Room Spray - Gingerbread House

Ming Fai Enterprise International Co., Ltd

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

SDS No. HKGH0334750901
Initial Date: **30/04/2026**
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SECTION 1 Identification

Product Identifier

Product name	Room Spray - Gingerbread House Contains: Cinnamaldehyde, Oils, orange, sweet
Synonyms	Room Spray - Gingerbread House
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

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 carefully and follow all instructions.

Precautionary statement(s) Prevention

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

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Precautionary statement(s) Response

P321	Specific treatment (see supplemental first aid instruction on this label).
P302+P352	IF ON SKIN: Wash with plenty of water.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Wash contaminated clothing 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	5-9	Castor oil, hydrogenated, ethoxylated
9005-64-5	1-5	Sorbitan monolaurate, ethoxylated
6132-04-3	1-5	Sodium citrate dihydrate
9038-95-3	1-5	Butyl alcohol propoxylated
Not Applicable	1-2	Fragrance – BT603237
5949-29-1	0.5-1	Citric acid, monohydrate
122-99-6	0.5-0.9	Ethylene glycol phenyl ether
25265-71-8	0.525-0.6	Dipropylene glycol (as part of fragrance)
121-32-4	0.375-0.45	Ethyl vanillin (as part of fragrance)
105-95-3	0.3-0.375	Ethylene brassylene (as part of fragrance)
8008-57-9	0.1-0.2	Orange essence oil (Oils, orange, sweet)
121-33-5	0.075-0.15	Vanillin (as part of fragrance)
70445-33-9	0.05-0.1	Ethylhexylglycerin
8024-06-4	0.05-0.1	Vanilla oleoresin (Vanilla planifolia fruit extract)
64-02-8	0.05-0.1	EDTA tetrasodium salt
104-55-2	0.015-0.075	Cinnamaldehyde (as part of fragrance)
123-11-5	0.015-0.075	P-anisaldehyde (as part of fragrance)
141773-73-1	0.015-0.075	Musk propanoate (as part of fragrance)
600-14-6	0.0015-0.015	2,3-pentanedione (as part of fragrance)
57-55-6	0.005-0.01	Propylene glycol
10191-41-0	≤0.0001	Alpha-tocopherol

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.

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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

The product contains a substantial proportion of water, therefore there are no restrictions on the type of extinguishing media which may be used. Choice of extinguishing media should take into account surrounding areas.
Though the material is non-combustible, evaporation of water from the mixture, caused by the heat of nearby fire, may produce floating layers of combustible substances.
In such an event consider:

- foam.
- dry chemical powder.
- carbon dioxide.

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.

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. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire. ▸ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	The emulsion is not combustible under normal conditions. However, it will break down under fire conditions and the hydrocarbon component will burn. Combustible. Will burn if ignited. Combustion products include: ▸ carbon monoxide (CO) ▸ 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	▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by all means available, spillage from entering drains or water courses. ▸ Consider evacuation (or protect in place). ▸ No smoking, naked lights or ignition sources. ▸ Increase ventilation. ▸ Stop leak if safe to do so. ▸ Water spray or fog may be used to disperse / absorb vapour. ▸ Contain or absorb spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▸ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ Avoid skin 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 come in direct contact with human skin or eyes. ▸ **DO NOT** allow material to come in contact with exposed food or food contact surfaces.

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	▸ Suitable PPE must be worn at all times. ▸ 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.
Other information	

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Glass container is suitable for laboratory quantities ▸ Polyethylene or polypropylene container. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	None known

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
sodium citrate dihydrate	9.3 mg/m3	100 mg/m3	610 mg/m3
butyl alcohol propoxylated	27 mg/m3	300 mg/m3	1,800 mg/m3
EDTA tetrasodium salt	82 mg/m3	900 mg/m3	5,500 mg/m3
EDTA tetrasodium salt	75 mg/m3	830 mg/m3	5,000 mg/m3
ethylene glycol phenyl ether	1.5 ppm	16 ppm	97 ppm
propylene glycol	30 mg/m3	1,300 mg/m3	7,900 mg/m3
p-anisaldehyde	21 mg/m3	230 mg/m3	300 mg/m3

Ingredient	Original IDLH	Revised IDLH
water	Not Available	Not Available
castor oil, hydrogenated, ethoxylated	Not Available	Not Available
sorbitan monolaurate, ethoxylated	Not Available	Not Available
sodium citrate dihydrate	Not Available	Not Available
butyl alcohol propoxylated	Not Available	Not Available
EDTA tetrasodium salt	Not Available	Not Available
ethylene glycol phenyl ether	Not Available	Not Available
citric acid, monohydrate	Not Available	Not Available
orange essence oil	Not Available	Not Available
ethylhexylglycerin	Not Available	Not Available
alpha-tocopherol	Not Available	Not Available
vanilla oleoresin	Not Available	Not Available
propylene glycol	Not Available	Not Available
2,3-pentanedione	Not Available	Not Available
cinnamaldehyde	Not Available	Not Available
p-anisaldehyde	Not Available	Not Available
musk propanoate	Not Available	Not Available
vanillin	Not Available	Not Available
ethylene brassylene	Not Available	Not Available
ethyl vanillin	Not Available	Not Available
dipropylene glycol	Not Available	Not Available

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard 'physically' away from the worker and ventilation that strategically 'adds' and 'removes' air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. ▸ Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area.

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	▸ Work should be undertaken in an isolated system such as a 'glove-box' . Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system. ▸ Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within. ▸ Open-vessel systems are prohibited. ▸ Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation. ▸ Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system. ▸ For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▸ Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas). ▸ Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air. ▸ Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed.
Individual protection measures, such as personal protective equipment	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 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	▸ Rubber gloves (nitrile or low-protein, powder-free latex, latex/ nitrile). Employees allergic to latex gloves should use nitrile gloves in preference. ▸ Double gloving should be considered. ▸ PVC gloves. ▸ Change gloves frequently and when contaminated, punctured or torn. ▸ Wash hands immediately after removing gloves. ▸ Protective shoe covers. [AS/NZS 2210] ▸ Head covering. ▸ Insulated gloves: ▸ NOTE: Insulated gloves should be loose fitting so that may be removed quickly if liquid is spilled upon them. Insulated gloves are not made to permit hands to be placed in the liquid; they provide only short-term protection from accidental contact with the liquid.
Body protection	See Other protection below
Other protection	▸ Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent] ▸ Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent] ▸ Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely. ▸ Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. ▸ Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▸ For quantities up to 500 grams a laboratory coat may be suitable. ▸ For quantities up to 1 kilogram a disposable laboratory coat or coverall of low permeability is recommended. Coveralls should be buttoned at collar and cuffs. ▸ For quantities over 1 kilogram and manufacturing operations, wear disposable coverall of low permeability and disposable shoe covers. ▸ For manufacturing operations, air-supplied full body suits may be required for the provision of advanced respiratory protection. ▸ Eye wash unit. ▸ Ensure there is ready access to an emergency shower. ▸ For Emergencies: Vinyl suit

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
'Forsberg Clothing Performance Index'.
Not available

Respiratory protection

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

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

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

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up to 100	10000	-	A-3 P2
100+			Airline**

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

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	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)	5.84	**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)	>65.6	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**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	Product is considered stable and hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

a) Acute Toxicity	Based on available data, the classification criteria are not met.
b) Skin Irritation/Corrosion	Based on available data, the classification criteria are not met.
c) Serious Eye Damage/Irritation	Based on available data, the classification criteria are not met.
d) Respiratory or Skin sensitisation	There is sufficient evidence to classify this material as sensitising to skin or the respiratory system

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e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	There is sufficient evidence to classify this material as carcinogenic
g) Reproductivity	There is sufficient evidence to classify this material as toxic to reproductivity
h) STOT - Single Exposure	Based on available data, the classification criteria are not met.
i) STOT - Repeated Exposure	Based on available data, the classification criteria are not met.
j) Aspiration Hazard	Based on available data, the classification criteria are not met.
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. Not normally a hazard due to non-volatile nature of product
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.
Eye	Although the material 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	There is sufficient evidence to suggest that this material directly causes cancer in humans. Ample evidence from experiments exists that there is a suspicion this material directly reduces fertility. The material may inhibit protein kinase. This may suppress cell or tissue growth or development. Prolonged or repeated skin contact may cause degreasing, followed by drying, cracking and skin inflammation.

Room Spray - Gingerbread House

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)

sorbitan monolaurate, ethoxylated

TOXICITY	IRRITATION
Dermal (Guinea Pig) LD50: >3000 mg/kg	Eye: no adverse effect observed (not irritating)
Inhalation (Rat) LC50: >5.1 mg/l4h	Skin (Human): 15mg/3D (intermittent) - Mild
Oral (Mouse) LD50; >33000 mg/kg	Skin: no adverse effect observed (not irritating)

sodium citrate dihydrate

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg	Not Available
Oral (Mouse) LD50; 5000-6000 mg/kg	

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

EDTA tetrasodium salt

TOXICITY	IRRITATION
Oral (Rat) LD50: 630 mg/kg	Eye (Rodent - rabbit): 100mg/24H - Moderate
	Eye (Rodent - rabbit): 1900ug
	Eye: adverse effect observed (irritating)
	Skin (Human - man): 0.2%
	Skin (Rodent - rabbit): 500mg/24H - Moderate
	Skin: no adverse effect observed (not irritating)

ethylene glycol phenyl ether

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

citric acid, monohydrate

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

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orange essence oil

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

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)

vanilla oleoresin

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg	Skin (Rodent - rabbit): 500mg/24H - Moderate
Oral (Rat) LD50: >5000 mg/kg	

propylene glycol

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 11890 mg/kg	Eye (Rodent - rabbit): 100mg - Mild
Inhalation (Rat) LC50: >44.9 mg/l4h	Eye (Rodent - rabbit): 500mg/24H - Mild
Oral (Rat) LD50: 20000 mg/kg	Eye: no adverse effect observed (not irritating)
	Skin (Human - child): 30%/96H(continuous) - Moderate
	Skin (Human - man): 10%/2D
	Skin (Human - woman): 30%/96H - Mild
	Skin (Human): 104mg/3D (intermittent) - Moderate
	Skin (Human): 20%
	Skin (Human): 500mg/7D - Mild
	Skin: no adverse effect observed (not irritating)

2,3-pentanedione

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2500 mg/kg	Skin (Rodent - rabbit): 500mg/24H - Moderate
Oral (Rat) LD50: 3000 mg/kg	

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)

p-anisaldehyde

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg	Skin (Rodent - rabbit): 500mg/24H - Moderate
Oral (Guinea) LD50; 1260 mg/kg	

musk propanoate

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg	Not Available
Oral (Rat) LD50: >2000 mg/k	

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	

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ethylene brassylene

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

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)

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)

WATER

No significant acute toxicological data identified in literature search.

CASTOR OIL, HYDROGENATED, ETHOXYLATED

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

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	functioning of cells. Infertility in women: One 2007 study found, 'Each 2% increase in the intake of energy from trans unsaturated fats, as opposed to that from carbohydrates, was associated with a 73% greater risk of ovulatory infertility...'. Major depressive disorder: Spanish researchers analysed the diets of 12,059 people over six years and found those who ate the most trans fats had a 48 per cent higher risk of depression than those who did not eat trans fats. One mechanism may be trans-fats' substitution for docosahexaenoic acid (DHA) levels in the orbitofrontal cortex (OFC). Very high intake of trans-fatty acids (43% of total fat) in mice from 2 to 16 months of age was associated with lowered DHA levels in the brain (p=0.001) When the brains of 15 major depressive subjects who had committed suicide were examined post-mortem and compared against 27 age-matched controls, the suicidal brains were found to have 16% less (male average) to 32% (female average) less DHA in the OFC. The OFC is known to control reward, reward expectation and empathy, which are all negatively impacted in depressive mood disorders, as well as regulating the limbic system>
SORBITAN MONOLAURATE, ETHOXYLATED	The Cosmetic Ingredient Review (CIR) Expert Panel concluded that listed polysorbates are safe in cosmetics when formulated to be non-irritating. This conclusion supersedes the conclusion reached in the 1984, 2000, and 2001 CIR safety assessments. This safety assessment combines polysorbates reviewed in 3 previous safety assessments with other polysorbates that have not been reviewed by the CIR Panel into a group of 80 polyethoxylated sorbitan or sorbitol esters of fatty acid. Following oral administration of polysorbate 20 to rats, ester bonds of polysorbates are hydrolyzed within the digestive tract by pancreatic lipase.24 Free fatty acids were absorbed from the digestive tract and oxidized and excreted, mainly as carbon dioxide in exhaled breath. No migration of the polyoxyethylene sorbitan into the thymus lymph nodes has been demonstrated. No sex difference has been detected in the disposition of polysorbates in rats. Following oral ingestion of polysorbate 20 in humans, 90% or more of the administered substance was excreted in the feces as metabolites, with the polyoxyethylene sorbitan structure maintained, and 2%-3% of these metabolites were excreted in the urine The Panel considered the data available to characterize the potential for polysorbates to cause systemic toxicity, irritation, sensitization, reproductive and developmental toxicity, and genotoxicity. They noted the lack of systemic toxicity at low and moderate doses in several acute and repeated-dose oral exposure studies, and low toxicity at high doses; little or no irritation or sensitization in multiple tests of dermal and ocular exposure; the absence of genotoxicity in multiple Ames tests and chromosome aberration tests, and minimal irritation and lack of sensitization in tests of dermal exposure at concentration of use. The Panel recognizes that there are data gaps regarding use and concentration of these ingredients. However, the overall information available on the types of products in which these ingredients are used, concentrations of use and the similar pattern of use raise no safety concerns. The Panel note that polysorbate 20, polysorbate 65, and polysorbate 80 were shown to enhance dermal drug absorption. The Panel cautions that care should be taken in formulating cosmetic products that may contain these ingredients in combination with any ingredients whose safety was based on their lack of dermal absorption, or when dermal absorption was a concern. Especially, care should be taken when creating formulations intended for use on infants. To address the possible presence of 1,4-dioxane and ethylene oxide impurities in these ingredients, the Panel stressed that the cosmetics industry should continue to use the necessary procedures to limit these impurities from the PEG ingredients before blending them into cosmetic formulations. The Panel expressed concern about pesticide residues and heavy metals that may be present in botanical (ie, coconut-derived) ingredients. They stressed that the cosmetics industry should continue to use current good manufacturing practices (cGMPs) to limit impurities. Data from the 1984 safety assessment suggested that polysorbates caused a slight enhancement of tumor development caused by 7,12-dimethyl-benz[a]anthracene (DMBA) and N-methyl-N -nitro-N-nitrosoguanidine (MNNG); however, the data were not consistent. For other compounds, the tumorigenic properties of 3-methyl-cholanthrene (MCA) and 3,4-benz[a]pyrene (BP) were not enhanced by polysorbates. Since the tumor enhancement effects were inconsistent and depended on the simultaneous exposure to strong chemical carcinogens, which are not present in cosmetics, the Panel felt that the weak tumor enhancement effects were not relevant to cosmetic formulations. Because some studies showed minimal irritation at concentrations that are used in cosmetics, the Panel cautioned that products containing these ingredients should be formulated to be non-irritating. It was noted that at the time of the 2001 safety assessment on sorbeth beeswaxes, the Panel had recommended that cosmetic formulations containing PEGs not be used on damaged skin because of the possibility of renal toxicity when PEGs were applied to severely damaged skin, such as in burn patients. Since then, PEGs have been re-reviewed and the additional data demonstrated minimal dermal penetration of low-molecular weight PEGs. The amount of PEGs that would penetrate the stratum corneum barrier, even if damaged, from the use of cosmetics was well below the level of renal toxicity. Therefore, the Panel has removed the caveat that PEGs should not be used on damaged skin. The Panel strongly asserted that it is inappropriate to apply cosmetic products containing high concentrations of PEGs to individuals exhibiting barrier skin disruption through both the stratum corneum and the epidermis. The Panel discussed the issue of incidental inhalation exposure from spray products, including aerosol and pump hair sprays, spray deodorants, spray body and hand products, and spray moisturizing products. The limited acute exposure data available from 1 new inhalation study and 1 historical tracheal study suggest little potential for respiratory effects at relevant doses. These ingredients are reportedly used at concentrations up to 4% in cosmetic products that may be aerosolized. The Panel noted that 95%-99% of droplets/particles would not be respirable to any appreciable amount. Coupled with the small actual exposure in the breathing zone and the concentrations at which the ingredients are used, the available information indicates that incidental inhalation would not be a significant route of exposure that might lead to local respiratory or systemic effects. Safety Assessment of Polysorbates as Used in CosmeticJuly 2015 https://www.cir-safety.org/sites/default/files/PSorba_062015_FR_0.pdf For sorbitan esters, ethoxylated (syn: polyoxyethylene sorbitan esters): Some of the early short-term studies with these polyoxyethylene sorbitan esters in rats and hamsters showed deleterious effects. Subsequent work suggests that these were largely due to diarrhoea resulting from a large amount of unabsorbed polyglycol, possibly aggravated in some experiments by the use of an unsuitable basal diet. Since that time there has been considerable improvement in testing procedures,and more extensive long-term studies have been carried out. It seems reasonable therefore to base the evaluation of these substances on the levels causing no adverse effects indicated by the results of the more recent investigations. The significance of the local tumours which were produced by injection has been discussed at the meeting of the Scientific Group (1966). No increase in tumour incidence has followed the oral intake of polyoxyethylene sorbitan esters. Furthermore, large doses of the oleate and stearate have been well tolerated by human subjects. Polyoxyethylene (20) sorbitan monoester of lauric, oleic, palmitic and stearic acid and triester of stearic acid Seventeenth Report of the Joint FAO/WHO Expert Committee on Food Additives, Wld Hlth Org. Techn. Rep. Ser., 1974, No. 539; FAO Nutrition Meetings Report Series, 1974, No. 53. The sorbitan esters are agents that typically find use as emulsifiers, stabilizers, and thickeners in foods, cosmetics and medical products. They do not represent a toxicological concern since they are derived from naturally occurring materials and are ultimately metabolised back to these same natural constituents.
SODIUM CITRATE DIHYDRATE	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.
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,

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	PPG-3 Butyl Ether had a NOAEL of 1000 mg/kg bw; polypropylene glycol butyl ethers had a NOEL of 100 mg/kg bw/day for clinical observations, higher absolute and relative liver weights, and an increased incidence of liver and thyroid gland hypertrophy; and 1-(2-butoxy-1-methylethoxy)propan-2-ol had a NOAEL of 100 mg/kg/day based on very slight to slight hepatocellular hypertrophy with no corresponding increases in liver weights in low-dose males. In a 90-day oral toxicity study, administration of up to 1000 mg/kg bw/day PPG-3 Butyl Ether to rats in drinking water produced treatment-related increases in absolute and relative liver and kidney weights. The NOAELs in rats and mice exposed to=3000 ppm methoxyisopropanol via inhalation for 2 yrs were 1000 ppm (based on slight body wt decreases in males and females) and 300 ppm (based on altered hepatocellular foci in males), respectively.Dermal application of propylene glycol butyl ether was not embryotoxic or teratogenic to rabbits (=100 mg/kg bw/day applied on days 7-18 of. gestation) or rats (=1.0 ml/kg bw/day applied on days 6-16 of gestation). 1-(2-Butoxy-1-methyl-ethoxy)propan-2-ol (applied on days 6-16 or 6-15 of gestation) also was not embryotoxic or teratogenic in rats. No test-article related adverse developmental or reproductive effects were observed in rats dosed by gavage with up to 1000 mg/kg Buteth-3 or 1-(2-butoxy-1-methylethoxy)propan-2-ol or up to 500 mg/kg bw/day polypropylene glycol butyl ethers. In inhalation studies, exposure of rats to =1.0 mg/l air PPG-3 Methyl Ether did not have any teratogenic or reproductive effects. Exposure to 1000 and 3000 ppm methoxyisopropanol produced some adverse effects in a two-generation study in rats; adverse effects were not observed with 300 ppm. PPG-3 Butyl Ether was not genotoxic in vitro in the Ames test or in vivo in a mouse micronucleus assay. Propylene glycol butyl ether was not genotoxic in an Ames test or a mammalian chromosomal aberration assay in rat lymphocytes, andneither propylene glycol butyl ether or 1-(2-butoxy-1-methylethoxy)propan-2-ol were genotoxic in a mammalian cellmutation assay in CHO cell. In inhalation carcinogenicity studies, mice and rats were exposed by whole body exposure to =3000 ppm methoxyisopropan-ol for 2 yrs. An increase in S-phase DNA synthesis and in MFO activity in the liver was observed in high-dose male mice and rats. Renal epithelial tumors were not observed, and the NOEL for carcinogenicity was 3000 ppm for mice and rats. Undiluted PPG-3 Butyl Ether was not irritating to rabbit skin or eyes, and it was not an irritant or sensitizer in guinea pigs. Polypropylene glycol butyl ethers were classified as non-corrosive in an EpiDermTM study Humans have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents and other cleaning products. Exposure to these chemicals can occur through swallowing, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that relatively high volumes would have to occur to produce any toxic response. No death due to poisoning with alcohol ethoxylates has ever been reported. Studies show that alcohol ethoxylates have low toxicity through swallowing and skin contact. Animal studies show these chemicals may produce gastrointestinal irritation, stomach ulcers, hair standing up, diarrhea and lethargy. Slight to severe irritation occurred when undiluted alcohol ethyoxylates were applied to the skin and eyes of animals. These chemicals show no indication of genetic toxicity or potential to cause mutations and cancers. Toxicity is thought to be substantially lower than that of nonylphenol ethoxylates. 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.
EDTA TETRASODIUM SALT	* Sigma Aldrich - for the dihydrate For ethylendiaminetetraacetic acid (EDTA) and its salts: EDTA is a strong organic acid, with a high affinity for alkaline-earth ions (for example, calcium and magnesium) and heavy-metal ions (such as lad and mercury), resulting in highly stable chelate complexes. The ability of EDTA to complex is used commercially to either promote or inhibit chemical reactions, depending on application. EDTA and its salts are expected to be absorbed by the lungs and the gastrointestinal tract; absorption through skin is unlikely. They cause mild skin irritation, and severe eye irritation. The greatest risk in the human body will occur when the EDTA attempts to scavenge the trace metals used and required by the body. The binding of divalent and trivalent cations by EDTA can cause mineral deficiencies, such as zinc deficiency. These appear to be responsible for all of the known pharmacological effects. EDTA and its salts are mostly eliminated through the urine, with 5% eliminated via the bile, along with the metal ions which are bound to it. Trisodium EDTA has not been found to cause cancer. EDTA and its salts are not likely to cause harm to children and infants at levels likely to be encountered.
ETHYLENE GLYCOL PHENYL ETHER	Bacterial cell mutagen 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. **WARNING:** This substance has been classified by the IARC as Group 1: **CARCINOGENIC TO HUMANS**. For ethylene glycol monoalkyl ethers and their acetates (EGMAEs): Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates. EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers. **Acute Toxicity:** Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE *in vitro* than those of rats. **Repeat dose toxicity:** The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA *in vitro* and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA *in vitro*. **Mutagenicity:** In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in *S. typhimurium* strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. *In vitro* cytogenicity and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and in vivo micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic. **Carcinogenicity:** In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity **Reproductive and developmental toxicity**. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity.

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	The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes). Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m3 and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m3), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m3), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m3) indicate that the members of the category are not teratogenic. The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m3 (rabbit-EGPE), 100 ppm or 425 mg/m3 (rat-EGPE), 50 ppm or 241 mg/m3 (rat EGBE) and 100 ppm or 483 mg/m3 (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m3 (rat and rabbit-EGHE).
CITRIC ACID, MONOHYDRATE	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
ORANGE ESSENCE 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. Fragrance allergens act as haptens, which are small molecules that cause an immune reaction only when attached to a carrier protein. However, not all sensitizing fragrance chemicals are directly reactive, but some require previous activation. A prehapten is a chemical that itself causes little or no sensitization, but it is transformed into a hapten outside the skin by a chemical reaction (oxidation in air or reaction with light) without the requirement of an enzyme. For prehaptens, it is possible to prevent activation outside the body to a certain extent by different measures, for example, prevention of air exposure during handling and storage of the ingredients and the final product, and by the addition of suitable antioxidants. When antioxidants are used, care should be taken that they will not be activated themselves, and thereby form new sensitisers. Prehaptens: Most terpenes with oxidisable allylic positions can be expected to self-oxidise on air exposure. Depending on the stability of the oxidation products that are formed, the oxidized products will have differing levels of sensitization potential. Tests shows that air exposure of lavender oil increased the potential for sensitization. Prohaptens: Compounds that are bioactivated in the skin and thereby form haptens are referred to prohaptens. The possibility of a prohapten being activated cannot be avoided by outside measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Various enzymes play roles in both activating and deactivating prohaptens. Skin-sensitizing prohaptens can be recognized and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or studies of sensitization. QSAR prediction: Prediction of sensitization activity of these substances is complex, especially for those substances that can act both as pre- and prohaptens. d-Limonene is readily absorbed by inhalation and swallowing. Absorption through the skin is reported to the lower than by inhalation. It is rapidly distributed to different tissues in the body, readily metabolized and eliminated, primary through the urine. Limonene shows low acute toxicity by all three routes in animals. Limonene is a skin irritant in both experimental animals and humans. Limited data is available on the potential to cause eye and airway irritation. Autooxidised products of d-limonene have the potential to sensitise the skin. Limited data is available on the potential to cause respiratory sensitization in humans. Limonene will automatically oxidize in the presence of light in air, forming a variety of oxygenated monocyclic terpenes. When contact with these oxidation products occurs, the risk of skin sensitization is high. Limonene does not cause genetic toxicity of birth defects, and it is not toxic to the reproductive system.
ETHYLHEXYLGLYCERIN	Oral (-) LD50: >2000 mg/kg OECD 401 Skin: non-irritant OECD 404 Dermal (-) LD50: >2000 mg/kg OECD 402 Eye: irritant OECD 405 Non-sensitising (OECD 406) The no toxic effect level for oral application to rats over 28 days is 100 mg/kg/day. A NOEL cannot be determined. OECD 407 No experimental information on genotoxicity in vitro or in vivo available. * Schulke Alkyl glyceryl ethers (AGEs) often act as surfactants or skin conditioning agents in cosmetics. These substances show minimal dermal penetration. Furthermore, a review of the available data on toxicity revealed: an absence of genotoxicity in studies using ethylhexylglycerin, chimyl alcohol, batyl alcohol, and glyceryl allyl ether; an absence of reproductive and developmental toxicity in oral studies using ethylhexylglycerin; negative skin irritation/sensitization data in studies using ethylhexylglycerin and chimyl alcohol; and negative phototoxicity/photoallergenicity data in studies using ethylhexylglycerin. Overall, the available toxicity data, coupled with the limited dermal penetration, suggested that these ingredients could be used safely in the present practices of use and concentration. **Oral toxicity:** Using chimyl alcohol a a surrogate of this group approximately 95% is absorbed following oral administration with 40% recovered (as metabolites) in the urine after 12 hours. The lymph shows significant absorption (50%) whilst triglycerides, phospholipids and free fatty acids also seem to incorporate the absorbed substance. No mortalities or exposure-related toxicological findings were observed in rats dosed orally with undiluted ethylhexylglycerin or chimyl alcohol. Ethylhexylglycerin administered orally to rats, at doses up to 800 mg/kg/day, in a 13-week study did not result in any treatment-related deaths, macroscopic observations,or neurotoxicity. A statistically significant increase in absolute and relative-to-body weight liver weights was observed in males of all dose groups and females of the highest dose group. A dose of 50 mg/kg/day (lowest dose) was considered the lowest observed adverse effect level (LOAEL) in one study and no observed adverse effect level (NOAEL) in another. There were no treatment-related mortalities in rats dosed orally with ethylhexylglycerin at doses up to 1,500 mg/kg for 28 days. Increased liver and adrenal weights were observed in the highest dose group, and microscopic findings included slight to moderate liver hypertrophy in rats of all 3 dose groups. The 100 mg/kg dose was defined as the no-observed-adverse effect-level (NOAEL). **Dermal toxicity:** Mean absorption of another surrogate, ethylhexylglycerin through the skin of rabbits is insignificant (0.2% at approximately 2 hours post application) and there were no signs of skin irritation. The quantity of ethylhexylglycerin in the plasma was below the detection limit at the end of the 4 h application period. Over a range of 3 concentrations (44.65, 47.15, and 54.94%) applied to human skin in vitro, mean penetration rates of 2.38, 8.19, and 20.38 ug/cm2/h were reported. Chimyl alcohol was classified as a mild skin irritant in rabbits after a single application, but was a non-irritating to the skin of rabbits in a cumulative skin irritation study. Skin sensitisation was not observed in guinea pigs tested with 0.5% ethylhexylglycerin during induction and challenged with a higher concentration (50%) in the maximization test. Local lymph node assay results for ethylhexylglycerin at concentrations up to 50% were also negative. Products containing ethylhexylglycerin at concentrations ranging from 0.4% to ~1% were neither skin irritants nor sensitisers. Ethylhexylglycerin was not phototoxic or photoallergenic in guinea pigs when tested at concentrations up to 100% in the presence of UVA/UVB light. Chimyl alcohol suppressed the production of chemical mediators of UVB-irradiated keratinocytes in vitro and substantially suppressed UV-induced tanning in human skin. Based on these findings, a new concept for skin whitening via controlling keratinocyte function was proposed 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.

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	Parenteral toxicity: Batyl alcohol stimulated haematopoiesis (both red and white blood cells, following subcutaneous injection) in repeated dose studies involving rats and guinea pigs. **Developmental toxicity:** The results of visceral and skeletal examinations in litters of female rats given oral doses of ethylhexylglycerin (up to 800 mg/kg/day) were negative. In the one-generation developmental toxicity study (same doses) involving male and female rats, oestrous cycles were comparable between groups, but the fertility index for rats of the highest dose group was lower when compared to controls. There were no treatment-related effects on implantation. Necropsy findings in dosed rats found dead or killed did not indicate any treatment-related changes. The no-observed-effect-level (NOEL) for developmental toxicity in both sexes was 50/mg/kg/day **Genotoxicity:** Ethylhexylglycerin, chimyl alcohol, batyl alcohol, glyceryl allyl ether were all non-genotoxic in the Ames test under a variety of conditions. No genotoxicity or clastogenic was exhibited in any of the AGEs using the micronucleus, chromosomal aberration assays assays, Studies on the carcinogenicity of the AGEs were not found in the published literature
ALPHA-TOCOPHEROL	[ROCHE] * Bronson and Jacobs SDS (for similar products) Use in foodstuffs is consistent with low order of toxicity. Based on laboratory and animal testing, exposure to the material may result in irreversible effects and mutations in humans. alpha-Tocopherol was non-mutagenic and non-carcinogenic, and the results of reproduction/ teratology studies did not indicate that alpha-tocopherol had adverse effects on reproductive function. However, in a long-term study in rats, a no-effect level could not be established with respect to effects on blood clotting and liver histology, and there was evidence from human studies that excessive intakes of alpha-tocopherol could cause haemorrhage. Other adverse effects noted in clinical studies at doses of > 720 mg alpha-tocopherol/day included weakness, fatigue, creatinuria and effects on steroid hormone metabolism. Clinical studies indicate that, generally, intakes of below about 720 mg/day are without adverse effects in man, but one investigation in elderly patients showed an increase in serum cholesterol at doses of 300 mg alpha-tocopherol daily. Incidences of allergic reactions seem to be very rare. alpha-Tocopherol may be an essential nutrient. The U.S. National Academy of Sciences/National Research Council has recommended a dietary allowance of 0.15 mg/kg b.w./day. However, excessive intakes of alpha-tocopherol produce adverse clinical and biochemical effects, and self-medication with large doses of vitamin E preparations could present a hazard. The previously-allocated ADI was amended to include a lower value, which reflects the fact that alpha-tocopherol may be an essential nutrient. The upper value, which represents the maximum value for the AID, is based on clinical experience in man. IPCS Inchem: https://www.inchem.org/documents/jecfa/jecmono/v21je05.htm
VANILLA OLEORESIN	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. 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. 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.
PROPYLENE GLYCOL	The acute oral toxicity of propylene glycol is very low; large amounts are needed to cause perceptible health damage in humans. Serious toxicity generally occurs only at blood concentrations over 1 g/L, which requires extremely high intake over a relatively short period of time; this is nearly impossible with consuming foods or supplements which contain 1g/kg of PG at most. Poisonings are usually due to injection through a vein or accidental swallowing of large amounts by children. The potential for long-term oral toxicity is also low. Prolonged contact with propylene glycol is essentially non-irritating to the skin. Undiluted propylene glycol is minimally irritating to the eye, and can produce a slight, temporary inflammation of the conjunctiva. Exposure to mists may cause irritation of both the eye and the upper airway. Inhalation of propylene glycol vapours may be irritating to some individuals. It is therefore recommended that propylene glycol not be used in applications where inhalation exposure or human eye contact with the spray mists of these materials is likely, such as fogs for theatrical productions or antifreeze solutions for emergency eye wash stations. Propylene glycol is metabolized in humans to pyruvic acid, acetic acid, lactic acid and propionaldehyde; the last of which is potentially hazardous. Propylene glycol show s no evidence of causing cancer or genetic toxicity. Research has suggested that individuals who cannot tolerate propylene glycol probably experience a special form of irritation, but they only rarely develop allergic contact dermatitis. Other investigators believe that the incidence of allergic contact dermatitis in people exposed to propylene glycol may be greater than 2% in patients with eczema. One study strongly suggests a connection between airborne concentrations of propylene glycol in houses and development of asthma and allergic reactions, such as inflammation of the nose and hives, in children. Another study suggested that the concentration of PGEs (propylene glycol and glycol ethers) in indoor air is linked to increased risk of developing numerous respiratory and immune disorders in children, including asthma, hay fever, eczema and allergies, with increased risk ranging from 50% to 180%. This concentration has been linked to use of water-based paints and water-based system cleansers. Patients with bladder inflammation and vulvodynia (chronic pain of the vulva) may be especially sensitive to propylene glycol. Women suffering with yeast infections may notice that some over the counter creams cause intense burning. Post-menopausal women who require the use of an oestrogen cream may notice that creams made with propylene glycol often cause extremely uncomfortable burning along the vulva and around the anus. Some electronic cigarette users who inhale propylene glycol vapour may experience dryness of the throat or shortness of breath. Adverse responses to administration of drugs which use propylene glycol as an incipient have been seen in a number of people especially at high doses. These include low blood pressure, slow heart rate, ECG abnormalities, heartbeat irregularities, lactic acidosis, breakdown of red cells and cardiac arrest.
2,3-PENTANEDIONE	A member or analogue of EFSA Chemical Group 10 secondary aliphatic saturated or unsaturated alcohols, ketones, ketals and esters with a secondary or tertiary oxygenated functional group used as flavourings No safety concern would arise for the consumer from the use of these compounds up to the highest proposed level in feeds. Hazards for skin and eye contact and respiratory exposure are recognised for the majority of the compounds under application. Most are classified as irritating to the respiratory system. Aliphatic acyclic and alicyclic alpha-diketones and alpha-hydroxyketones are generally used as flavouring agents up to average maximum levels of 200 ppm. In rats and mice, orally administered aliphatic alpha-diketones are rapidly absorbed from the gastrointestinal tract. It is anticipated that at low levels of exposure, humans will metabolize aliphatic acyclic alpha-diketone principally by alpha-hydroxylation and subsequent oxidation of the terminal methyl group to yield the corresponding ketocarboxylic acid. The acid may undergo oxidative decarboxylation to yield carbon dioxide and a simple aliphatic carboxylic acid, which may be completely metabolized in the fatty acid pathway and citric acid cycle. At high concentrations, another detoxification pathway is used which involves reduction to the diol and subsequent conjugation with glucuronic acid. Acyclic alpha-diketones and alpha-hydroxyketones without a terminal methyl group and alicyclic diketones and hydroxyketones are mainly metabolized by reduction to the corresponding diol, followed by glucuronic acid conjugation and excretion Compounds belonging to CG 10 are absorbed from the gastrointestinal tract and share common pathways of metabolism: (i) hydrolysis of esters by carboxylesterases, (ii) reduction of ketones to alcohols, (iii) oxidation of alcohols to acids, (iv) alpha–hydroxylation of the terminal methyl group to yield corresponding ketocarboxylic acids, (v) oxidative decarboxylation to yield carbon dioxide and an aliphatic carboxylic acid, and (vi) conjugation of alpha-hydroxyketones or their diol metabolites with glucuronic acid. Aliphatic acyclic diketones and alpha–hydroxyketones, which contain a carbonyl function at the 2-position (i.e. a methyl ketone) are expected to undergo alpha–hydroxylation and subsequent oxidation of the terminal methyl group to eventually yield corresponding ketocarboxylic acids. These compounds are intermediary metabolites (e.g.alpha-ketoacids), which may undergo oxidative decarboxylation to yield carbon dioxide and an aliphatic carboxylic acid. The acid is then metabolised via beta-oxidation and the citric acid cycle. beta-Ketoacids and derivatives readily undergo decarboxylation to yield breakdown products, which are incorporated into normal biochemical pathways. Alternatively, the methyl-substituted diketones may be successively reduced to the corresponding hydroxyketones and diols, which are excreted in the urine as glucuronic acid conjugates. This pathway is favoured at elevated in vivo concentrations, especially for longer chain length ketones. If the carbonyl function is located elsewhere on the chain, reduction is the predominant pathway. alpha-hydroxyketones or their diol metabolites may be excreted as glucuronic acid conjugates. Low concentrations of aliphatic acyclic methyl ketones are mainly metabolised by oxidation of the terminal methyl group. At higher concentrations, acyclic

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	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 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)
CINNAMALDEHYDE	Animal testing suggests that the toxicity through swallowing cinnamyl aldehyde derivatives is very low. The potential for toxicity through skin exposure is similarly low. Cinnamaldehyde and its alkyl-substituted derivatives do not directly cause mutations or genetic damage. However, animal testing suggests that they may result in poor development of the skull and kidney in the foetus. 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. 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.
MUSK PROPANOATE	Sensitization (Guinea pig Maximisation)(OECD 406): Non-sensitizing * Sensitization (HRIPT): Non-sensitizing @ 20% * Sub-acute toxicity (28-day, gav., rat)(OECD 407): NOAEL 250 mg/kg/day * Mutagenicity (OECD 471): Non-mutagenic * Mutagenicity (OECD 473): Non-mutagenic * One generation reproduction (OECD 415): NOEL = 1000 mg/kg/day * * Vigon MSDS 551muskali
VANILLIN	Miosis, somnolence, muscle weakness, coma, respiratory stimulation, maternal effects involving ovaries, fallopian tubes, uterus, cervix and vagina recorded.
ETHYLENE BRASSYLENE	* Bedoukian 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
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.

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	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.
CASTOR OIL, HYDROGENATED, ETHOXYLATED & SORBITAN MONOLAURATE, 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.
SORBITAN MONOLAURATE, ETHOXYLATED & ETHYLENE GLYCOL PHENYL ETHER & PROPYLENE GLYCOL & 2,3-PENTANEDIONE & P-ANISALDEHYDE & ETHYLENE BRASSYLENE & ETHYL VANILLIN	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.
EDTA TETRASODIUM SALT & ORANGE ESSENCE OIL & VANILLA OLEORESIN & 2,3-PENTANEDIONE & CINNAMALDEHYDE & VANILLIN & ETHYL VANILLIN	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
EDTA TETRASODIUM SALT & ETHYLENE GLYCOL PHENYL ETHER & CITRIC ACID, MONOHYDRATE & 2,3-PENTANEDIONE & CINNAMALDEHYDE & P-ANISALDEHYDE & ETHYLENE BRASSYLENE & ETHYL VANILLIN	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.
ETHYLENE GLYCOL PHENYL ETHER & 2,3-PENTANEDIONE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
P-ANISALDEHYDE & VANILLIN	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.
P-ANISALDEHYDE & VANILLIN & ETHYL VANILLIN	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)
VANILLIN & ETHYL VANILLIN	For vanillin: 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.

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

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

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SECTION 12 Ecological information

Toxicity

Room Spray - Gingerbread House

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	72h	Algae or other aquatic plants	>1mg/l	2
EC50	48h	Crustacea	>1mg/l	2
EC50(ECx)	48h	Crustacea	>1mg/l	2
LC50	96h	Fish	>1mg/l	2

sorbitan monolaurate, ethoxylated

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	383mg/l	2

sodium citrate dihydrate

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	72h	Algae or other aquatic plants	>500mg/l	1
EC50	48h	Crustacea	>500mg/l	1
NOEC(ECx)	72h	Algae or other aquatic plants	62.5mg/l	2
EC50	96h	Algae or other aquatic plants	744.74mg/l	2
LC50	96h	Fish	1350mg/l	1
EC50	72h	Algae or other aquatic plants	445mg/l	2
EC50	48h	Crustacea	>100mg/l	2
EC50	96h	Algae or other aquatic plants	315mg/l	2
NOEC(ECx)	96h	Algae or other aquatic plants	<15.9mg/l	2
LC50	96h	Fish	564mg/l	2
EC50	48h	Crustacea	89-101mg/L	4
EC50(ECx)	48h	Crustacea	89-101mg/L	4
LC50	96h	Fish	48-52mg/L	4

EDTA tetrasodium salt

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	1.01mg/l	1
EC50	48h	Crustacea	>100mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	0.39mg/l	1
LC50	96h	Fish	>100mg/l	2

ethylene glycol phenyl ether

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

citric acid, monohydrate

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

orange essence oil

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.36mg/l	2
EC50	48h	Crustacea	0.45mg/l	2
LC50	96h	Fish	0.32mg/l	2
EC50(ECx)	72h	Algae or other aquatic plants	0.36mg/l	2

ethylhexylglycerin

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

Continued...

alpha-tocopherol

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

vanilla oleoresin

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

propylene glycol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	19300mg/l	2
EC50	48h	Crustacea	>114.4mg/L	4
EC50	96h	Algae or other aquatic plants	19000mg/l	2
NOEC(ECx)	336h	Algae or other aquatic plants	<5300mg/l	1
LC50	96h	Fish	710mg/L	4

2,3-pentanedione

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	20.1mg/l	2
EC50	48h	Crustacea	22.4mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	15.5mg/l	2

cinnamaldehyde

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

p-anisaldehyde

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	504h	Crustacea	0.71mg/l	2
LC50	96h	Fish	148.32mg/l	2
EC50	72h	Algae or other aquatic plants	68.4mg/l	2
EC50	48h	Crustacea	82.8mg/l	2

musk propanoate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>1.1mg/l	Not Available
EC50	48h	Crustacea	3.3mg/l	Not Available
EC50(ECx)	72h	Algae or other aquatic plants	>1.1mg/l	Not Available
EC50	96h	Algae or other aquatic plants	>1.1mg/l	2
LC50	96h	Fish	3.6mg/l	Not Available

vanillin

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

ethylene brassylene

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>6.94mg/l	2
EC50	48h	Crustacea	3.65mg/l	2
NOEC(ECx)	504h	Crustacea	0.088mg/l	2
EC50	96h	Algae or other aquatic plants	0.788mg/l	2
LC50	96h	Fish	1.23mg/l	2

ethyl vanillin

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

dipropylene glycol

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

Continued...

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, monohydrate	LOW	LOW
orange essence oil	HIGH	HIGH
alpha-tocopherol	HIGH	HIGH
propylene glycol	LOW	LOW
2,3-pentanedione	HIGH	HIGH
cinnamaldehyde	LOW	LOW
p-anisaldehyde	LOW	LOW
vanillin	LOW	LOW
ethylene brassylene	LOW	LOW
ethyl vanillin	LOW	LOW
dipropylene glycol	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
water	LOW (LogKOW = -1.38)
sorbitan monolaurate, ethoxylated	LOW (LogKOW = -2.03)
sodium citrate dihydrate	LOW (LogKOW = -0.28)
butyl alcohol propoxylated	LOW (LogKOW = 1.2706)
ethylene glycol phenyl ether	LOW (LogKOW = 1.16)
citric acid, monohydrate	LOW (LogKOW = -1.64)
orange essence oil	HIGH (LogKOW = 5.6842)
alpha-tocopherol	LOW (LogKOW = 12.18)
vanilla oleoresin	LOW (LogKOW = 1.21)
propylene glycol	LOW (BCF = 1)
2,3-pentanedione	LOW (LogKOW = -0.8453)
cinnamaldehyde	LOW (BCF = 10)
p-anisaldehyde	LOW (LogKOW = 1.76)
vanillin	LOW (LogKOW = 1.21)
ethylene brassylene	HIGH (LogKOW = 4.71)
ethyl vanillin	LOW (LogKOW = 1.58)
dipropylene glycol	LOW (BCF = 4.6)

Mobility in soil

Ingredient	Mobility
butyl alcohol propoxylated	LOW (Log KOC = 10)
ethylene glycol phenyl ether	LOW (Log KOC = 12.12)
citric acid, monohydrate	LOW (Log KOC = 10)
orange essence oil	LOW (Log KOC = 2899)
alpha-tocopherol	LOW (Log KOC = 51280000)
propylene glycol	HIGH (Log KOC = 1)
2,3-pentanedione	HIGH (Log KOC = 1)
cinnamaldehyde	LOW (Log KOC = 102.4)
p-anisaldehyde	LOW (Log KOC = 23.26)
vanillin	LOW (Log KOC = 38.45)
ethylene brassylene	LOW (Log KOC = 879.2)
ethyl vanillin	LOW (Log KOC = 70.92)
dipropylene glycol	HIGH (Log KOC = 1)

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. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

Marine Pollutant	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

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

Product name	Group
water	Not Applicable
castor oil, hydrogenated, ethoxylated	Not Applicable
sorbitan monolaurate, ethoxylated	Not Applicable
sodium citrate dihydrate	Not Applicable
butyl alcohol propoxylated	Not Applicable
EDTA tetrasodium salt	Not Applicable
ethylene glycol phenyl ether	Not Applicable
citric acid, monohydrate	Not Applicable
orange essence oil	Not Applicable
ethylhexylglycerin	Not Applicable
alpha-tocopherol	Not Applicable
vanilla oleoresin	Not Applicable
propylene glycol	Not Applicable
2,3-pentanedione	Not Applicable
cinnamaldehyde	Not Applicable
p-anisaldehyde	Not Applicable
musk propanoate	Not Applicable
vanillin	Not Applicable
ethylene brassylene	Not Applicable
ethyl vanillin	Not Applicable
dipropylene glycol	Not Applicable

Continued...

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
water	Not Applicable
castor oil, hydrogenated, ethoxylated	Not Applicable
sorbitan monolaurate, ethoxylated	Not Applicable
sodium citrate dihydrate	Not Applicable
butyl alcohol propoxylated	Not Applicable
EDTA tetrasodium salt	Not Applicable
ethylene glycol phenyl ether	Not Applicable
citric acid, monohydrate	Not Applicable
orange essence oil	Not Applicable
ethylhexylglycerin	Not Applicable
alpha-tocopherol	Not Applicable
vanilla oleoresin	Not Applicable
propylene glycol	Not Applicable
2,3-pentanedione	Not Applicable
cinnamaldehyde	Not Applicable
p-anisaldehyde	Not Applicable
musk propanoate	Not Applicable
vanillin	Not Applicable
ethylene brassylene	Not Applicable
ethyl vanillin	Not Applicable
dipropylene glycol	Not Applicable

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

sorbitan monolaurate, ethoxylated is found on the following regulatory lists

US EPA Pesticide Chemical Search - Conventional Chemical
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

sodium citrate dihydrate 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 EPA Pesticide Chemical Search - Antimicrobial
US EPA Pesticide Chemical Search - Biopesticides
US EPA Pesticide Chemical Search - Conventional Chemical
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

EDTA tetrasodium salt is found on the following regulatory lists

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

ethylene glycol phenyl ether is found on the following regulatory lists

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

citric acid, monohydrate is found on the following regulatory lists

US EPA Pesticide Chemical Search - Antimicrobial
US EPA Pesticide Chemical Search - Biopesticides
US EPA Pesticide Chemical Search - Conventional Chemical
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

Continued...

orange essence oil is found on the following regulatory lists

US EPA Pesticide Chemical Search - Biopesticides
US EPA Pesticide Chemical Search - Conventional Chemical
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

vanilla oleoresin is found on the following regulatory lists

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

propylene glycol is found on the following regulatory lists

US - New Jersey Right to Know Hazardous Substances
US - Pennsylvania - Hazardous Substance List
US AIHA Workplace Environmental Exposure Levels (WEELs)
US ATSDR Minimal Risk Levels for Hazardous Substances (MRLs)
US DOE Temporary Emergency Exposure Limits (TEELs)
US EPA Integrated Risk Information System (IRIS)
US EPA Pesticide Chemical Search - Antimicrobial
US EPA Pesticide Chemical Search - Conventional Chemical
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US Toxicology Excellence for Risk Assessment (TERA) Workplace Environmental Exposure Levels (WEEL)

2,3-pentanedione is found on the following regulatory lists

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

cinnamaldehyde is found on the following regulatory lists

US EPA Pesticide Chemical Search - Biopesticides
US EPA Pesticide Chemical Search - Conventional Chemical
US OSHA Appendix A to § 1910.119—List of Highly Hazardous Chemicals, Toxics and Reactives (Mandatory)
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

p-anisaldehyde is found on the following regulatory lists

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

musk propanoate 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)

ethylene brassylene 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

dipropylene glycol is found on the following regulatory lists

US - Pennsylvania - Hazardous Substance List
US EPA Pesticide Chemical Search - Antimicrobial
US EPA Pesticide Chemical Search - Conventional Chemical
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

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