the eye; however, the risk of sensitization under current levels of use is generally small. No evidence of light-mediated toxicity or sensitization has been found. Developmental toxicity was not observed, and in testing using cells from bacteria and mammals, no mutation-causing activity or genetic toxicity was seen.
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.
CINNAMON OIL	Lowered blood pressure, acute pulmonary oedema, respiratory stimulation recorded.
EUGENOL	Equivocal tumorigen by RTECS criteria

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	For eugenol: The acute toxicity of eugenol is low. High doses may cause damage to the stomach lining, bleeding, inflammation of the stomach, liver discoloration and congestion in animals. Eugenol is readily absorbed through the skin; products containing eugenol or clove oil may irritate the skin and eyes. Inhalation may be a substantial route of exposure. Eugenol relaxes the blood vessels, causing low blood pressure and a slow heart rate. Animal testing shows that eugenol can cause genetic damage. It is believed that it does not cause cancer, and may even reduce the cancer-causing effect of certain other substances. Incidents of liver injury or failure among modern antifungal medicines are very low to non-existent. However, some can cause allergic reactions in people.[There are also many drug interactions. Patients must read in detail the enclosed data sheet(s) of any medicine. For example, the azole antifungals such as ketoconazole or itraconazole can be both substrates and inhibitors of the P-glycoprotein, which (among other functions) excretes toxins and drugs into the intestines.] Azole antifungals also are both substrates and inhibitors of the cytochrome P450 family CYP3A4,[] causing increased concentration when administering, for example, calcium channel blockers, immunosuppressants, chemotherapeutic drugs, benzodiazepines, tricyclic antidepressants, macrolides and SSRIs.[35] Before oral antifungal therapies are used to treat nail disease, a confirmation of the fungal infection should be made.[Approximately half of suspected cases of fungal infection in nails have a non-fungal cause.[The side effects of oral treatment are significant and people without an infection should not take these drugs.[Azoles are the group of antifungals which act on the cell membrane of fungi. They inhibit the enzyme 14-alpha-sterol demethylase, a microsomal CYP, which is required for biosynthesis of ergosterol for the cytoplasmic membrane. This leads to the accumulation of 14-alpha-methylsterols resulting in impairment of function of certain membrane-bound enzymes and disruption of close packing of acyl chains of phospholipids, thus inhibiting growth of the fungi. Some azoles directly increase permeability of the fungal cell membrane. vntifungal resistance is a subset of antimicrobial resistance, that specifically applies to fungi that have become resistant to antifungals. Resistance to antifungals can arise naturally, for example by genetic mutation or through aneuploidy. Extended use of antifungals leads to development of antifungal resistance through various mechanisms. Some fungi (e.g. Candida krusei and fluconazole) exhibit intrinsic resistance to certain antifungal drugs or classes, whereas some species develop antifungal resistance to external pressures. Antifungal resistance is a One Health concern, driven by multiple extrinsic factors, including extensive fungicidal use, overuse of clinical antifungals, environmental change and host factors.] Unlike resistance to antibacterials, antifungal resistance can be driven by antifungal use in agriculture. Currently there is no regulation on the use of similar antifungal classes in agriculture and the clinic. The emergence of Candida auris as a potential human pathogen that sometimes exhibits multi-class antifungal drug resistance is concerning and has been associated with several outbreaks globally. The WHO has released a priority fungal pathogen list, including pathogens with antifungal resistance
CINNAMON OIL, CEYLON	For cinnamon: While small amounts of cinnamon have been used occasionally as a spice for thousands of years, no reliable data are available on the daily long-term ingestion of amounts in the gram range on the basis of which the risk of high cinnamon exposure of this kind could be assessed. When it comes to individual ingredients the coumarin concentration in cassia cinnamon is particularly problematic. The values measured in cinnamon capsules (CVUA Stuttgart) confirm the high coumarin levels in cassia cinnamon (between approximately 2100 and approximately 4400 mg/kg cinnamon powder) as had also been previously measured by CVUA (Münster, BfR 2006). By contrast, coumarin can only be found in traces or below the measurement limit in Ceylon cinnamon.High coumarin levels in cinnamon and cinnamon-containing biscuits, or major consumption of cinnamon as a spice with a high coumarin level (e.g. frequent helpings of rice pudding with sugar and cinnamon by infants) can lead to the exceeding of the TDI of 0.1 mg/kg body weight established by the European Food Safety Authority (EFSA 2004). The consumption of capsules containing cassia cinnamon powder is also likely to lead to an exceeding of the above-mentioned TDI for coumarin. Besides coumarin cinnamon also contains other ingredients which could be problematic from a toxicological point of view when high levels of cinnamon are ingested daily. The main natural

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	ingredient is cinnamaldehyde which, in the worst case, may be contained in concentrations in the upper single-digit percentage range. What appears to be particularly problematic in this context is the consumption of high amounts of cinnamon by pregnant women. Other undesirable substances in cinnamon are safrole and styrene. Normally only traces of safrole are found in cinnamon. Higher concentrations are, however, found in oils from cinnamon leaves which may be used for blending purposes with other cinnamon oils. Styrene is formed in cinnamon under unfavourable transport and storage conditions. The concentrations of up to 22 mg/kg cinnamon measured by the Food Institute Braunschweig are safe in toxicological terms. However, in the past far higher levels have been determined. A comprehensive Monograph on cinnamon consumption (World Health Organisation WHO, 1999) lists as contraindications a high temperature of unknown origin and ulcerations of the stomach and duodenum. Furthermore, it points out that not enough data are available to assess the carcinogenic potential and that there are contradictory data about the mutagenic potential of cinnamon. When it comes to dosage a daily dose of 2 to 4 g is indicated in the WHO monogram. Besides the two above-mentioned sources there is an anecdotal information which refers to a comment which is not described in any more detail from the beginning of the 20th century (in Lewin (1992)): 'pregnant women who consume large amounts of cinnamon may, as I have observed, develop methaemoglobinuria, haematinuria, albuminuria and cylindruria. Urine of this kind does not decompose. A miscarriage may be triggered by the oil.' The dose recommendation of 2 to 4 g must be viewed as problematic as it is not supported by clinical or epidemiological data. Furthermore, it does not take into account problematic ingredients (first and foremost coumarin and cinnamaldehyde)
MENTHOL	Bacterial mutagenicity (Ames) test: negative * No evidence of carcinogenic, mutagenic or teratogenic effects After inhalation ; mucosal irritation After swallowing: gastric spasms, nausea, vomiting Systemic effects: dizziness, ataxia (impaired locomotor coordination), tiredness, depressed respiration. Risk of methaemoglobin formation. *Merck MSDS For kappa-opioid agonists: Kappa-opioid receptors are widely distributed in the brain, spinal cord and in pain neurons. Kappa-opioid receptor agonists produce unpleasant moods such as sadness, but their effects have been shown to vary between sexes. The receptors are thought to play a major role in mediating addiction and its remission, as well as the hallucinogenic side effects of opioids such as pentazocine. It is now widely accepted that kappa-opioid partial agonists block signals to the conscious mind from other parts of the brain and cause stupor and confusion. Although some of the agents are thought to have reduced potential for abuse due to their hallucinogenic side effects, some drugs in this group are abused even though the substance causes low mood. Kappa-opioid receptors have associated with a reduction in self-administration of alcohol and have been used to treat heroin dependence. Kappa-opioid receptor ligands cause a diuretic effect (increasing urine output), kappa-opioid agonists may also be protective to the nervous system where oxygen deficiency occurs, and this may be the target of new treatments.
D-LIMONENE	Tumorigenic by RTECS criteria
ETHYL-4-TRIMETHYLCYCLOPENTENYL-2-BUTENOL	Did not cause sensitisation on laboratory animals.Test substance 5% In a GLP acute oral toxicity study (limit test) performed according to OECD guideline 401, no mortality and no clinical signs related to treatment were observed, therefore LD50 was higher than 2000 mg/kg bw. In an older study, also performed according to OECD guideline 401, the test item tested at 5000 mg/kg bw led to the following clinical signs without death: pilo-erection, abnormal body carriage (hunched posture), abnormal gait (waddling), diarrhea and increased salivation. LD50 was higher than 5000 mg/kg bw. n a Magnusson & Kligman maximisation study performed according to OECD guideline 406 and in compliance with GLP, groups of 20 female guinea pigs were induced intradermally at10% w/v and then topically with undiluted material. When challenged at 75 % v/v, no positive response was observed in induced animals. In an acute dermal toxicity study (limit test), no mortality was observed in rats exposed to 5 mL/kg bw (corresponding to 4.6 g/kg bw). In an oral repeated dose toxicity range-finding experiment, male and female rats exposed for 14 days showed increased liver weight in both sexes and a slight increase in kidney weight in females. These effects show clear signs of oral bioavailability of the test material. In a 28-day repeated dose toxicity study conducted according to OECD guideline 422, biochemical changes in females at 1000 mg/kg bw/day, such as increased alanine aminophosphatase, alanine

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	aminotransferase, cholesterol and bilirubin levels also confirmed that the metabolic function of the liver was affected by administration of (2E)-ethyl-4-(2,2,3-trimethyl-3-cyclopenten-1-yl) but-2-en-1-ol. Also, in a 13-week repeated dose toxicity study conducted acording to OECD guideline 408, males and/or females exposed by diet to the substance showed slight non-adverse and reversible changes in haematological parameters (erythrocyte and haemoglobin concentrations, prothrombin time and haematocrit), biochemical parameters (urea, blood urea nitrogen and creatinine concentrations, glucose, alkaline phosphatase, albumin concentrations and albumin/globulin ratio), associated with increases in liver and/or kidney weights. These observations are thought to be indicative of adaptations of metabolism/excretion in the liver and kidneys, with absence of degenerative or functional change in liver or kidneys. Taken together, these observations clearly show absorption, systemic exposure and metabolism/excretion by liver and kidneys via oral route. As the test item is negative in two Ames tests, in a chromosome aberration test in CHO cells and in a gene mutation test (HPRT) in MLA L5178Y tk+/- cells, it is not classified according to Directive 67/548/EEC and CLP Regulation (EC) n° 1272/2008. rIn epeated dose toxicity studies (OECD guideline 422 and 408 studies), only slight adaptative and reversible systemic toxicity changes were observed in males and females rats exposed to the registered substance up to the highest dose tested. Furthermore, no effects on fertility, on or via lactation or on reproduction parameters were reported in the combined repeated dose and reproductive/developmental toxicity screening test and no effects on reproductive organs were detected in the 90-day repeated dose toxicity study. The only adverse effects were observed in gestating females at 750 mg/kg bw/day in a OECD guideline 414 study and at 1000 mg/kg bw/day in an OECD guideline 422 study, therefore the NOAEL for maternal toxicity was set at 300 mg/kg bw/day REACh Dossier
BERGAMOT OIL	tert
LAVANDIN OIL	Camphor appears to have moderate acute oral toxicity, and a higher toxicity when inhaled. Long term inhalation may cause emphysema. There is no observed tumour potential. Reproductive toxicity studies were not available for camphor, however, in developmental toxicity studies, it demonstrated no foetal toxicity.
VANILLIN	Miosis, somnolence, muscle weakness, coma, respiratory stimulation, maternal effects involving ovaries, fallopian tubes, uterus, cervix and vagina recorded.
COUMARIN	umarin is moderately toxic to the liver and kidneys of rodents, with a median lethal dose (LD50) of 293 mg/kg in the rat,[] a low toxicity compared to related compounds. Coumarin is hepatotoxic in rats, but less so in mice. Rodents metabolize it mostly to 3,4-coumarin epoxide, a toxic, unstable compound that on further differential metabolism may cause liver cancer in rats and lung tumors in mice] Humans metabolize it mainly to 7-hydroxycoumarin, a compound of lower toxicity, and no adverse affect has been directly measured in humans.[The German Federal Institute for Risk Assessment has established a tolerable daily intake (TDI) of 0.1 mg coumarin per kg body weight, but also advises that higher intake for a short time is not dangerous.[] The Occupational Safety and Health Administration (OSHA) of the United States does not classify coumarin as a carcinogen for humans European health agencies have warned against consuming high amounts of cassia bark, one of the four main species of cinnamon, because of its coumarin content] According to the German Federal Institute for Risk Assessment (BFR), 1 kg of (cassia) cinnamon powder contains about 2.1 to 4.4 g of coumarin.] Powdered cassia cinnamon weighs 0.56 g/cm3,[33] so a kilogram of cassia cinnamon powder equals 362.29 teaspoons. One teaspoon of cassia cinnamon powder therefore contains 5.8 to 12.1 mg of coumarin, which may be above the tolerable daily intake value for smaller individuals.[32] However, the BFR only cautions against high daily intake of foods containing coumarin. Its repor] specifically states that Ceylon cinnamon (Cinnamomum verum) contains 'hardly any' coumarin he European Regulation (EC) No 1334/2008 describes the following maximum limits for coumarin: 50 mg/kg in traditional and/or seasonal bakery ware containing a reference to cinnamon in the labeling, 20 mg/kg in breakfast cereals including muesli, 15 mg/kg in fine bakery ware, with the exception of traditional and/or seasonal bakery ware containing a reference to cinnamon in the labeling, and 5 mg/kg in desserts. An investigation from the Danish Veterinary and Food Administration in 2013 shows that bakery goods characterized as fine bakery ware exceeds the European limit (15 mg/kg) in almost 50% of the cases.[34] The paper also mentions tea as an additional important contributor to the overall coumarin intake, especially for children with a sweet habit. Coumarin was banned as a food additive in the United States in 1954, largely because of the hepatotoxicity results in rodent] Coumarin is currently listed by the Food and

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	Drug Administration (FDA) of the United States among 'Substances Generally Prohibited From Direct Addition or Use as Human Food,' according to 21 CFR 189.130,[36][37] but some natural additives containing coumarin, such as the flavorant sweet woodruff are allowed 'in alcoholic beverages only' under 21 CFR 172.510. In Europe, popular examples of such beverages are Maiwein, white wine with woodruff, and Zubrówka, vodka flavoured with bison grass. umarin is subject to restrictions on its use in perfumery,[39] as some people may become sensitized to it, however the evidence that coumarin can cause an allergic reaction in humans is disputed nor neurological dysfunction was found in children exposed to the anticoagulants acenocoumarol or phenprocoumon during pregnancy. A group of 306 children were tested at ages 7–15 years to determine subtle neurological effects from anticoagulant exposure. Results showed a dose–response relationship between anticoagulant exposure and minor neurological dysfunction. Overall, a 1.9 (90%) increase in minor neurological dysfunction was observed for children exposed to these anticoagulants, which are collectively referred to as 'coumarins.' In conclusion, researchers stated, 'The results suggest that coumarins have an influence on the development of the brain which can lead to mild neurologic dysfunctions in children of school age. Alcoholic beverages sold in the European Union are limited to a maximum of 10 mg/L coumarin by law. Cinnamon flavor is generally cassia bark steam-distilled to concentrate the cinnamaldehyde, for example, to about 93%. Clear cinnamon-flavored alcoholic beverages generally test negative for coumarin, but if whole cassia bark is used to make mulled wine, then coumarin shows up at significant levels
ISOCYCLEMONE E	Dermal (Rat) LD50: >5000 mg/kg(OECD 402)* Eye: non-irritant * (QSAR) * Sensitisation: Component: 68155-66-8 LLNA mouse: Result: Causes sensitization. Method: OECD 429 Repeated dose toxicity: Component: 68155-66-8 Oral rat Number of exposures: 1x /day NOEL: 150 mg/kg Method: OECD Test Guideline 407 Remarks: Repeated dose (28 days) toxicity (oral) Teratogenicity : Component: 68155-66-8 Application Route: Oral rat Number of exposures: 1x /day *IFF MSDS The substance is an individual isomer of the fragrance ingredient OTNE [predominant isomer: 1-(1,2,3,4,5,6,7,8-octahydro-2,3,8,8-tetramethyl-2-naphthyl)ethan-1- one; synonyms - tetramethylacetyloctahydronaphthalene, Iso-E Super; other isomers: 1-(1,2,3,4,5,6,7,8-octahydro 2,3,8,8,-tetramethyl-2-naphthyl)ethan-1-one, and 1,2,3,4,5,6,7,8-octahydro-2,3,8,8-tetramethyl-2-aceto naphthalenone]. A synthetic terpenoid considered to be a petroleum-derived aroma chemical No data were available regarding chemical disposition, metabolism, or toxicokinetics; acute, short term, subchronic, or chronic toxicity; synergistic or antagonistic activity; reproductive or teratological effects; carcinogenicity; genotoxicity; or immunotoxicity of OTNE Several compounds were considered as structural analogues of OTNE. Data are provided for the tetralin derivatives AHTN (CAS RN: 21145-77-7; Tonalide, 1-(5,6,7,8-tetrahydro-3,5,5,6,8,8 hexamethyl-2-naphthalenyl)ethanone) and AETT, (*CAS RN: 88-29-9; Versalide, 1-(3-ethyl-5,6,7,8-tetrahydro-5,5,8,8 tetramethyl-2-naphthalenyl)ethanone) which are also polycyclic synthetic musks. Both compounds have been detected in human adipose tissue and human milk. In one rat study, AHTN produced acute hepatic damage but in another had no adverse effects when administered to lactating rats beginning the third week of pregnancy at doses producing levels in the milk ~1000 times those reported in human milk. Administered by gavage at 50 mg/kg/day on gestation days 7 through 17, AHTN produced clinical signs and reduced weight gain and feed consumption in dams but had no adverse effect on embryo-fetal viability, growth, or morphology. In female rats, AETT induced classic degenerative changes in the liver and effects on the nucleolus and was neurotoxic. Effects included demyelination, hyperirritability, limb weakness, and gait abnormality that became severe ataxia. AHTN gave negative results in several genotoxicity studies (e.g., the Salmonella typhimurium/Escherichia coli plate incorporation and liquid preincubation assays and in vivo mouse micronucleus assays) Human Data is available ISO-E super (CAS RN: 54464-57-2): In dermatological patients, two cases of an allergic reaction towards Iso-E Super were observed on day 3 or 4 of application (patch test); however, this was not proved to be clinically relevant. Chronic exposure may result in permanent hypersensitivity] In a study with female mice, Iso E Super was positive in the local lymph node assay (LLNA) and irritancy assay (IRR), but negative in the mouse ear swelling test (MEST).
PHENETHYL ALCOHOL	Mutation mouse ascites tumour

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	Unlike benzylic alcohols, the beta-hydroxyl group of the members of benzyl alkyl alcohols contributes to break down reactions but do not undergo phase II metabolic activation. Though structurally similar to cancer causing ethyl benzene, phenethyl alcohol is only of negligible concern due to limited similarity in their pattern of activity. This is a member or analogue of a group of phenethyl, aldehyde, acid and related acetals generally regarded as safe (GRAS), intended for use as flavouring ingredients, 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.
BETA-IONONE	Not sensitising in guinea pig * Not mutagenic * Not photosensitising * * Givaudin
BENZYL SALICYLATE	The salicylates are well absorbed by mouth, and oral bioavailability is assumed to be total. In humans, absorption through skin is more limited. The salicylates are expected to be broken down to salicylic acid, mostly in the liver, and then conjugated with glycine or glucuronide and excreted in the urine. The expected metabolism of the salicylates do not present toxicological concerns. Animal testing shows that acute toxicity by skin contact is very low, while acute toxicity by mouth is moderate. Salicylates do not possess genetic toxicity, and generally do not have the potential to cause cancer. The reproductive and developmental toxicity data on methyl salicylate shows that high doses which are toxic to the mother may cause toxicity to the embryo and birth defects. At concentrations likely to be encountered through their use as fragrance ingredients, salicylates are considered to be non-irritating to the skin. The salicylates in general have no, or very limited, potential to sensitise skin. They do not possess light-mediated toxicity and do not cause light-mediated irritation or allergies.
GERANIOL	Geraniol does have sensitising properties, but the response it exhibits tends to be weak and variable. Animal testing revealed an oral semi-lethal dose of more than 3.6 g/kg in rats and an acute semi-lethal dose via skin absorption of over 5.0 g/kg.
GAMMA-UNDECALACTONE	Gamma-butyrolactone may cause thymus atrophy, brain damage, severe weakness and low body weight in rats. It causes no foetal development defects but may decrease testicular weight in the male rat. There is insufficient evidence from animal testing to show that gamma-butyrolactone has cancer-causing effects.
7-ACETYL-1,1,3,4,4,6-HEXAMETHYLTETRALINE	Liver changes, maternal effects recorded.
allyl cyclohexanepropionate	Somnolence, haemorrhage recorded.
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
P-ISOPROPYL-ALPHA-METHYLHYDROCINNAMALDEHYDE	Ataxia, coma, lachrymation, somnolence recorded.
CARVONE	- for (+/-)-carvone for (R)-(-)-carvone [RTECS No.: OS 8650000]
DODECYL ALDEHYDE	For n-alkyl aldehydes: Acute toxicity hazard of the n-alkyl aldehyde cluster members is moderate via inhalation and low via oral and dermal routes of exposure. Cluster members have been shown to be eye and skin irritants, but not skin sensitisers. Positive results for genotoxicity were reported for cluster members with lower molecular weights (<100), while members with molecular weights > 100 were negative, with the exception of nonanal (124-19-6). Although cancer bioassay data are not available for this cluster, several members of this cluster are considered potential carcinogens due to structural analogy to their carcinogenic lower homologs, acetaldehyde and formaldehyde . The primary metabolism of linear saturated aliphatic aldehydes and acids is a fundamental part of cell biochemistry. Aldehydes are successively oxidized to their corresponding carboxylic acids. To a minor extent, aldehydes also may be reduced to alcohols or conjugated with labile sulfhydryl-containing substances, such as glutathione In general, the inhalation route is expected to be of higher concern than the oral or dermal route because of rapid oxidation of the reactive aldehyde group to the relatively innocuous acid. However, individuals with genetic deficiency of aldehyde dehydrogenase may still be susceptible via the oral route.

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ROSE KETONES	These should not be used as fragrance ingredients at concentrations more than 0.02%, individually or in combination with other isomers of damascone. This is based on data showing potential for sensitisation and evidence of cross-reactivity.
BETA-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 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.
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
ISOEUGENOL	Somnolence, coma recorded. ADI: 0.2 mg/kg/day NOEL: 500 mg/kg/day For isoeugenol:

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	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
METHYL EUGENOL	For methyl eugenol: Past research has shown that the flavoring causes cancer of the liver, stomach, kidneys, and connective tissues in mice and rats. No study has proved a carcinogenic effect in people, but the biennial Report on Carcinogens released by the federal government's National Toxicology Program (NTP) in 2002, for the first time listed methyleugenol as 'reasonably anticipated to be a human carcinogen.' Also, 98 percent of 206 adults tested for methyleugenol while participating in the third National Health and Nutrition Examination Survey (NHANES III) from 1988 to 1994 had detectable concentrations of the chemical in their blood. Methyleugenol is reasonably anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity from studies in experimental animals, which indicates there is an increased incidence of malignant and/or combination of malignant and benign tumors at multiple tissue sites in multiple species of experimental animals. In animal studies, methyleugenol given orally to rats induced liver and stomach tumors in both sexes and kidney, mammary gland, and skin tumors in males. Methyleugenol given orally to mice induced benign and malignant tumors of the liver. Tumors of the stomach in male mice also were considered related to exposure to methyleugenol (NTP 1998). Earlier studies found that methyleugenol and two similar compounds, the structurally related allylbenzenes, safrole and estragole, induced liver tumors in mice after intraperitoneal injection (IARC 1976, Miller et al. 1983). Safrole is listed in the Report on Carcinogens as reasonably anticipated to be a human carcinogen and by IARC as possibly carcinogenic to humans (Group 2B). No adequate human studies of the relationship between exposure to methyleugenol and human cancer were found.

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WATER & OMEGA-PENTADECALACTONE & NUTMEG OIL, EXPRESSED & SPEARMINT OIL & CINNAMON OIL, CEYLON & ORANGE OIL & BERGAMOT OIL & LAVANDIN OIL	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.
SODIUM CITRATE & CITRIC ACID	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 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.
ETHYLENE GLYCOL PHENYL ETHER & MENTHOL	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
ETHYLENE GLYCOL PHENYL ETHER & CITRIC ACID & 2,6-DI-TERT-BUTYL-4-METHYLPHENOL & GALAXOLIDE & NUTMEG OIL, EXPRESSED & DIHYDROMYRCENOL & ETHYL VANILLIN & 4-TERT-BUTYLCYCLOHEXYL ACETATE & PHENETHYL ALCOHOL & MYRCENE & 10-UNDECENAL & P-ISOPROPYL-ALPHA-METHYLHYDROCINNAMALDEHYDE & DODECYL ALDEHYDE & L-LIMONENE & 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 & PHENETHYL ALCOHOL	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 & 2,6-DI-TERT-BUTYL-4-METHYLPHENOL & CINNAMON OIL & EUGENOL & CINNAMALDEHYDE & CINNAMON OIL, CEYLON & MENTHOL & BERGAMOT OIL & LAVANDIN OIL & COUMARIN & LINALYL ACETATE & ETHYL VANILLIN & 2-TERT-BUTYLCYCLOHEXYL ACETATE & 4-TERT-BUTYLCYCLOHEXYL ACETATE & PHENETHYL ALCOHOL & BENZYL SALICYLATE & GERANIOL & BETA-CITRONELLOL & GAMMA-UNDECALACTONE &	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.

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MYRCENE & DODECYL ALDEHYDE & BETA-PINENE & ALPHA-PINENE & ISOEUGENOL & METHYL EUGENOL	
ALPHA-TOCOPHEROL & 7-ACETYL-1,1,3,4,4,6-HEXAMETHYLTETRALINE	Based on laboratory and animal testing, exposure to the material may result in irreversible effects and mutations in humans.
2,6-DI-TERT-BUTYL-4-METHYLPHENOL & CINNAMON OIL & COUMARIN & 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.
2,6-DI-TERT-BUTYL-4-METHYLPHENOL & EUGENOL & D-LIMONENE & COUMARIN	The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
3-METHYLCYCLOPENTADECENONE (MIXED ISOMERS)	Sensitization (Guinea pig Maximization)(OECD 406): Non sensitizing
3-METHYLCYCLOPENTADECENONE (MIXED ISOMERS) & 8-CYCLOHEXADECEN-1-ONE	* Vigon SDS Current opinion holds that there are no safety concerns for macrocyclic ketones (MKs - naturally occuring or synthetic musks) at reported levels of use and exposure as fragrance ingredients · The MKs had low acute toxicity and no significant repeat dose toxicity. Liver weight and blood biochemistry effects were reversible after 2 weeks. · MKs are generally not irritating after one application. Animal studies showed irritation for some materials at concentrations higher than current consumer exposure. For high end users, calculated maximum dermal exposures vary from 0.13% to 1.10%; systemic exposures vary from 0.0005 to 0.0441 mg/kg/day. . · In animals, some MKs are sensitizers only at concentrations of 20%, 30%, or 100%, which are higher than current consumer exposure. No evidence of sensitization was observed in human tests. In patients with fragrance allergy, reactions were seen with cyclopentadecanone (3/178). · In humans, At rates consistent with current human exposure, phototoxicity and photosensitization were not observed. · Reproductive toxicity was not observed for 3-methylcyclopentadecenone in an OECD compliant study. · No genotoxicity in bacteria and mammalian cell lines was observed. The common structural element of the MK group of fragrance ingredients is a keto group, R–C (=O)-R', contained within a macrocyclic ring of C15 to C17 carbon chain length. The macrocyclic ketone fragrance ingredients are generally lipophilic and log Kow increases with increasing with ring size. log Kow values range from 6.31 for the C17, cycloheptadeca-9-en-1-one (CAS RN 542-46-1), to 5.33 for the C15,a4-cyclopentadecen-1-one (CAS RN 35720-57-1) macrocyclic ketone. It is proposed that the macrocyclic ketone may also be acted upon by reductases to generate a macrocyclic alcohol metabolite, which may also be either converted back to the macrocyclic ketone or conjugated with glucuronic acid and excreted. It is believed that all the materials in this group have similar metabolism and are detoxified in the same manner. Their toxicological profiles would, then, be similar. The Research Institute for Fragrance Materials (RIFM) Expert Panel
3-METHYLCYCLOPENTADECENONE (MIXED ISOMERS) & MENTHOL & BETA-IONONE & allyl cyclohexanepropionate & CARVONE & ROSE KETONES	A member or analogue of a group of aliphatic and alicyclic terpenoid tertiary alcohols and structurally related substances generally regarded as safe. Most alicyclic substances used as flavour ingredients are mono- and bicyclic terpenes which occur naturally in a wide variety of foods. With the exception of pulegone, alicyclic substances show very low oral acute toxicity. In most subchronic studies performed on animals, no adverse effects were observed at any dose level.
OMEGA-PENTADECALACTONE & GALAXOLIDE & METHYL DIHYDROJASMONATE & CINNAMON OIL & EUGENOL & CINNAMALDEHYDE & VANILLIN &	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),

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COUMARIN & ETHYL VANILLIN & ALPHA-HEXYLCINNAMALDEHYDE & PHENETHYL ALCOHOL & BENZYL SALICYLATE & BETA-CITRONELLOL & GAMMA-UNDECALACTONE & ROSE KETONES & EUGENYL ACETATE & ISOEUGENOL	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.
OMEGA-PENTADECALACTONE & GAMMA-UNDECALACTONE	This is a member or analogue of a group of lactones generally considered as safe (GRAS). Aliphatic lactones occur naturally at high concentrations (up to 100 parts per million) in food having a high fat content such as meat, cheese, milk and coconuts.
GALAXOLIDE & 7-ACETYL-1,1,3,4,4,6-HEXAMETHYLTETRALINE	There is increasing evidence emerging that some nitromusks and polycyclic musks, including those commonly used in perfumes, may be capable (either as parent compounds or as metabolites) of interfering with hormone communication systems in fish, amphibians and mammals, and may exacerbate the effects of exposure to other toxic chemicals.
CORIANDER OIL & LINALOOL & DIHYDROMYRCENOL & BERGAMOT OIL & LAVANDIN OIL & LINALYL ACETATE	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.
CORIANDER OIL & CINNAMON OIL & LINALOOL & EUGENOL & CINNAMALDEHYDE & BERGAMOT OIL & LAVANDIN OIL & LINALYL ACETATE & ALPHA-HEXYLCINNAMALDEHYDE & GERANIOL & GAMMA-UNDECALACTONE & 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.
CORIANDER OIL & LINALOOL & BERGAMOT OIL & LAVANDIN OIL & LINALYL ACETATE	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.
NUTMEG OIL, EXPRESSED & P-ISOPROPYL-ALPHA-METHYLHYDROCINNAMALDEHYDE	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
SPEARMINT OIL & LINALOOL & ORANGE OIL & BERGAMOT OIL & LAVANDIN OIL & GERANIOL & MYRCENE	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.
SPEARMINT OIL & CARVONE	Carvone substances have been reported to occur naturally in foods, including fruits, spices, and berries. Currently there are no safety concerns for any of the carvones based on current levels of intake. Following short term intake, increases in serum cholesterol and lipid concentrations have been reported in rats, as well as decreases in food consumption and body weight. Repeated intake may also cause shrinking of the testes. They appear to be protective against liver, lung, stomach and intestine tumours in rats.
CINNAMON OIL & D-LIMONENE & BERGAMOT OIL & LAVANDIN OIL & GERANIOL & BETA-CITRONELLOL & MYRCENE & ROSE KETONES & BETA-CARYOPHYLLENE	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.

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CINNAMON OIL & CINNAMALDEHYDE & CINNAMON OIL, CEYLON & ALPHA-HEXYLCINNAMALDEHYDE	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.
CINNAMON OIL & CINNAMALDEHYDE & ALPHA-HEXYLCINNAMALDEHYDE	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.
CINNAMON OIL & SAFROLE & METHYL EUGENOL	**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
CINNAMON OIL & CINNAMON OIL, CEYLON & SAFROLE	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 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

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	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.
LINALOOL & BERGAMOT OIL & LAVANDIN OIL & LINALYL ACETATE	Opinion holds that there are no safety concerns for linalool and the linalyl esters, as fragrance ingredients, under the present declared levels of use and exposure for the following reasons: · Linalool and the linalyl esters have a low order of acute toxicity. · No significant toxicity was observed in subchronic tests; it is concluded that these materials have dermal and oral NOAELS of 50 mg/kg/day or greater. · Based on a critical review of all available mutagenicity and genotoxicity studies,it has been determined that these materials are negative in short-term tests and therefore would have no significant potential to produce genotoxic effects. · The metabolic fate of linalool and the linalyl esters is either known or assumed from analogies with structurally related substances that indicate no production of toxic or persistent metabolites and the structural analogies indicate no concern. · Human dermatological studies show that these materials are not irritating, phototoxic or sensitizing. · These materials are used at low levels of exposure relative to doses that elicit adverse effects. The estimate for maximum systemic exposure by humans using cosmetic products is 0.3 mg/kg/ day for linalool and linalyl acetate and 0.1 mg/ kg/day or lower for the other linalyl esters. Using the NOAELs (50 mg/kg/day or greater) and the maximum exposure estimates and assuming 100% absorption,a margin of safety for the exposure of humans to linalool and the linalyl esters may conservatively be calculated as 167 times the maximum daily exposure for linalool and linalyl acetate (50 mg/kg/day 0.3 mg/kg/day for linalool or linalyl acetate=167) and 500 times the maximum daily exposure for the other individual linalyl esters (50 mg/kg/day / 0.1 mg/kg/day for the other individual linalyl esters=500). In general, linalool esters are hydrolyzed to their corresponding alcohol (linalool) and carboxylic acid. Hydrolysis is catalyzed by carboxylesterases or esterases . Tertiary alcohols such as linalool are metabolized primarily through conjugation with glucuronic acid and are excreted in the urine and to a lesser extent faeces. Alkyl or alkenyl substituents may undergo oxidation to form polar metabolites that may also be excreted free or in the conjugated form. Oxidation is mediated by cytochrome P-450 dependant mono-oxygenases. The carboxylic acids formed by hydrolysis of the linalyl esters included in this summary are all known to be easily and rapidly metabolized. The linear saturated carboxylic acids are metabolized normally as fatty acids that undergo beta-oxidation. The branched-chain carboxylic acids from linalyl isovalerate and isobutyrate are similarly oxidized,but the end product is acetone. The carboxylic acids from linalyl benzoate and phenylacetate are conjugated and excreted. The cinnamic acid from linalyl cinnamate is conjugated and excreted,or metabolized to benzoic acid. No sensitization was observed with linalool in guinea pig sensitization studies at concentrations up to 20%. With linalyl acetate at a concentration of 10%,weak to moderate sensitization effects were observed in guinea pig sensitization studies. Linalyl acetate was non-sensitizing when tested at 5% in these same guinea pig sensitization studies. No sensitization reactions were observed with linalyl isobutyrate and linalyl propionate (data were not available for the other linalyl esters) when tested at 8% in open epicutaneous tests in guinea pigs The Research Institute for Fragrance Materials (RIFM) Expert Panel A member or analogue of a group of aliphatic and alicyclic terpenoid tertiary alcohols and structurally related substances generally regarded as safe.

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	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.
LINALOOL & MENTHOL & LAVANDIN OIL & GERANIOL & BETA-CITRONELLOL	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
LINALOOL & DIHYDROMYRCENOL & LAVANDIN OIL & GERANIOL & BETA-CITRONELLOL	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.
LINALOOL & DIHYDROMYRCENOL & BERGAMOT OIL & LAVANDIN OIL & BETA-CITRONELLOL	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.
CINNAMON OIL, CEYLON & 7-ACETYL-1,1,3,4,4,6-HEXAMETHYLTETRALINE	**NOTE:** Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.
D-LIMONENE & BERGAMOT OIL & MYRCENE & BETA-CARYOPHYLLENE & 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.

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TERP is a reaction product of MMTC and dimethyltin dichloride (DMTC), Na2S, and tall oil fatty acid [a mixture of carboxylic acids, predominantly C-18]. The reaction product is a mixture of carboxylic esters and includes short oligomers of mono/dimethyltins bridged by sulfide groups. Although the tall oil component of TERP is not structurally similar to EHTG, TERP s conversion to MMTC justifies its inclusion. While the contribution of the various ligands to the overall toxicity may vary, the contribution is expected to be small relative to that of the MMTC. Further, the EHTG ligand from MMT(EHTG) is likely to be more toxic than the oleic or linoleic acid from TERP so inclusion of TERP in the category is a rather conservative approach. The other possible degradate of tall oil and EHTG is 2-mercaptoethanol (2-ME), and it is common to both ligands.
Data for MMT(EHTG) and MMT(IOTG) are used interchangeably because they are isomers differing only slightly in the structure of the C-8 alcohol of the mercaptoester ligand. In addition, the breakdown products of MMT(EHTG) and MMT(IOTG) are the thioglycolate esters (EHTG and IOTG), which have the common degradates, thioglycolic acid and C-8 alcohols (either 2-ethylhexanol or isooctanol). EHTG and IOTG also have similar physicochemical and toxicological properties.
The chemistry of the alkyl 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 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:

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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.
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).

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	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)
ORANGE OIL & BERGAMOT 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.
METHYLIONONE, ISOMERS & BETA-IONONE & ROSE KETONES	Beta-ionone is absorbed after oral exposure. Metabolism takes place mainly in the liver, and beta-ionone is excreted via urine. It produces abnormal liver, kidney and thyroid changes, and may cause depression and tremors. It causes dose dependent eye and skin irritation but no evidence of cancer-causing effect, nerve or genetic toxicity was observed. For ionones and rose ketones, when used as fragrance ingredients: Ionones have low to moderate toxicity if swallowed. Acute toxicity by skin contact is low. Animal testing has not shown subchronic toxicity. Under intended conditions of use as fragrance ingredients, they do not have significant potential for genetic, reproductive or developmental toxicity. Ionones are non-irritating when used as fragrance ingredients, while the rose ketones have limited irritation potential in sensitive subjects. The ionones are considered to be without significant potential to sensitise the skin, while the rose ketones are sensitisers when present at concentrations greater than 0.2%. The safety margin is considered to be high. A member or analogue of EFSA Chemical Group 10 secondary aliphatic saturated or unsaturated alcohols, ketones, ketals and esters with a secondary or tertiary oxygenated functional group used as flavourings

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No safety concern would arise for the consumer from the use of these compounds up to the highest proposed level in feeds.
Hazards for skin and eye contact and respiratory exposure are recognised for the majority of the compounds under application. Most are classified as irritating to the respiratory system.
Aliphatic acyclic and alicyclic alpha-diketones and alpha-hydroxyketones are generally used as flavouring agents up to average maximum levels of 200 ppm.
In rats and mice, orally administered aliphatic alpha-diketones are rapidly absorbed from the gastrointestinal tract. It is anticipated that at low levels of exposure, humans will metabolize aliphatic acyclic alpha-diketone principally by alpha-hydroxylation and subsequent oxidation of the terminal methyl group to yield the corresponding ketocarboxylic acid. The acid may undergo oxidative decarboxylation to yield carbon dioxide and a simple aliphatic carboxylic acid, which may be completely metabolized in the fatty acid pathway and citric acid cycle. At high concentrations, another detoxification pathway is used which involves reduction to the diol and subsequent conjugation with glucuronic acid. Acyclic alpha-diketones and alpha-hydroxyketones without a terminal methyl group and alicyclic diketones and hydroxyketones are mainly metabolized by reduction to the corresponding diol, followed by glucuronic acid conjugation and excretion
Compounds belonging to CG 10 are absorbed from the gastrointestinal tract and share common pathways of metabolism: (i) hydrolysis of esters by carboxylesterases, (ii) reduction of ketones to alcohols, (iii) oxidation of alcohols to acids, (iv) alpha–hydroxylation of the terminal methyl group to yield corresponding ketocarboxylic acids, (v) oxidative decarboxylation to yield carbon dioxide and an aliphatic carboxylic acid, and (vi) conjugation of alpha-hydroxyketones or their diol metabolites with glucuronic acid. Aliphatic acyclic diketones and alpha–hydroxyketones, which contain a carbonyl function at the 2-position (i.e. a methyl ketone) are expected to undergo alpha–hydroxylation and subsequent oxidation of the terminal methyl group to eventually yield corresponding ketocarboxylic acids. These compounds are intermediary metabolites (e.g.alpha-ketoacids), which may undergo oxidative decarboxylation to yield carbon dioxide and an aliphatic carboxylic acid. The acid is then metabolised via beta-oxidation and the citric acid cycle. beta-Ketoacids and derivatives readily undergo decarboxylation to yield breakdown products, which are incorporated into normal biochemical pathways. Alternatively, the methyl-substituted diketones may be successively reduced to the corresponding hydroxyketones and diols, which are excreted in the urine as glucuronic acid conjugates. This pathway is favoured at elevated in vivo concentrations, especially for longer chain length ketones. If the carbonyl function is located elsewhere on the chain, reduction is the predominant pathway. alpha-hydroxyketones or their diol metabolites may be excreted as glucuronic acid conjugates. Low concentrations of aliphatic acyclic methyl ketones are mainly metabolised by oxidation of the terminal methyl group. At higher concentrations, acyclic
alpha-diketones are metabolised via a reduction pathway to the diol and subsequent conjugation with glucuronic acid
In a 13-week study in rats (males/females, 15 animals/group), 3-hydroxybutan-2-one was administered with the diet at doses of 0, 85, 330 and 1,345 mg/kg bw per day. No treatment-related effects on body weight gain, haematological and urinary parameters, serum chemistry, organ weight and histopathology were seen up to 330 mg/kg bw per day. Several effects were observed at the highest dose tested, i.e. a reduction in body weight gain associated with a reduction in food and water
consumption, an increase in relative liver weight and a slight anaemia. From this study, a no observed adverse effect level (NOAEL) of 330 mg/kg bw per day could be derived.
A NOAEL of 90 mg/kg bw per day was derived from a 13-week study in rats (15 males/15 females each group), in which diacetyl [07.052] was administered by gavage at nominal doses of 0, 10, 30, 90 and 540 mg/kg bw per day. No adverse effects were seen at the three low doses tested on haematological and urinary parameters, serum chemistry, absolute and relative organ weight and histopathology. Several effects were observed at the highest dose tested (540 mg/kg bw), i.e. a decrease in weight gain associated with an increase in water consumption, anaemia, increased leucocyte count, increased relative weights of the liver, kidneys, adrenals and pituitary glands. At the same dose, stomach lesions seen at
necropsy revealed necrosis with in filtration by inflammatory cells.
A trial was conducted to assess the chronic toxicity of 3-ethylcyclopentan-1,2-dione ((due to keto-enol tautomerism this substance can exist as two isomers; the keto-isomer is 3-ethylcyclopentan-1,2-dione a synonym for the keto-isomer is ethylcyclopentenolone) on

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	reproduction and development in rats (male and female Charles River CD-COBS) following administration to three successive generations. In each generation, rats received diet containing 3-ethylcyclopentan-1,2-dione corresponding to dose levels of 0 (untreated controls), 0 (propylene glycol vehicle), 30, 80, and 200 mg/kg body weight/day. The F0 group (20 animals/sex/treatment) entered the study at weaning and were mated on day 64. Animals from the control groups and the high-dose group were maintained on trial for 12 months. The F1 generation 50 animals/sex per treatment except control, 100 animals/sex) was exposed to the test substance in utero, via milk until weaning and then through the diet for a further 23 months. The final examination of the F1 generation included ophthalmology, clinical chemistry, haematology and a full histopathology. The F1 generation was bred twice (days 99 and 155) and 20 litters/treatment group from the first mating selected to provide the F2 generation which were in turn mated at day 84. The F3 generation were killed after weaning. Survival, food consumption, growth, reproductive performance, haematological and clinical chemistry parameters were not adversely affected. Gross pathological and histopathological examination revealed no significant treatment-related effects. The incidence of benign or malignant tumours in treated animals was not significantly different to that in controls in the F0 and F1 generations. From this study, it is concluded that ethylcyclopentan-1,2-dione was not carcinogenic in rats under the study conditions and that a NOAEL of 200 mg/kg body weight (the highest dose tested) can be derived for chronic and developmental effects. A structural alert for genotoxicity is overruled for 3-ethyl-2-hydroxy-2-cyclopenten-1-one as well as for the nine structurally related substances (alpha,beta-unsaturated alicyclic ketones and their precursors) Maltol and ethyl maltol were considered separately because in contrast to the other substances in this subgroup they contain a ring-oxygen atom. Ethyl maltol induced gene mutations in bacteria Maltol induced gene mutations in bacteria and sister chromatid exchanges (SCE) in human lymphocytes In vivo, maltol induced micronuclei in mouse bone marrow after intraperitoneal application. Negative results were obtained in a sex-linked recessive lethal mutation assay in Drosophila. However, the micronucleus assay is considered more relevant than the Drosophila assay.Ethyl maltol induced gene mutations in bacteria EFSA Scientific Opinion October 2016: Safety and efficacy of secondary aliphatic saturated or unsaturated alcohols, ketones, ketals and esters with a second secondary or tertiary oxygenated functional group belonging to chemical group 10 when used as flavourings for all animal species Safety Evaluation of Aliphatic, Acyclic and Alicyclic alpha-Diketones and related Hydroxyketones; WHO Food Additive Series Joint FAO/ WHO Expert Committee on Food Additives 1999 The alpha,beta-unsaturated aldehyde and ketone structures are considered by the Panel to be structural alerts for genotoxicity. Flavouring Group Evaluation 213: alpha,beta-Unsaturated alicyclic ketones and precursors from chemical subgroup 2.7 of FGE.19: Scientific Opinion of the Panel on Food Contact Materials, Enzymes, Flavourings and Processing Aids (CEF)
METHYLIONONE, ISOMERS & ISOCYCLEMONE E	The alkyl cyclic ketone (ACK) fragrance ingredients are a diverse group of structures with similar metabolic and toxicity profiles. ACK fragrance materials have low acute toxicity. Repeated exposure causes some adverse effects in biochemical tests and blood cell counts. They are not considered to be irritating to the skin of humans. In animals, mild to moderate eye irritation was seen; however, full recovery usually occurred. Human studies showed that ACK fragrance ingredients have low potential for sensitization. Phototoxicity and photosensitization were not demonstrated in humans. Developmental toxicity occurred only when toxicity also appeared in the mother. Tests showed that this group of substances did not cause genetic toxicity.
VANILLIN & 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.
VANILLIN & ETHYL VANILLIN	For vanillin:

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	Vanillin generally does not cause irritation or sensitisation of the skin but sometimes does cause inflammation. It causes positive reactions to people already sensitised to Balsam of Peru, and is considered a secondary allergen. It is not considered to cause reproductive toxicity or toxic effects to the embryo. Vanillin does not cause birth defects. It may cause mutations according to some tests. There is no indication that vanillin causes cancer. Tests show that vanillin is not toxic to the immune system, but are conflicting in that one test suggests that it stimulates while another suggests it suppresses the immune system.
VANILLIN & ETHYL VANILLIN & 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)
2-TERT-BUTYLCYCLOHEXYL ACETATE & 4-TERT-BUTYLCYCLOHEXYL ACETATE	There are no safety concerns regarding cyclic acetates under the present declared levels of use, for the reasons outlined below. Cyclic acetates have low acute toxicity. Cyclic acetates and cyclic alcohols also have low whole-body toxicity, after repeated application to skin. At concentrations encountered in current use, minimal, if any, skin irritation occurs. These substances have little or no sensitizing potential. Available data does not indicate that these substances cause genetic toxicity or mutations, so they are unlikely to cause cancer. They have a very wide safety margin.
GERANIOL & BETA-CITRONELLOL	Citronellol, geraniol, nerol, and geranyl acetate are currently generally regarded as safe by the US FDA for their intended use as flavouring substances. They are ubiquitous in the plant kingdom. Terpenoid alcohol, formed in the gastrointestinal tract, as a result of hydrolysis, is rapidly absorbed, metabolised and excreted via the urine. It has no repeat dose effect, no genetic and cancer causing effect but may harm the unborn child of a pregnant woman.
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.
SAFROLE & METHYL EUGENOL	Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [*National Toxicology Program: U.S. Dep. of Health & Human Services 2002*]

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

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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 (Spiced Chestnut)

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
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

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>50mg/l	2

Continued...

	EC50(ECx)	48h	Crustacea	>50mg/l	2
	EC50	72h	Algae or other aquatic plants	990mg/l	2
	LC50	96h	Fish	>100mg/l	2

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

2,6-di-tert-butyl-4-methylphenol

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1344h	Fish	220-2800	7
EC50	48h	Crustacea	>0.17mg/l	2
EC50	72h	Algae or other aquatic plants	>0.42mg/l	1
ErC50	72h	Algae or other aquatic plants	>0.42mg/l	1
EC50	96h	Algae or other aquatic plants	0.758mg/l	2
EC0(ECx)	48h	Crustacea	>=0.31mg/l	1
LC50	96h	Fish	0.199mg/l	2

3-methylcyclopentadecenone (mixed isomers)

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	0.39mg/l	Not Available
EC50	72h	Algae or other aquatic plants	>30mg/l	Not Available
NOEC(ECx)	96h	Fish	0.001mg/l	Not Available
LC50	96h	Fish	0.22mg/l	Not Available

8-cyclohexadecen-1-one

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	0.275mg/L	2
EC50	72h	Algae or other aquatic plants	>1.35mg/l	Not Available
EC50(ECx)	72h	Algae or other aquatic plants	>1.35mg/l	Not Available
LC50	96h	Fish	0.75mg/l	Not Available

omega-pentadecalactone

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>0.17mg/l	2
EC50	72h	Algae or other aquatic plants	0.4mg/l	2
NOEC(ECx)	792h	Fish	0.027mg/l	2
LC50	96h	Fish	>0.797mg/l	2

galaxolide

Continued...

NOEC(ECx)	3h	Crustacea	<=0.001mg/L	4
LC50	96h	Fish	0.464-0.512mg/L	4

methyl dihydrojasmonate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	8.25mg/l	2
EC50	72h	Algae or other aquatic plants	18.2mg/l	2
EC50(ECx)	504h	Crustacea	0.732mg/l	2
LC50	96h	Fish	19mg/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

coriander oil

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

nutmeg oil, expressed

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

spearmint oil

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

cinnamon oil

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

linalool

Endpoint	Test Duration (hr)	Species	Value	Source
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

eugenol

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

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

Continued...

cinnamon oil, Ceylon

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	1.61mg/l	2
EC50	96h	Algae or other aquatic plants	1.68mg/l	2
EC50(ECx)	48h	Crustacea	1.61mg/l	2

menthol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	26.6mg/l	2
EC50	72h	Algae or other aquatic plants	0.33mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	0.089mg/l	2
LC50	96h	Fish	18.9mg/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

orange oil

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

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

ethyl-4-trimethylcyclopentenyl-2-butenol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	1.34mg/l	Not Available
EC50	96h	Algae or other aquatic plants	2.5mg/l	Not Available
EC50(ECx)	48h	Crustacea	1.34mg/l	Not Available
LC50	96h	Fish	1.1mg/l	Not Available

methylionone, isomers

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	9mg/l	2
EC50	72h	Algae or other aquatic plants	>20mg/l	2
NOEC(ECx)	48h	Crustacea	1mg/l	2
LC50	96h	Fish	6.8mg/l	2
EC50	48h	Crustacea	1.3mg/l	2
EC50	72h	Algae or other aquatic plants	1.1mg/l	2
EC50(ECx)	72h	Algae or other aquatic plants	1.1mg/l	2

bergamot oil

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

lavandin oil

Endpoint	Test Duration (hr)	Species	Value	Source

Continued...

EC50	48h	Crustacea	0.41mg/l	2
EC50	72h	Algae or other aquatic plants	0.5mg/l	2
EC50(ECx)	48h	Crustacea	0.41mg/l	2
LC50	96h	Fish	0.29mg/l	2

vanillin

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>10<100mg/l	2
EC50	72h	Algae or other aquatic plants	120mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	>2mg/l	1
LC50	96h	Fish	53-61.3mg/L	4

coumarin

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

linalyl acetate

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

ethyl vanillin

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	26.2mg/l	2
EC50	72h	Algae or other aquatic plants	>100mg/l	2
NOEC(ECx)	504h	Crustacea	5.9mg/l	2
LC50	96h	Fish	81.4-94.3mg/L	4

isocyclemone E

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	1.3mg/l	2
EC50	48h	Crustacea	1.38mg/l	2
EC50	72h	Algae or other aquatic plants	>2.6mg/l	2
NOEC(ECx)	504h	Crustacea	0.028mg/l	2

2-tert-butylcyclohexyl acetate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	17mg/l	2
EC50	72h	Algae or other aquatic plants	4.2mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	0.57mg/l	2
LC50	96h	Fish	5.6mg/l	2

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

Continued...

NOEC(ECx)	504h	Crustacea	0.063mg/L	2
LC50	96h	Fish	~1.7mg/l	2

4-tert-butylcyclohexyl acetate

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

Ionone, methyl-

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

phenethyl alcohol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	287.17mg/l	1
EC50	72h	Algae or other aquatic plants	490mg/l	1
LC50	96h	Fish	>215<464mg/l	2
NOEC(ECx)	96h	Fish	100mg/l	1

beta-ionone

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	1.641mg/l	2
EC50	72h	Algae or other aquatic plants	3.223mg/l	2
EC50	96h	Algae or other aquatic plants	2.92mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	0.656mg/l	2
LC50	96h	Fish	1.462mg/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

geraniol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	10.8mg/l	2
EC50	72h	Algae or other aquatic plants	13.1mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	1mg/l	2
LC50	96h	Fish	2.3-3mg/l	4

beta-citronellol

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

gamma-undecalactone

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	4mg/l	2
EC50	72h	Algae or other aquatic plants	7.218mg/l	2

Continued...

	NOEC(ECx)	504h	Crustacea	0.138mg/l	2
	EC50	96h	Algae or other aquatic plants	5mg/l	2
	LC50	96h	Fish	5.5mg/l	2

7-acetyl-1,1,3,4,4,6-hexamethyltetraline

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.438-0.508mg/l	4
NOEC(ECx)	3h	Crustacea	0.004-0.009mg/L	4
LC50	96h	Fish	0.95-1.05mg/l	4

allyl cyclohexanepropionate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	3.8mg/l	2
EC50	72h	Algae or other aquatic plants	2.1mg/l	2
EC50	96h	Algae or other aquatic plants	2.3mg/l	2
EC0(ECx)	96h	Fish	0.058mg/l	2
LC50	96h	Fish	0.13mg/l	2

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

10-undecenal

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

p-isopropyl-alpha-methylhydrocinnamaldehyde

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	1.4mg/l	2
EC50	72h	Algae or other aquatic plants	2.7mg/l	2
NOEC(ECx)	96h	Algae or other aquatic plants	0.2mg/l	2
EC50	96h	Algae or other aquatic plants	2.7mg/l	2
LC50	96h	Fish	1.42mg/l	2

carvone

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

Continued...

dodecyl aldehyde

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

rose ketones

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

l-limonene

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	0.702mg/L	2
EC50	48h	Crustacea	0.36mg/l	2
EC50	72h	Algae or other aquatic plants	>1.6mg/l	2
EC0(ECx)	48h	Crustacea	0.074mg/l	2
EC50	96h	Algae or other aquatic plants	0.904mg/l	2

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

Continued...

	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

methyl eugenol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	10.842mg/l	2
EC50	72h	Algae or other aquatic plants	9.6mg/l	2
EC50	96h	Algae or other aquatic plants	8.3mg/l	2
NOEC(ECx)	504h	Crustacea	1.06mg/l	2
LC50	96h	Fish	4.9-7.2mg/L	4

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
water	LOW	LOW
butyl alcohol propoxylated	LOW	LOW
ethylene glycol phenyl ether	LOW	LOW
citric acid	LOW	LOW
alpha-tocopherol	HIGH	HIGH
2,6-di-tert-butyl-4-methylphenol	HIGH	HIGH
omega-pentadecalactone	LOW	LOW
galaxolide	HIGH	HIGH
methyl dihydrojasmonate	LOW	LOW
dipropylene glycol	LOW	LOW
linalool	HIGH	HIGH
eugenol	HIGH	HIGH
cinnamaldehyde	LOW	LOW
menthol	HIGH	HIGH
d-limonene	HIGH	HIGH

Continued...

dihydromyrcenol	HIGH	HIGH
ethyl-4-trimethylcyclopentenyl-2-butenol	HIGH	HIGH
methylionone, isomers	HIGH	HIGH
vanillin	LOW	LOW
coumarin	LOW	LOW
linalyl acetate	HIGH	HIGH
ethyl vanillin	LOW	LOW
2-tert-butylcyclohexyl acetate	HIGH	HIGH
alpha-hexylcinnamaldehyde	LOW	LOW
4-tert-butylcyclohexyl acetate	HIGH	HIGH
Ionone, methyl-	HIGH	HIGH
phenethyl alcohol	LOW	LOW
beta-ionone	HIGH	HIGH
benzyl salicylate	HIGH	HIGH
geraniol	LOW	LOW
beta-citronellol	LOW	LOW
gamma-undecalactone	LOW	LOW
7-acetyl-1,1,3,4,4,6-hexamethyltetraline	HIGH	HIGH
allyl cyclohexanepropionate	LOW	LOW
myrcene	HIGH	HIGH
10-undecenal	LOW	LOW
carvone	HIGH	HIGH
dodecyl aldehyde	LOW	LOW
rose ketones	HIGH	HIGH
beta-caryophyllene	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
methyl eugenol	HIGH	HIGH

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	LOW (LogKOW = -1.64)
alpha-tocopherol	LOW (LogKOW = 12.18)

Continued...

2,6-di-tert-butyl-4-methylphenol	HIGH (BCF = 2500)
3-methylcyclopentadecenone (mixed isomers)	HIGH (LogKOW = 5.75)
omega-pentadecalactone	HIGH (LogKOW = 6.15)
galaxolide	HIGH (LogKOW = 5.9)
methyl dihydrojasmonate	LOW (LogKOW = 2.975)
dipropylene glycol	LOW (BCF = 4.6)
coriander oil	LOW (LogKOW = 3.33)
nutmeg oil, expressed	LOW (LogKOW = 18.04)
spearmint oil	LOW (LogKOW = 2.71)
cinnamon oil	LOW (LogKOW = 1.9)
linalool	LOW (LogKOW = 2.97)
eugenol	LOW (LogKOW = 2.27)
cinnamaldehyde	LOW (BCF = 10)
cinnamon oil, Ceylon	LOW (LogKOW = 1.9)
menthol	LOW (BCF = 15)
d-limonene	HIGH (LogKOW = 4.8275)
orange oil	MEDIUM (LogKOW = 4.38)
dihydromyrcenol	LOW (LogKOW = 3.47)
ethyl-4-trimethylcyclopentenyl-2-butenol	HIGH (LogKOW = 5.1374)
methylionone, isomers	LOW (LogKOW = 8.56)
bergamot oil	MEDIUM (LogKOW = 3.93)
lavandin oil	LOW (LogKOW = 2.97)
vanillin	LOW (LogKOW = 1.21)
coumarin	LOW (LogKOW = 1.39)
linalyl acetate	MEDIUM (LogKOW = 3.93)
ethyl vanillin	LOW (LogKOW = 1.58)
isocyclemone E	HIGH (LogKOW = 5.18)
2-tert-butylcyclohexyl acetate	MEDIUM (LogKOW = 4.42)
alpha-hexylcinnamaldehyde	HIGH (LogKOW = 4.82)
4-tert-butylcyclohexyl acetate	HIGH (LogKOW = 4.8)
Ionone, methyl-	HIGH (LogKOW = 4.84)
phenethyl alcohol	LOW (LogKOW = 1.36)
beta-ionone	MEDIUM (LogKOW = 3.84)
benzyl salicylate	MEDIUM (LogKOW = 4.31)
geraniol	LOW (LogKOW = 3.47)
beta-citronellol	MEDIUM (LogKOW = 3.91)
gamma-undecalactone	LOW (LogKOW = 3.06)
7-acetyl-1,1,3,4,4,6-hexamethyltetraline	HIGH (LogKOW = 5.7)
allyl cyclohexanepropionate	MEDIUM (LogKOW = 4.4707)
myrcene	MEDIUM (LogKOW = 4.17)

Continued...

10-undecenal	MEDIUM (LogKOW = 4.12)
p-isopropyl-alpha-methylhydrocinnamaldehyde	MEDIUM (LogKOW = 3.91)
carvone	LOW (LogKOW = 2.71)
dodecyl aldehyde	HIGH (LogKOW = 4.75)
rose ketones	MEDIUM (LogKOW = 4.4235)
beta-caryophyllene	HIGH (LogKOW = 6.3)
l-limonene	MEDIUM (LogKOW = 4.38)
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)
methyl eugenol	LOW (LogKOW = 3.03)

Mobility in soil

Ingredient	Mobility
butyl alcohol propoxylated	LOW (Log KOC = 10)
ethylene glycol phenyl ether	LOW (Log KOC = 12.12)
citric acid	LOW (Log KOC = 10)
alpha-tocopherol	LOW (Log KOC = 51280000)
2,6-di-tert-butyl-4-methylphenol	LOW (Log KOC = 23030)
omega-pentadecalactone	LOW (Log KOC = 5994)
galaxolide	LOW (Log KOC = 10380)
methyl dihydrojasmonate	LOW (Log KOC = 142.3)
dipropylene glycol	HIGH (Log KOC = 1)
linalool	LOW (Log KOC = 56.32)
eugenol	LOW (Log KOC = 1124)
cinnamaldehyde	LOW (Log KOC = 102.4)
menthol	LOW (Log KOC = 66.19)
d-limonene	LOW (Log KOC = 1324)
dihydromyrcenol	LOW (Log KOC = 54.78)
ethyl-4-trimethylcyclopentenyl-2-butenol	LOW (Log KOC = 685.5)
methylionone, isomers	LOW (Log KOC = 1034)
vanillin	LOW (Log KOC = 38.45)
coumarin	LOW (Log KOC = 146.1)
linalyl acetate	LOW (Log KOC = 517.9)
ethyl vanillin	LOW (Log KOC = 70.92)
2-tert-butylcyclohexyl acetate	LOW (Log KOC = 528.1)
alpha-hexylcinnamaldehyde	LOW (Log KOC = 4025)

Continued...

4-tert-butylcyclohexyl acetate	LOW (Log KOC = 517.4)
Ionone, methyl-	LOW (Log KOC = 1208)
phenethyl alcohol	LOW (Log KOC = 28.89)
beta-ionone	LOW (Log KOC = 625.2)
benzyl salicylate	LOW (Log KOC = 5156)
geraniol	LOW (Log KOC = 70.79)
beta-citronellol	LOW (Log KOC = 70.79)
gamma-undecalactone	LOW (Log KOC = 476.5)
7-acetyl-1,1,3,4,4,6-hexamethyltetraline	LOW (Log KOC = 8564)
allyl cyclohexanepropionate	LOW (Log KOC = 878.9)
myrcene	LOW (Log KOC = 1269)
10-undecenal	LOW (Log KOC = 370)
carvone	LOW (Log KOC = 123.7)
dodecyl aldehyde	LOW (Log KOC = 682.4)
rose ketones	LOW (Log KOC = 668.6)
beta-caryophyllene	LOW (Log KOC = 22290)
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)
methyl eugenol	LOW (Log KOC = 494)

Other adverse effects

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

Continued...

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

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

Continued...

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	Not Available
ethylhexylglycerin	Not Available
alpha-tocopherol	Not Available
2,6-di-tert-butyl-4-methylphenol	Not Available
3-methylcyclopentadecenone (mixed isomers)	Not Available
8-cyclohexadecen-1-one	Not Available
omega-pentadecalactone	Not Available
galaxolide	Not Available
methyl dihydrojasmonate	Not Available
dipropylene glycol	Not Available
coriander oil	Not Available
nutmeg oil, expressed	Not Available
spearmint oil	Not Available
cinnamon oil	Not Available
linalool	Not Available
eugenol	Not Available
cinnamaldehyde	Not Available
cinnamon oil, Ceylon	Not Available
menthol	Not Available
d-limonene	Not Available
orange oil	Not Available
dihydromyrcenol	Not Available
ethyl-4-trimethylcyclopentenyl-2-butenol	Not Available
methylionone, isomers	Not Available
bergamot oil	Not Available
lavandin oil	Not Available
vanillin	Not Available
coumarin	Not Available
linalyl acetate	Not Available
ethyl vanillin	Not Available
isocyclemone E	Not Available
2-tert-butylcyclohexyl acetate	Not Available
alpha-hexylcinnamaldehyde	Not Available

Continued...

4-tert-butylcyclohexyl acetate	Not Available
Ionone, methyl-	Not Available
phenethyl alcohol	Not Available
beta-ionone	Not Available
benzyl salicylate	Not Available
geraniol	Not Available
beta-citronellol	Not Available
gamma-undecalactone	Not Available
7-acetyl-1,1,3,4,4,6-hexamethyltetraline	Not Available
allyl cyclohexanepropionate	Not Available
myrcene	Not Available
10-undecenal	Not Available
p-isopropyl-alpha-methylhydrocinnamaldehyde	Not Available
carvone	Not Available
dodecyl aldehyde	Not Available
rose ketones	Not Available
beta-caryophyllene	Not Available
l-limonene	Not Available
beta-pinene	Not Available
eugenyl acetate	Not Available
safrole	Not Available
alpha-pinene	Not Available
isoeugenol	Not Available
methyl eugenol	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	Not Available
ethylhexylglycerin	Not Available
alpha-tocopherol	Not Available
2,6-di-tert-butyl-4-methylphenol	Not Available
3-methylcyclopentadecenone (mixed isomers)	Not Available
8-cyclohexadecen-1-one	Not Available
omega-pentadecalactone	Not Available
galaxolide	Not Available
methyl dihydrojasmonate	Not Available

Continued...

dipropylene glycol	Not Available
coriander oil	Not Available
nutmeg oil, expressed	Not Available
spearmint oil	Not Available
cinnamon oil	Not Available
linalool	Not Available
eugenol	Not Available
cinnamaldehyde	Not Available
cinnamon oil, Ceylon	Not Available
menthol	Not Available
d-limonene	Not Available
orange oil	Not Available
dihydromyrcenol	Not Available
ethyl-4-trimethylcyclopentenyl-2-butenol	Not Available
methylionone, isomers	Not Available
bergamot oil	Not Available
lavandin oil	Not Available
vanillin	Not Available
coumarin	Not Available
linalyl acetate	Not Available
ethyl vanillin	Not Available
isocyclemone E	Not Available
2-tert-butylcyclohexyl acetate	Not Available
alpha-hexylcinnamaldehyde	Not Available
4-tert-butylcyclohexyl acetate	Not Available
Ionone, methyl-	Not Available
phenethyl alcohol	Not Available
beta-ionone	Not Available
benzyl salicylate	Not Available
geraniol	Not Available
beta-citronellol	Not Available
gamma-undecalactone	Not Available
7-acetyl-1,1,3,4,4,6-hexamethyltetraline	Not Available
allyl cyclohexanepropionate	Not Available
myrcene	Not Available
10-undecenal	Not Available
p-isopropyl-alpha-methylhydrocinnamaldehyde	Not Available
carvone	Not Available
dodecyl aldehyde	Not Available
rose ketones	Not Available
beta-caryophyllene	Not Available

Continued...

l-limonene	Not Available
beta-pinene	Not Available
eugenyl acetate	Not Available
safrole	Not Available
alpha-pinene	Not Available
isoeugenol	Not Available
methyl eugenol	Not Available

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
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

Continued...

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 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

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
US - Alaska Air Quality Control - Concentrations Triggering an Air Quality Episode for Air Pollutants Other Than PM-2.5
US - Massachusetts - Right To Know Listed Chemicals
US - New Jersey Right to Know Hazardous Substances
US - Pennsylvania - Hazardous Substance List
US New York City Community Right-to-Know: List of Hazardous Substances
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Limits (PELs) Table Z-1
US OSHA Permissible Exposure Limits (PELs) Table Z-3
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

3-methylcyclopentadecenone (mixed isomers) is found on the following regulatory lists

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

8-cyclohexadecen-1-one is found on the following regulatory lists

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

omega-pentadecalactone is found on the following regulatory lists

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

galaxolide is found on the following regulatory lists

US EPCRA Section 313 Chemical List
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

methyl dihydrojasmonate 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

coriander oil is found on the following regulatory lists

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

nutmeg oil, expressed is found on the following regulatory lists

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

spearmint oil is found on the following regulatory lists

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

cinnamon oil is found on the following regulatory lists

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

Continued...

linalool 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

cinnamaldehyde is found on the following regulatory lists

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

cinnamon oil, Ceylon is found on the following regulatory lists

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

menthol is found on the following regulatory lists

US AIHA Workplace Environmental Exposure Levels (WEELs)

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

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

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)

orange oil 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

ethyl-4-trimethylcyclopentenyl-2-butenol is found on the following regulatory lists

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

methylionone, isomers is found on the following regulatory lists

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

bergamot oil is found on the following regulatory lists

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

lavandin oil is found on the following regulatory lists

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

vanillin is found on the following regulatory lists

US AIHA Workplace Environmental Exposure Levels (WEELs)

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

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

coumarin is found on the following regulatory lists

FEI Equine Prohibited Substances List - Banned Substances

FEI Equine Prohibited Substances List (EPSL)

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

US Clean Air Act - Hazardous Air Pollutants

US DOE Temporary Emergency Exposure Limits (TEELs)

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

Continued...

linalyl acetate is found on the following regulatory lists

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

ethyl vanillin is found on the following regulatory lists

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

isocyclemone E is found on the following regulatory lists

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

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

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

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

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

Ionone, methyl- is found on the following regulatory lists

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

phenethyl alcohol is found on the following regulatory lists

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

beta-ionone 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

geraniol is found on the following regulatory lists

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

beta-citronellol is found on the following regulatory lists

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

gamma-undecalactone is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List

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

7-acetyl-1,1,3,4,4,6-hexamethyltetraline is found on the following regulatory lists

US Clean Air Act - Hazardous Air Pollutants

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

allyl cyclohexanepropionate is found on the following regulatory lists

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

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

10-undecenal is found on the following regulatory lists

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

p-isopropyl-alpha-methylhydrocinnamaldehyde is found on the following regulatory lists

Continued...

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

carvone is found on the following regulatory lists

US - Massachusetts - Right To Know Listed Chemicals

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

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

dodecyl aldehyde is found on the following regulatory lists

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

rose ketones is found on the following regulatory lists

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

l-limonene is found on the following regulatory lists

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

beta-pinene is found on the following regulatory lists

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

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

Continued...

humans

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

methyl eugenol 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 2A: Probably carcinogenic to humans

US - California Proposition 65 - Carcinogens

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

US - New Jersey Right to Know - Special Health Hazard Substance List (SHHSL): Carcinogens

US - New Jersey Right to Know Hazardous Substances

US EPCRA Section 313 Chemical List

US National Toxicology Program (NTP) 15th Report Part B. Reasonably Anticipated to be a Human Carcinogen

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

Additional Regulatory Information

Not Applicable

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