



JTC IMPORT/EXPORT P/L

PowerHouse - Lemon Cleaner 5in1 Action 28 fl oz

JTC Import Export Pty Ltd

Chemwatch: 7963-81

Version No: 2.1

Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Chemwatch Hazard Alert Code: 3

Initial Date: 20/06/2025

Revision Date: 20/06/2025

Print Date: 23/06/2025

S.GHS.AUS.EN

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

Product Identifier

Product name	PowerHouse - Lemon Cleaner 5in1 Action 28 fl oz
Chemical Name	Not Applicable
Synonyms	Synonyms; Product Code: 82393 PowerHouse® - Lemon Cleaner 5in1 Action 28 fl oz
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Multipurpose detergent. Use according to manufacturer's directions.
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Details of the manufacturer or importer of the safety data sheet

Registered company name	JTC Import Export Pty Ltd
Address	98-126 South Park Drive Dandenong South VIC 3175 Australia
Telephone	+61 3 9532 5100
Fax	+61 3 9532 6102
Website	https://www.jtcimportexport.com.au
Email	sales@jtcimportexport.com.au

Emergency telephone number

Association / Organisation	JTC Import Export Pty Ltd	CHEMWATCH EMERGENCY RESPONSE (24/7)
Emergency telephone number(s)	+61 3 9532 5100 (Mon-Thurs 8.30am to 5.30pm; Friday 8.30am to 3pm)	+61 1800 951 288 (ID#: 7963-81)
Other emergency telephone number(s)	Not Available	+61 3 9573 3188

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification ^[1]	Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2A
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
Signal word	Warning

Hazard statement(s)

H317	May cause an allergic skin reaction.
H319	Causes serious eye irritation.

Precautionary statement(s) Prevention

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P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P302+P352	IF ON SKIN: Wash with plenty of water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

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.
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No further product hazard information.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
68891-38-3	1-5	<u>sodium linear-(C12-14)alkyl ether sulfate</u>
68515-73-1	0.5-1	<u>D-glucose, decyl octyl ethers, oligomeric</u>
64-17-5	0.5-1	<u>ethanol</u>
61788-85-0	0.1-0.5	<u>castor oil, hydrogenated, ethoxylated</u>
5989-27-5	0.01-0.05	<u>d-limonene</u>
8000-41-7	<0.01	<u>alpha-terpineol</u>
18479-58-8	<0.01	<u>dihydromyrcenol</u>
5949-29-1	<0.01	<u>citric acid, monohydrate</u>
101-86-0	<0.01	<u>alpha-hexylcinnamaldehyde</u>
106-22-9	<0.01	<u>beta-citronellol</u>
5392-40-5	<0.01	<u>citral</u>
99-87-6	<0.01	<u>p-cymene</u>
55965-84-9	<0.01	<u>isothiazolinones, mixed</u>
8008-56-8	<0.01	<u>lemon oil</u>
1934-21-0	<0.01	<u>C.I. Acid Yellow 23</u>
8004-92-0	<0.01	<u>C.I. Acid Yellow 3</u>
2783-94-0	<0.01	<u>C.I. Food Yellow 3</u>
90480-35-6	<0.01	<u>citral diethyl acetal</u>
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▶ Other measures are usually unnecessary.
Ingestion	▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice.

Indication of any immediate medical attention and special treatment needed
Treat symptomatically.

SECTION 5 Firefighting measures

- Extinguishing media**
- ▶ Water spray or fog.
 - ▶ Foam.
 - ▶ Dry chemical powder.
 - ▶ BCF (where regulations permit).
 - ▶ Carbon dioxide.

Special hazards arising from the substrate or mixture

Fire Incompatibility	▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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Advice for firefighters

Fire Fighting	▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear full body protective clothing with breathing apparatus. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ Avoid spraying water onto liquid pools. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	▶ Combustible. ▶ Slight fire hazard when exposed to heat or flame. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. ▶ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO2) sulfur oxides (SOx) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures
See section 8

Environmental precautions
See section 12

Methods and material for containment and cleaning up

Minor Spills	▶ Remove all ignition sources. ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb spill with sand, earth, inert material or vermiculite. ▶ Wipe up. ▶ Place in a suitable, labelled container for waste disposal.
Major Spills	▶ 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	▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ Avoid smoking, naked lights or ignition sources.
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	▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	▶ Store in original containers. ▶ Keep containers securely sealed. ▶ No smoking, naked lights or ignition sources. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

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

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	ethanol	Ethyl alcohol	1000 ppm / 1880 mg/m3	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
sodium linear-(C12-14)alkyl ether sulfate	Not Available	Not Available
D-glucose, decyl octyl ethers, oligomeric	Not Available	Not Available
ethanol	Not Available	Not Available
castor oil, hydrogenated, ethoxylated	Not Available	Not Available
d-limonene	Not Available	Not Available
alpha-terpineol	Not Available	Not Available
dihydromyrcenol	Not Available	Not Available
citric acid, monohydrate	Not Available	Not Available
alpha-hexylcinnamaldehyde	Not Available	Not Available
beta-citronellol	Not Available	Not Available
citral	Not Available	Not Available
p-cymene	Not Available	Not Available
isothiazolinones, mixed	Not Available	Not Available
lemon oil	Not Available	Not Available
C.I. Acid Yellow 23	Not Available	Not Available
C.I. Acid Yellow 3	Not Available	Not Available
C.I. Food Yellow 3	Not Available	Not Available
citral diethyl acetal	Not Available	Not Available

Exposure controls

Appropriate engineering controls	If exposure to workplace dust is not controlled, respiratory protection is required; wear SAA approved dust respirator.
Individual protection measures, such as personal protective equipment	
Eye and face protection	▶ Safety glasses with side shields. ▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].
Skin protection	See Hand protection below
Hands/feet protection	▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber

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NOTE:

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

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.

The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.

Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

- frequency and duration of contact,
- chemical resistance of glove material,
- glove thickness and
- dexterity

Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).

- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
- Contaminated gloves should be replaced.

As defined in ASTM F-739-96 in any application, gloves are rated as:

- Excellent when breakthrough time > 480 min
- Good when breakthrough time > 20 min
- Fair when breakthrough time < 20 min
- Poor when glove material degrades

For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.

It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.

Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.

Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:

- Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.
- Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential

Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

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.
- ▶ Overalls.
- ▶ P.V.C apron.
- ▶ Barrier cream.
- ▶ Skin cleansing cream.
- ▶ Eye wash unit.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the: "Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:
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Material	CPI
NITRILE	A
BUTYL	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
VITON	C

* CPI - Chemwatch Performance Index
A: Best Selection

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 5 x ES	Air-line*	AK-2 P2	AK-PAPR-2 P2 ^
up to 10 x ES	-	AK-3 P2	-
10+ x ES	-	Air-line**	-

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

- ▶ Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- ▶ The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

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	Yellow liquid with characteristic odour; mixes with water.		
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)	Not Available	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Available	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	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Heat of Combustion (kJ/g)	Not Available	Ignition Distance (cm)	Not Available
Flame Height (cm)	Not Available	Flame Duration (s)	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	Enclosed Space Ignition Deflagration Density (g/m3)	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

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	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	There is sufficient evidence to classify this material as sensitising to skin or the respiratory system
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	Based on available data, the classification criteria are not met.
g) Reproductivity	Based on available data, the classification criteria are not met.
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 (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.
Ingestion	The material has NOT been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence.

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Skin Contact	There is some evidence to suggest that this material can cause inflammation of the skin on contact in some persons.	
Eye	This material causes serious eye irritation.	
Chronic	Skin contact with the material is more likely to cause a sensitisation reaction in some persons compared to the general population. Ample evidence exists from experimentation that reduced human fertility is directly caused by exposure to the material. Ample evidence exists, from results in experimentation, that developmental disorders are directly caused by human exposure to the material. A number of common flavor and fragrance chemicals can form peroxides surprisingly fast in air. Antioxidants can in most cases minimize the oxidation. Fragrance terpenes are easily oxidized in air. Non-oxidised forms are very weak sensitizers; however, after oxidation, the hydroperoxides are strong sensitisers which may cause allergic reactions. Autooxidation of fragrance terpenes contributes greatly to fragrance allergy. There is the need to test for compounds the patients are actually exposed to, not only the ingredients originally applied in commercial formulations. Peroxidisable terpenes and terpenoids should only be used when the level of peroxides is kept to the lowest practicable level, for instance by adding antioxidants at the time of production. This should be less than 10 millimoles of peroxide per litre. This is because peroxides may have sensitizing properties. Prolonged exposure to ethanol may cause damage to the liver and cause scarring. It may also worsen damage caused by other agents.	
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	Not Available	Not Available
sodium linear-(C12-14)alkyl ether sulfate	TOXICITY	IRRITATION
	dermal (rat) LD50: >540 mg/kg ^[1]	Eye (Rodent - rabbit): 100uL/24H - Severe
	Oral (Rat) LD50: >540 mg/kg ^[1]	Eye (Rodent - rabbit): 20mg/24H - Moderate
		Eye: adverse effect observed (irreversible damage) ^[1]
		Skin (Rodent - guinea pig): 5%/9H (intermittent)
		Skin (Rodent - rabbit): 25mg/24H - Moderate
		Skin (Rodent - rabbit): 500mg/24H - Severe
D-glucose, decyl octyl ethers, oligomeric	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
ethanol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 17100 mg/kg ^[1]	Eye (Rodent - rabbit): 0.1mL
	Inhalation (Rat) LC50: 64000 ppm4h ^[2]	Eye (Rodent - rabbit): 100mg/4S - Moderate
	Oral (Rat) LD50: 7060 mg/kg ^[2]	Eye (Rodent - rabbit): 100uL - Moderate
		Eye (Rodent - rabbit): 500mg - Severe
		Eye (Rodent - rabbit): 500mg/24H - Mild
		Eye: adverse effect observed (irritating) ^[1]
		Eye: no adverse effect observed (not irritating) ^[1]
		Skin (Human): 70%/2D
		Skin (Rodent - rabbit): 20mg/24H - Moderate
castor oil, hydrogenated, ethoxylated	TOXICITY	IRRITATION
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
d-limonene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Skin (Rodent - mouse): 700mg/7D (intermittent) - Severe
		Skin (Rodent - rabbit): 10%/24H - Mild
		Skin (Rodent - rabbit): 500mg/24H - Moderate
alpha-terpineol		Skin (Rodent - rat): 100%/1H
		Skin: no adverse effect observed (not irritating) ^[1]
	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (Mammal - species unspecified): 12.5% - Mild
	Inhalation (Rat) LC50: >4.76 mg/l4h ^[1]	Eye (Mammal - species unspecified): 12.5% - Mild
	Oral (Mouse) LD50: 12.8 mg/kg ^[2]	Eye: adverse effect observed (irreversible damage) ^[1]
		Eye: adverse effect observed (irritating) ^[1]
		Skin (Rodent - mouse): 50% - Severe
		Skin (Rodent - rabbit): 100%/4H - Mild
		Skin (Rodent - rabbit): 500mg/24H - Moderate
		Skin (Rodent - rabbit): 500mg/24H - Moderate

		Skin: adverse effect observed (irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
dihydromyrcenol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	Eye (Rodent - rabbit): 0.05% - Mild
	Oral (Rat) LD50: 3600 mg/kg ^[2]	Eye (Rodent - rabbit): 0.1mL
		Eye (Rodent - rabbit): 0.1mL
		Eye (Rodent - rabbit): 5%
		Eye (Rodent - rabbit): 7.5%
		Eye (Rodent - rabbit): 7.5% - Mild
		Eye: adverse effect observed (irritating) ^[1]
		Skin (Rodent - rabbit): 0.5mL/4H
		Skin (Rodent - rabbit): 0.5mL/4H - Mild
		Skin (Rodent - rabbit): 500mg/24H - Mild
		Skin: adverse effect observed (irritating) ^[1]
citric acid, monohydrate	TOXICITY	IRRITATION
	Oral (Mouse) LD50; 5790 mg/kg ^[2]	Eye (Rodent - rabbit): 5mg/30S - Mild
		Eye (Rodent - rabbit): 750ug/24H - Severe
		Skin (Rodent - rabbit): 0.5mL - Moderate
		Skin (Rodent - rabbit): 500mg/24H - Mild
alpha-hexylcinnamaldehyde	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >3000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Mouse) LD50; 2300 mg/kg ^[2]	Skin (Human): 5%/2D
		Skin (Rodent - guinea pig): 100mg/24H - Severe
		Skin (Rodent - mouse): 30%/3D(intermittent)
		Skin (Rodent - rabbit): 100mg/24H - Severe
		Skin (Rodent - rabbit): 500mg/24H - Moderate
beta-citronellol		Skin: adverse effect observed (irritating) ^[1]
	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 2650 mg/kg ^[2]	Eye (Rodent - rabbit): 0.42% - Moderate
	Oral (Rat) LD50: 3450 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]
		Skin (Human - man): 16mg/48H - Moderate
		Skin (Human): 2%/2D
		Skin (Human): 32%/48H
		Skin (Human): 5%
		Skin (Human): 5%
		Skin (Mammal - pig): 100%/48H
		Skin (Rodent - guinea pig): 100%/48H
		Skin (Rodent - guinea pig): 100mg/24H - Severe
		Skin (Rodent - rabbit): 0.42%/4H - Moderate
		Skin (Rodent - rabbit): 0.5mL/4H - Severe
		Skin (Rodent - rabbit): 100mg/24H - Severe
		Skin: adverse effect observed (irritating) ^[1]
citral	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Skin (Human - man): 16mg/48H - Severe
	Oral (Rat) LD50: 4960 mg/kg ^[2]	Skin (Human - woman): 2%
		Skin (Human): 2%/2D
		Skin (Human): 40mg/24H - Mild
		Skin (Mammal - pig): 50mg/48H - Severe
		Skin (Rodent - guinea pig): 1%/48H - Moderate
		Skin (Rodent - guinea pig): 100mg/24H - Severe
		Skin (Rodent - rabbit): 100mg/24H - Severe
		Skin (Rodent - rabbit): 500mg/24H - Moderate
p-cymene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
	Oral (Rat) LD50: 4750 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]

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		Skin (Rodent - rabbit): 500mg/24H - Moderate
		Skin: adverse effect observed (irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
isothiazolinones, mixed	TOXICITY	IRRITATION
	dermal (rat) LD50: >1008 mg/kg ^[1]	Eye: adverse effect observed (irreversible damage) ^[1]
	Inhalation (Rat) LC50: 0.171 mg/l4h ^[1]	Skin (Human - woman): 0.01%
	Oral (Rat) LD50: 53 mg/kg ^[2]	Skin (Human): 0.01% - Severe
		Skin (Human): 0.1%/48H
		Skin: adverse effect observed (corrosive) ^[1]
		Skin: adverse effect observed (irritating) ^[1]
lemon oil	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	Skin (Rodent - mouse): 100 - Mild
	Oral (Rat) LD50: 2840 mg/kg ^[2]	Skin (Rodent - rabbit): 500mg/24H - Moderate
C.I. Acid Yellow 23	TOXICITY	IRRITATION
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
C.I. Acid Yellow 3	TOXICITY	IRRITATION
	Oral (Rat) LD50: 2000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
C.I. Food Yellow 3	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
citral diethyl acetal	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >5000 mg/kg ^[2]	Skin (Rodent - rabbit): 500mg/24H - Moderate
		Skin: adverse effect observed (irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]

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

SODIUM LINEAR-(C12-14)ALKYL ETHER SULFATE	Alcohol ethoxysulfates (AES) are of low acute toxicity. Neat AES are irritant to the skin and eyes.
D-GLUCOSE, DECYL OCTYL ETHERS, OLIGOMERIC	At very high concentrations, alkyl glycosides are considered irritant, with the risk of serious damage to the eyes. However, it does not irritate the skin.
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.

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	There are suggestions that the negative consequences of trans fat consumption go beyond the cardiovascular risk. In general, there is much less scientific consensus asserting that eating trans fat specifically increases the risk of other chronic health problems: It has been suggested that the intake of both trans fats and saturated fats promote the development of Alzheimer disease, although not confirmed in an animal model. It has been found that trans fats impaired memory and learning in middle-age rats. The rats' brains of trans-fat eaters had fewer proteins critical to healthy neurological function. Inflammation in and around the hippocampus, the part of the brain responsible for learning and memory. These are the exact types of changes normally seen at the onset of Alzheimer's, but seen after six weeks, even though the rats were still young. There is a growing concern that the risk of type 2 diabetes increases with trans fat consumption.[52] However, consensus has not been reached. For example, one study found that risk is higher for those in the highest quartile of trans fat consumption. Another study has found no diabetes risk once other factors such as total fat intake and BMI were accounted for. Research indicates that trans fat may increase weight gain and abdominal fat, despite a similar caloric intake. A 6-year experiment revealed that monkeys fed a trans fat diet gained 7.2% of their body weight, as compared to 1.8% for monkeys on a mono-unsaturated fat diet. Although obesity is frequently linked to trans fat in the popular media, this is generally in the context of eating too many calories; there is not a strong scientific consensus connecting trans fat and obesity, although the 6-year experiment did find such a link, concluding that "under controlled feeding conditions, long-term TFA consumption was an independent factor in weight gain. TFAs enhanced intra-abdominal deposition of fat, even in the absence of caloric excess, and were associated with insulin resistance, with evidence that there is impaired post-insulin receptor binding signal transduction. Liver Dysfunction: Trans fats are metabolised differently by the liver than other fats and interfere with delta 6 desaturase. Delta 6 desaturase is an enzyme involved in converting essential fatty acids to arachidonic acid and prostaglandins, both of which are important to the functioning of cells. Infertility in women: One 2007 study found, "Each 2% increase in the intake of energy from trans unsaturated fats, as opposed to that from carbohydrates, was associated with a 73% greater risk of ovulatory infertility...". Major depressive disorder: Spanish researchers analysed the diets of 12,059 people over six years and found those who ate the most trans fats had a 48 per cent higher risk of depression than those who did not eat trans fats. One mechanism may be trans-fats' substitution for docosahexaenoic acid (DHA) levels in the orbitofrontal cortex (OFC). Very high intake of trans-fatty acids (43% of total fat) in mice from 2 to 16 months of age was associated with lowered DHA levels in the brain (p=0.001) When the brains of 15 major depressive subjects who had committed suicide were examined post-mortem and compared against 27 age-matched controls, the suicidal brains were found to have 16% less (male average) to 32% (female average) less DHA in the OFC. The OFC is known to control reward, reward expectation and empathy, which are all negatively impacted in depressive mood disorders, as well as regulating the limbic system>
D-LIMONENE	Tumorigenic by RTECS criteria The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
ALPHA-TERPINEOL	A member or analogue of a group of aliphatic and alicyclic terpenoid tertiary alcohols and structurally related substances generally regarded as safe. Animal testing suggests that the acute toxicity of tertiary alcohols and related esters is extremely low. Genetic toxicity: Tests on bacterial and animal cells showed no evidence of genetic toxicity or potential to cause mutations.
ALPHA-HEXYLCINNAMALDEHYDE	Animal testing suggests that the toxicity through swallowing cinnamyl aldehyde derivatives is very low. The potential for toxicity through skin exposure is similarly low. Cinnamaldehyde and its alkyl-substituted derivatives do not directly cause mutations or genetic damage. However, animal testing suggests that they may result in poor development of the skull and kidney in the foetus. 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.
BETA-CITRONELLOL	The mechanisms associated with common adverse reactions to certain cytokine-chemokine inhibitors include dose-related toxicity that occurs in a certain proportion of patients. When a critical plasma concentration or total dose is exceeded, toxicity that is unpredictable and mediated through allergic or idiosyncratic mechanisms. Certain classes of inhibitors are associated with the dose-related toxicity ;while, in contrast, the major toxicity of others is due to allergic reactions. These differences are explained in part by the relative ability of specific compounds to inhibit enzymatic pathways in the host versus their stimulation of specific immune responses. Since chemokines play a central role in regulating the immune system, and in particular, the trafficking of leukocytes, inhibiting their action may represent a powerful new therapeutic strategy for treating diseases with an inflammatory component. However, this potential will only be realized if it is possible to interfere with chemokine signaling networks, without inducing unacceptable side effects. Although very little, direct human toxicology has been carried out using chemokine inhibitors, there is now a sufficient body of indirect and circumstantial evidence (for example, from genetically modified mice and from animal model studies using chemokine inhibitors) to allow a tentative assessment of the biological impact of chemokine inhibition. The tentative conclusion is that anti-inflammatory therapy achieved through chemokine inhibition may have fewer side effects than originally expected, even when the actions of multiple chemokines are inhibited simultaneously. Being chemokines and chemokine receptors expressed by both tumor cells and leukocyte infiltrate they represent an ideal target for immunotherapy. However, better understanding of their roles in different malignancies is still necessary to avoid potential side effects. In hematological malignancies targeting of overexpressed chemokine receptors directly kill tumor cells but can potentially induce unwanted immune reactions (e.g., CCR4). In the context of solid tumors, chemokine receptor inhibitors are giving encouraging results when used in combination with chemotherapy or with antibodies against immune checkpoints. For this reason, it is possible to envisage that chemokine receptor inhibitors will be used in the future to modulate the stromal component, to overcome chemotherapy resistance and to optimize the immune response of the patients. Inhibition of NF-kB in vivo can be detrimental. NF-kB controls multiple functions in homeostasis including a functional immune response, cell cycle, and cell death. Genetic studies in mice and analysis of naturally occurring mutations in humans point to specific developmental and immune consequences due to altering NF-kB activity. The same functions that make NF-kB attractive for developing inhibitors for treating disease also play a role in homeostasis, and disruption of the NF-kB pathway during development or in adults leads to unfavorable and potentially unhealthy consequences. NF-kB plays a role in multiple homeostatic cellular processes including response to stimuli, cell proliferation, and death, regulating communication between cells, but is also tightly linked with other signaling pathways within the cell, such as p38 and JNK. In addition to mediating proinflammatory responses, NF-kB may regulate apoptotic and cell cycle changes induced by cellular stress, DNA damage or oncogenes by communication with the tumor suppressor p53. Disruption of normal cellular responses by inhibiting NF-kB can have adverse consequences such as immune suppression and tissue damage. Understanding the consequences of lack of NF-kB activity in adult humans comes from observation of naturally occurring genetic deficiencies in this pathway. Mutations have been discovered in humans in signaling molecules upstream of NF-kB resulting in defects in development or immunity. Genetic defects have also been discovered in genes that immediately affect NF-kB activation including IKK gamma (NEMO), a subunit of the IKK complex, and Ikbalpha. The IKK gamma mutations result in a defective IKK complex and the Ikbalpha mutation results in an Ikbalpha protein that cannot be phosphorylated and degraded. Both genetic defects result in suppressed NF-kB activation and ectodermal dysplasia with immunodeficiency. In general patients with these genetic defects have multiple immunological defects including impaired innate immunity, impaired antibody production, and ultimately severe bacterial infections. Understanding the immune defects and susceptibilities in patients with genetic defects in the NF-kB pathway will help prepare for potential adverse effects of pharmacologic NF-kB inhibitors The requirement for NF-kB in the development and maintenance of the immune system is well documented. NF-kB is required for survival during fetal development and for normal lymphocyte generation in adult mice. Removal of the p65 (RelA) subunit of NF-kB or the IKKbeta gene results in death during fetal development primarily due to massive liver apoptosis Fetal liver stem cells from p65 or IKKbeta deficient mice have been transplanted into irradiated hosts revealing a specific requirement of NF-kB for T-cells, B-cells, and common lymphoid progenitor development but not for myeloid cells or stem cells. The failure to produce lymphocytes is mediated through hypersensitivity to TNF due to lack of NF-kB activity. Lymphocyte depletion with chemical or genetic inhibition of NF-kB have implications for therapeutic potential use in humans. The double-sided nature of NF-kB inhibition is clear in this

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instance where chemical inhibition in vivo mimics genetic experiments inducing rapid TNF-dependent apoptosis. Rapid induction of apoptosis may be an advantage for treating some forms of cancer, but at the same time cause depletion of some lymphocyte populations. In addition to controlling lymphocyte development, NF- κ B plays a major role in both adaptive and innate immunity. Various signaling pathways responding to receptor recognition of immune challenge converge on NF- κ B which then regulates genes that control the immune response. Both T-cell receptor and B-cell receptors activate NF- κ B through phosphorylation of CARMA1 by PKC θ and PKC β respectively, resulting in recruitment and activation of IKK and ultimately expression of genes that control cellular activation, proliferation, and survival. In addition, NF- κ B plays a role in T-cell response to costimulatory signals. Cells respond to pathogenic microorganisms in part through recognition by Toll-like receptors (TLRs). TLR-family members recognize different molecular structures present in microbes and respond by activating signaling pathways including NF- κ B leading to expression of anti-microbial effector molecules, as well as molecules that help in development of the adaptive immune response. Inhibition of NF- κ B during TLR stimulation can lead to macrophage apoptosis, a mechanism used by some pathogens to help evade immune response. NF- κ B is clearly required for normal mature B-cell and T-cell maintenance and function, including regulatory, memory, and natural killer-like T cells. Inhibition of NF- κ B activation in lymphocytes results in defects in growth, survival, and cytokine production and blocks multiple steps in germinal center formation. Given the diverse roles NF- κ B plays in immune response to pathogens it is not surprising to find mice genetically deficient in components of the NF- κ B pathway are susceptible to parasitic and bacterial infection.

The role of NF- κ B in inhibition of apoptosis is one of the factors that make it a potential target for cancer therapy. NF- κ B deficient mice die during embryogenesis in part due to TNF-mediated liver damage. Adult mice with impaired NF- κ B targeted to the liver have normal liver function, but have severe liver damage after challenge with concanavalin A, a pan-T cell activator. Liver damage occurs due to sustained activation of JNK due to accumulation of reactive oxygen species (ROS) in the absence of normal NF- κ B activation.

In vitro studies suggested that TNF inhibitors selectively decrease pro-inflammatory cytokines while preserving both the humoral and cell-mediated arms of the immune response. However, a number of relevant safety issues regarding the use of TNF inhibitors have emerged in post-marketing pharmacovigilance assessments. Adverse events associated with TNF inhibitors can be broadly classified as target/class-related or agent-related. Target-related adverse events include those potentially attributable to the immunosuppression inherent in blocking a key component of the immune system, such as an inflammatory cytokine that may lead to increased susceptibility to infections and malignancies. In addition, specific inhibition of TNF can predispose patients to increased susceptibility to tuberculosis (TB), autoantibody production, hepatotoxicity, demyelinating disease, and clinical worsening CHF.

Agent-related adverse events such as allergic reactions and antigenicity are idiosyncratic reactions that relate to the particular agent used. Safety data from clinical trials and registries have shown a small but consistent increase in infections among TNF inhibitor treated patients. However, the risk of infection with TNF inhibitors increased significantly when TNF inhibitors were combined with other biologic agents.

Another potential sequel of immunosuppression is malignancy. With a few notable exceptions, the bulk of data do not support an increased risk of solid tumors related to TNF inhibitor therapy. However, greater numbers of hematological malignancies, particularly non-Hodgkin's lymphoma, have been observed in some registries.

Inhibition of TNF may predispose patients to a variety of untoward effects that seem to be specific to inhibition of the TNF molecule. There is a fair amount of animal and ex vivo data supporting the important role played by TNF in controlling tuberculosis (TB). In contrast to typical presentation of acute TB as pneumonia, about half of the cases of TB related to TNF inhibitors have presented as extra-pulmonary or disseminated TB. The majority of these TB cases appear to be reactivation of latent TB, with infection occurring within the first few months of therapy; however, newly acquired cases have also been described.

Treatment with TNF inhibitors has also been associated with development of autoantibodies. While the mechanism of this is unknown, it does not appear to result from inhibition of TNF itself, but possibly through induction of apoptosis. The autoantibodies typically generated include antinuclear antibody (ANA; which develops in about half of RA patients treated with TNF inhibitors), antibodies to double-stranded DNA (anti-dsDNA; which develops in approximately 10-15% of patients treated with TNF inhibitors), and anticardiolipin antibodies. Although rare, progression to a lupus-like illness can occur in patients treated with TNF inhibitors. Also, mild to moderate liver function tests (LFTs) elevation (generally < 3 times the upper limit of normal) has been observed with TNF inhibitors. Many of these cases were confounded by concomitant use of potentially hepatotoxic drugs and underlying medical conditions. However, in light of liver failure of unidentifiable etiology in several cases, clinicians should be aware of these rare events and consider monitoring LFTs.

Several anti-TNF drugs are commonly prescribed by a number of autoimmune conditions. Some of them have been reported to produce a CNS-demyelination compatible with, and by current knowledge indistinguishable from, standard MS.

Several cases of multiple sclerosis (MS) and other demyelinating conditions have been identified among patients treated with TNF inhibitors, although the true impact of TNF inhibitors on the development of MS remains undefined.

Multiple adverse effects of TNF- α inhibition have been identified through both clinical trials and post-marketing surveillance. These include:

- Injection site reactions
- Infusion reactions
- Neutropenia
- Infections

The important side effects of TNF inhibitors include lymphomas, infections (especially reactivation of latent tuberculosis), congestive heart failure, demyelinating disease, a lupus-like syndrome, induction of auto-antibodies, injection site reactions, and systemic side effects.

The U.S. Food and Drug Administration continues to receive reports of a rare cancer of white blood cells (known as hepatosplenic T-cell lymphoma or HSTCL), primarily in adolescents and young adults being treated for Crohn's disease and ulcerative colitis with TNF blockers.

TNF inhibitors put patients at increased risk of certain opportunistic infections. The FDA has warned about the risk of infection from two bacterial pathogens, *Legionella* and *Listeria*. People taking TNF blockers are at increased risk for developing serious infections that may lead to hospitalization or death due to certain bacterial, mycobacterial, fungal, viral, and parasitic opportunistic pathogens.

Patients on TNF inhibitors are at increased risk of opportunistic fungal infections such as pulmonary and disseminated histoplasmosis, coccidioidomycosis, and blastomycosis.

Most TNF inhibitors are not actively transported through the placenta until 15 weeks' gestation, when elevated levels in cord blood can be found. A concerning report in 2009 described a clustering of congenital anomalies suggestive of VACTERL syndrome (vertebral anomalies, anal atresia, cardiac defects, tracheoesophageal fistula, esophageal atresia, renal anomalies, and limb dysplasia) in infants exposed to TNF inhibitors in utero. Although subsequent analysis did not support an association, this report informed decision-making for many rheumatologists who avoided prescribing these medications during pregnancy.

Citronellol, geraniol, nerol, and geranyl acetate are currently generally regarded as safe by the US FDA for their intended use as flavouring substances. They are ubiquitous in the plant kingdom. Terpenoid alcohol, formed in the gastrointestinal tract, as a result of hydrolysis, is rapidly absorbed, metabolised and excreted via the urine. It has no repeat dose effect, no genetic and cancer causing effect but may harm the unborn child of a pregnant woman.

tyrosine kinase inhibitors (TKIs) have gained prominence as the most effective pathway-directed anti-cancer agents. TKIs have shown significant utility in the treatment of multiple hematological malignancies and solid tumors, including chronic myelogenous leukemia, non-small cell lung cancers, gastrointestinal stromal tumors, and HER2-positive breast cancers. Given their widespread applications, an increasing frequency of TKI-induced adverse effects has been reported. Although TKIs are known to affect multiple organs in the body including the lungs, liver, gastrointestinal tract, kidneys, thyroid, blood, and skin, cardiac involvement accounts for some of the most serious complications. The most frequently reported cardiovascular side effects range from hypertension, atrial fibrillation, reduced cardiac function, and heart failure to sudden death.

Although tyrosine kinases have been more successfully targeted pharmacologically. STKs are part of the cell machinery that is involved in signal transduction pathways controlling metabolism, cell division, angiogenesis and other functions. STKs are hyperactivated across multiple malignancies including ovarian cancer, making them attractive therapeutic targets.

tyrosine kinase inhibitors treat a wide range of cancers and cause an equally wide range of side effects. For example, one group of tyrosine kinase inhibitors targets cancers such as non-small lung cancer, breast, colorectal and pancreatic cancer. Those treatment side effects may range from mild skin rashes to life-threatening skin issues like Steven-Johnson syndrome/toxic epidermal necrosis. Another group of TKIs that target non-small lung cancer and colorectal cancer may cause cardiovascular issues like high blood pressure (hypertension) and proteinuria.

Side effects vary based on the specific TKI and may include:

Fatigue.

Fever.

Weight loss/weight gain. Small molecule kinase inhibitors often block multiple enzymes meaning there is some cross over in the actions and side effects of these drugs.

The most common side effects are:

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Nonspecific rashes
Periocular oedema
Acral erythema
Splinter haemorrhage
Pigmentation changes
Photosensitivity

For G-protein inhibitors:/ antagonists/ modulators.

G protein-coupled receptors (GPCRs) are essential cell membrane signaling molecules and represent the most important class of drug targets. Some signaling pathways downstream of a GPCR may be responsible for drug adverse effects, while others mediate therapeutic efficacy. Biased ligands preferentially activate only a subset of all GPCR signaling pathways. They hold great potential to become next-generation GPCR drugs with less side effects due to their potential to exclusively activate desired signaling pathways.

GPCR ligands include odorants, tastants, and neurotransmitters, and vary in size and properties. Dramatic chemical diversity may occur even among ligands of the same receptor. Chemical variability of antagonists significantly correlates with the binding site hydrophobicity and anti-correlates with the number of hydrogen bond donors in the binding site. The number of disulfide bridges in the extracellular region of a receptor anti-correlates with the range of molecular weights of its antagonists, highlighting the role of the entrance pathway in determining the size selectivity for GPCR antagonists.

The number of protein targets included in the cross-pharmacology profile of the different GPCRs changes significantly upon varying the ligand similarity and binding affinity criteria. However, with the exception of muscarinic receptors, aminergic GPCRs distinguish themselves from the rest of the members in the family by their remarkably high levels of pharmacological similarity among them.

GPCRs are classified under the GRAFS system (Metabotropic Glutamate, Rhodopsin, Adhesion, Frizzled/taste2/Smoothed and Secretin), with therapies having been developed for about 30 GPCRs from the glutamate, rhodopsin and secretin families.

GPCR signaling requires significant conformational changes within the trans-membrane TM domain, triggered by agonist binding, and is often coupled to interactions from the extracellular domains or loops. It is becoming clear that many binding sites and mechanisms exist for positive and negative allosteric regulation, and for biased signaling pathways, likely in greater numbers than seen in most other protein systems.

When GPCRs are exposed to a neutral agonist, such as morphine on mu-opioid receptor, an occupied receptor can generate several signal waves (non-biased agonist). In GPCR signaling, the ability of a molecule to selectively activate one pathway without affecting another pathway is called biased agonism. Biased signaling occurs at different signaling proteins, including G proteins, GRKs, beta-arrestins, and even at levels of the allosteric binding site. Since GPCR activation-induced two distinct signal waves, G protein-dependent signaling followed by beta-arrestin-dependent signaling opens a new promising therapeutic future in the world of GPCRs. This is true since discovering such molecules dramatically lowers the adverse effects by turning off unwanted signals. For example, the analgesic effect of morphine (neutral agonist) through the activation of u-receptors is accompanied by several side effects, including constipation, respiratory depression, tolerance, nausea, and sedation.

Despite the long history and obvious desirability of developing drugs targeting GPCRs, there are several problems associated with their development. For example, the muscarinic M1 receptor is a well-validated target for agonists that could alleviate cognitive decline during neurodegeneration.

Muscarinic acetylcholine receptors (MRs, or mAChRs), which are more sensitive to muscarine than to nicotine, are a group of class A GPCRs comprising five distinct subtypes, named as muscarinic M1, M2, M3, M4, and M5 receptors (M1R-M5R). M1R, M3R, and M5R are coupled to the Gq/11 family of G proteins, whereas M2R and M4R are coupled to the Gi/o family of G proteins.

However, the orthosteric binding site of M1 is virtually identical to those of the related receptors M2, M3, M4, and M5 as they all bind the native ligand acetylcholine, and activation of M2 and M3 in particular gives rise to dose-limiting side effects (gastrointestinal [GI] disturbances, cardiovascular effects).

Atropine and other anticholinergic agents exert their bronchodilator effects through the blockade of MRs in the airways. As a tertiary ammonium derivative, atropine is a nonselective antagonist with similar affinity for all of the MR subtypes. The half-life of atropine for M3R residence is 3.5 hours. Although extensively used in the past, atropine is rarely used at the present time because it is well absorbed into the systemic circulation and penetrates the blood-brain barrier, leading to multiple systemic side effects, including tachycardia.

Several long-acting muscarinic antagonists (LAMAs) are under investigation or are available for the treatment of obstructive airway diseases.

LAMAs are considered to be safe drugs at recommended dosages. However, because MRs are expressed not only in the lungs, but also in the heart and the digestive and urinary tracts, the blockade of different MR subtypes in these organs by LAMA treatment can cause diverse, unwanted physiologic effects. For example, these agents can initially block prejunctional M2R on cholinergic airway nerves that normally reduce the release of the bronchoconstricting neurotransmitter acetylcholine, thus resulting in cough and paradoxical bronchoconstriction.

Side effects including cardiovascular morbidity and mortality of inhaled LAMA agents in asthma need to be further studied and defined.

Another potential source of side effects when targeting other receptors could arise due to signaling through multiple different pathways.

There are multiple signaling pathways for GPCRs, and it is sometimes possible to bias the signaling of a given GPCR through either a specific G protein or through beta arrestin which could reduce the side effects of some drugs.

Targeting G protein alpha-subunits has the potential for pleiotropic effects and could result in multiple side effects.

Particular targets of concern include ion channels such as the G protein-activated inward rectifier K⁺ channel (GIRK) and the N-type voltage-gated calcium channels. Gbeta-gamma activates GIRK channels in neurons and in atria, leading to a hyperpolarization-induced decrease in action potential firing. Therefore, when considering the use of Gbeta-gamma inhibitors in cardiac or immune therapy, interfering with the regulation of action potentials would have highly undesirable side effects, such as arrhythmias. However, empirical data using prototypical Gbeta-gamma blockers indicate that these pathways are unaffected by Gbeta-gamma inhibitors, and animals treated with gallein show no signs of arrhythmias or alterations in heart rate.

CITRAL

- Produces maternal effects (oogenesis, ovaries, fallopian tube changes) and effects live-birth index.

The terpenoid hydrocarbons are found in needle trees and deciduous plants. This category of chemicals shows very low acute toxicity. They are excreted in the urine. They are unlikely to cause genetic damage, but animal testing shows that they do cause increased rates of kidney cancer. They have low potential to cause reproductive and developmental toxicity.

A member or analogue of a group of aliphatic, linear alpha,beta-unsaturated aldehydes and structurally related substances

These substances are generally regarded as safe. They are found in flavouring substances in food and are rapidly absorbed and broken down in the body.

for citral

Citral is rapidly absorbed from the gastrointestinal tract. Much of an applied dermal dose is lost due to its extreme volatility, but the citral remaining on the skin was fairly well absorbed. Citral is rapidly metabolised and excreted as metabolites. Urine is the major route of elimination.

Acute toxicity of this chemical is low in rodents because the oral or dermal LD50 values were more than 1000 mg/kg. This chemical is irritating to skin and not irritating to eyes in rabbits. There is some evidence that this chemical is a human skin sensitiser.

Repeat dose toxicity: Several repeated dose oral studies show no adverse effect of citral at less than 1,000 mg/kg/day exposure and some histological changes in the nasal cavity or forestomach, the first exposure sites, probably due to irritation, at more than 1,000 mg/kg/day.

Male and female F344/N rats received microencapsulated citral in feed at concentrations of 0, 0.63, 1.25, 2.5, 5 and 10% (resultant doses: 0, 142, 285, 570, 1,140 and 2,280 mg/kg/day) for 14 days. Minimal to mild hyperplasia and/or squamous metaplasia of the respiratory epithelium was observed in nasal cavity without inflammatory response at 1,140 and 2,280 mg/kg/day of both sexes. The NOAEL was established at 570 mg/kg/day. In an OECD preliminary reproduction toxicity screening test [TG 421], citral was administered to Crlj:CD (SD) rats by gavage at doses of 0, 40, 200 and 1,000 mg/kg/day in males for 46 days and in females for 39-50 days including before and through mating and gestation periods and until day 3 of lactation. Squamous hyperplasia, ulcer and granulation in lamina propria were observed in the forestomach at 1,000 mg/kg/day of both sexes. Therefore, the NOAEL for repeated dose toxicity was 200 mg/kg/day for both sexes.

Developmental toxicity: in the above preliminary reproductive study, no effects were detected in reproductive ability, organ weights or histopathology of the reproductive organs of both sexes, and delivery or maternal behavior. However, body weights of male and female pups were reduced in the 1000 mg/kg group. Therefore, an oral NOAEL for developmental toxicity was 200 mg/kg/day.

In a teratogenicity study, SD pregnant rats were exposed to citral by inhalation for 6 hr/day on gestation days 6-15 at mean concentration of 0, 10 or 34 ppm as vapour, or 68 ppm as an aerosol/vapour mixture. Even in the presence of the maternal effects, no significant teratogenicity was noted at 68 ppm. An inhalation NOAEL of teratogenicity was established at 68 ppm (423 mg/m3).

	Genotoxicity: Seven bacterial reverse mutation studies indicate negative results with and without metabolic activation. As for non-bacterial in vitro study, two chromosomal aberration results in Chinese hamster cells are negative however one positive result in sister chromatid exchange is given in the same cells. Additionally, two in vivo micronucleus tests in rodents indicate negative results. Based on the above information, the genotoxic potential of citral can be considered to be negative. Carcinogenicity: A NTP study shows that there was no evidence of carcinogenic activity in male/female rats and male mice but some evidence of malignant lymphoma in female mice (up to 4,000 ppm in feed in rats and up to 2,000 ppm in feed in mice). Dermal application of citral induces prostate hyperplasia with low severity only in some strains of rats. However, the NTP oral carcinogenicity studies in rats and mice found no evidence of lesions (neoplastic or non-neoplastic) in any male reproductive organ, including the prostate. The health significance of the effects seen in the dermal studies in rats is uncertain due to dramatic strain differences and it is noted that the work has primarily been performed in a single laboratory. For dienaldehydes: Dienaldehydes are by-products of peroxidation of polyunsaturated lipids and commonly found in many foods or food-products. Both National Cancer Institute (NCI) and NTP have expressed great concern on the potential genotoxicity and carcinogenicity of dienaldehydes. 2,4-Decadienal and 2,4-hexadienal are autooxidation products of polyunsaturated fatty acids Several researchers have implied there could be a link between exposures to lipid peroxidation products in the diet and the development of human cancers. Lipid hydroperoxides have been shown to give rise to low intracellular levels of 2,4-decadienal and other alpha-beta-unsaturated aldehydes that are known to be reactive with DNA. Ingested lipid oxidation products and oxidized fats have been reported to cause increased excretion of mutagens, cellular injury to liver and kidneys, increased cell proliferation in the gastrointestinal tract, and other nonspecific tissue injury and irritation effects resulting from induced oxidative stress. Treatment related changes following gastric lavage administration for up to 3 months were similar for 2,4-hexadienal and 2,4-decadienal, and in both cases the forestomach and nose were identified as target organs. In two week studies of 2,4-hexadienal and 2,3 decadienal in rats and mice, forestomach lesions included necrosis and ulceration; epithelial hyperplasia was observed in rats and mice in the 2,4-hexadienal studies. In the 3-month studies of 2,4-hexadienal and 2,4-decadienal, forestomach epithelial hyperplasia and degeneration with or without chronic active inflammation occurred in addition to nasal olfactory epithelia atrophy and necrosis. Carcinogenicity and mutagenicity data from testing of dienals are limited. In the two year carcinogenicity studies, 2,4-hexadienal induced significantly increased incidences of forestomach neoplasms in rats and mice. NTP Technical Report 2,4-decadienal Trans, trans-2,4-decadienal (tt-DDE or 2,4-De), a specific type of dienaldehyde, is abundant in heated oils and has been associated with lung adenocarcinoma development in women due to their exposure to oil fumes during cooking. Cultured human bronchial epithelial cells (BEAS-2B cells) were exposed to 0.1 or 1.0 uM tt-DDE for 45 days, and oxidative stress, reactive oxygen species (ROS) production, GSH/GSSG ratio, cell proliferation, and expression of TNFalpha and IL-1beta were measured. The results show that tt-DDE induced oxidative stress, an increase in ROS production, and a decrease in GSH/GSSG ratio (glutathione status) in a dose-dependent manner. Treatment of BEAS-2B cells with 1.0 uM tt-DDE for 45 days increased cell proliferation and the expression and release of pro-inflammatory cytokines TNFalpha and IL-1beta. Cotreatment of BEAS-2B cells with antioxidant N-acetylcysteine prevented tt-DDE-induced cell proliferation and release of cytokines. Therefore, these results suggest that tt-DDE-induced changes may be due to increased ROS production and enhanced oxidative stress. Since increased cell proliferation and the release of TNF-alpha and IL-1beta are believed to be involved in tumor promotion, these results suggest that tt-DDE may play a role in cancer promotion. Previous studies on dienaldehydes have focused on their genotoxic or carcinogenic effects in the gastrointestinal tract; the present study suggests a potential new role of tt-DDE as a tumor promoter in human lung epithelial cells. Trans, Trans-2,4-Decadienal, a Product Found in Cooking Oil Fumes, Induces Cell Proliferation and Cytokine Production Due to Reactive Oxygen Species in Human Bronchial Epithelial Cells Louis W. Chang Wai-Sze Lo Pinpin Lin Toxicological Sciences, Volume 87, Issue 2, 1 October 2005, Pages 337–343, https://doi.org/10.1093/toxsci/kfi258 2,4-Decadienal is produced by the oxidation of linoleic acid. 2,4-Decadienal is found as a contaminant in water. It is generated from polyunsaturated fatty acids by the action of plant lipoxygenases and is produced in mammalian tissues in certain physiological and pathophysiological processes such as lipid peroxidation, arachidonic acid oxidation, and reactions with reactive oxygen species Alpha,beta-unsaturated aldehydes and ketones are potentially genotoxic. It is believed that nucleophilic sites in DNA react through a 1,4-nucleophilic addition (Michael reaction) with alpha,beta-unsaturated carbonyl compounds. The flavour industry provided genotoxicity studies for the substance 4,5-epoxydec-2(trans)-enal. Based on these data, a European Food Safety Authority (EFSA Panel concluded that 4,5-epoxydec-2(trans)-enal did not induce gene mutations in bacterial cells but was positive in an in vitro micronucleus assay, so, 4,5-epoxydec-2(trans)-enal is considered an in vitro genotoxic agent. The negative results obtained in an in vivo micronucleus assay cannot overrule the positive results of the in vitro micronucleus assay with and without S9-mix due to the lack of demonstration of bone marrow exposure. Following this, the flavour industry has provided plasma analysis of a satellite group of rats treated with 4,5-epoxydec-2(trans)-enal in order to investigate the systemic exposure of animals in the in vivo micronucleus assay. However, the plasma analysis did not provide enough evidence of target tissue exposure. An in vivo Comet assay in rodents was recommended in order to investigate possible genotoxic effects at the first site of contact (e.g. stomach/duodenum cells) and in the liver. An in vivo Comet assay in liver and duodenum was provided that suggests that 4,5-epoxydec-2(trans)-enal did not induce DNA damage in the duodenum of rats. However, the genotoxic effect observed in vitro was confirmed in the in vivo Comet assay in the liver of rats. The Panel concluded that 4,5-epoxydec-2(trans)-enal does raise a safety concern with respect to genotoxicity Scientific Opinion on Flavouring Group Evaluation 226 Revision 1 (FGE.226Rev1): consideration of genotoxicity data on one alpha,beta-unsaturated aldehyde from chemical subgroup 1.1.1(b) of FGE.19; May 2017 https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2017.4847
P-CYMENE	For aromatic terpenes: p-cymene and cumene have low toxic potential and are excreted in the urine. At very high doses in animal testing, inco-ordination, damage to the kidneys and lung inflammation, with decrease in thymus weight, occurred. This group of substances does not seem to cause cancer, genetic damage or developmental toxicity and has low potential for reproductive toxicity.
ISOTHIAZOLINONES, MIXED	In light of potential adverse effects, and to ensure a harmonised risk assessment and management, the EU regulatory framework for biocides has been established with the objective of ensuring a high level of protection of human and animal health and the environment. To this aim, it is required that risk assessment of biocidal products is carried out before they can be placed on the market. A central element in the risk assessment of the biocidal products are the utilization instructions that defines the dosage, application method and amount of applications and thus the exposure of humans and the environment to the biocidal substance. Humans may be exposed to biocidal products in different ways in both occupational and domestic settings. Many biocidal products are intended for industrial sectors or professional uses only, whereas other biocidal products are commonly available for private use by non-professional users. In addition, potential exposure of non-users of biocidal products (i.e. the general public) may occur indirectly via the environment, for example through drinking water, the food chain, as well as through atmospheric and residential exposure. Particular attention should be paid to the exposure of vulnerable sub-populations, such as the elderly, pregnant women, and children. Also pets and other domestic animals can be exposed indirectly following the application of biocidal products. Furthermore, exposure to biocides may vary in terms of route (inhalation, dermal contact, and ingestion) and pathway (food, drinking water, residential, occupational) of exposure, level, frequency and duration. Formaldehyde generators (releasers) are often used as preservatives. The maximum authorised concentration of free formaldehyde is 0.2% and must be labelled with the warning sign "contains formaldehyde" where the concentration exceeds 0.05%. The use of formaldehyde-releasing preservatives ensures that the level of free formaldehyde in the products is always low but sufficient to inhibit microbial growth - it disrupts metabolism to cause death of the organism. However there is a concern that formaldehyde generators can produce amines capable of causing cancers (nitrosamines) when used in formulations containing amines.
LEMON 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.

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	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. Cross-reactivity is also expected between ester derivatives and their parent alcohols, as the esters will be broken down by esterases in the skin. Esters of important contact allergens that can be activated by hydrolysis in the skin are isoeugenol acetate, eugenyl acetate and geranyl acetate all of which are known to be used as fragrance ingredients. Bicyclic terpenes are very low in acute toxicity. However, repeated dosing may have deleterious effects on the liver and kidney. Members of this category show no significant reproductive or developmental toxicity and may have a little, if any, potential to alter genetic material.
C.I. ACID YELLOW 23	Suspected allergen *[Hawley's] Allergic reactions involving the respiratory tract are usually due to interactions between IgE antibodies and allergens and occur rapidly. Allergic potential of the allergen and period of exposure often determine the severity of symptoms. Some people may be genetically more prone than others, and exposure to other irritants may aggravate symptoms. Allergy causing activity is due to interactions with proteins. Attention should be paid to atopic diathesis, characterised by increased susceptibility to nasal inflammation, asthma and eczema. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.
C.I. ACID YELLOW 3	Studies done on animals showed that quinoline and its metabolites are almost completely excreted via the urine but caused fatty liver and bile duct proliferation in some cases. There is no evidence to suggest that quinoline causes cancer in humans, but it is reported to cause liver cancer in rats and skin cancer in mice. As such, it is predicted to be a cancer causing agent. It is equally said to cause no toxic effect on human and animal gene but some of its metabolites may cause abnormal gene alterations (4-methyl isomer and 2-methyl isomer).
C.I. FOOD YELLOW 3	Acceptable daily intake as food additive = 2.5 mg/kg FAO/WHO 1982
CITRAL DIETHYL ACETAL	For acetals: Aliphatic acetals are reported to have little acute toxicity if given by mouth. They are generally broken down to their corresponding aldehydes and alcohols within several hours in the stomach and bowel; in the liver, they are expected, in humans, to be converted to alcohols and acids. There is not enough data to rule out significant amounts of the original acetals reaching the general circulation. The component alcohols and aldehydes are divided into linear, alpha,beta-unsaturated and branched chain structural types which differ in their breakdown process. The first two types are generally oxidised to carboxylic acids; branched-chain aliphatic aldehydes have been reported to be mainly oxidised to more polar breakdown products which are mainly excreted in the urine as a mixture of diacids and hydroxyacids. 32mutagen
SODIUM LINEAR-(C12-14)ALKYL ETHER SULFATE & CITRAL & ISOTHIAZOLINONES, MIXED & LEMON OIL	No significant acute toxicological data identified in literature search.
SODIUM LINEAR-(C12-14)ALKYL ETHER SULFATE & CASTOR OIL, HYDROGENATED, ETHOXYLATED	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 sensitizers. The oxidation products also cause irritation.
D-GLUCOSE, DECYL OCTYL ETHERS, OLIGOMERIC & D-LIMONENE & ALPHA-TERPINEOL & ALPHA-HEXYLCINNAMALDEHYDE & BETA-CITRONELLOL & CITRAL & ISOTHIAZOLINONES, MIXED & LEMON OIL & C.I. ACID YELLOW 23 & CITRAL DIETHYL ACETAL	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.
D-GLUCOSE, DECYL OCTYL ETHERS, OLIGOMERIC & LEMON OIL	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
D-GLUCOSE, DECYL OCTYL ETHERS, OLIGOMERIC & ETHANOL & DIHYDROMYRCENOL & P-CYMELE & ISOTHIAZOLINONES, MIXED & LEMON OIL & CITRAL DIETHYL ACETAL	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.
D-LIMONENE & BETA-CITRONELLOL & CITRAL & CITRAL DIETHYL ACETAL	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.
D-LIMONENE & ALPHA-TERPINEOL & DIHYDROMYRCENOL & ALPHA-HEXYLCINNAMALDEHYDE & BETA-CITRONELLOL & CITRAL & LEMON OIL	Adverse reactions to fragrances in perfumes and fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, sensitivity to light, immediate contact reactions, and pigmented contact dermatitis. Airborne and connubial contact dermatitis occurs. Contact allergy is a lifelong condition, so symptoms may occur on re-exposure. Allergic contact dermatitis can be severe and widespread, with significant impairment of quality of life and potential consequences for fitness for work. If the perfume contains a sensitizing component, intolerance to perfumes by inhalation may occur. Symptoms may include general unwellness, coughing, phlegm, wheezing, chest tightness, headache, shortness of breath with exertion, acute respiratory illness, hayfever, asthma and other respiratory diseases. Perfumes can induce excess reactivity of the airway without producing allergy or airway obstruction. Breathing through a carbon filter mask had no protective effect. Occupational asthma caused by perfume substances, such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms, even though the exposure is below occupational exposure limits. Prevention of contact sensitization to fragrances is an important objective of public health risk management. Hands: Contact sensitization may be the primary cause of hand eczema or a complication of irritant or atopic hand eczema. However hand eczema is a disease involving many factors, and the clinical significance of fragrance contact allergy in severe, chronic hand eczema may not be clear. Underarm: Skin inflammation of the armpits may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a skin specialist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy. Face: An important manifestation of fragrance allergy from the use of cosmetic products is eczema of the face. In men, after-shave products can cause eczema around the beard area and the adjacent part of the neck. Men using wet shaving as opposed to dry have been shown to have an increased risk of allergic to fragrances. Irritant reactions: Some individual fragrance ingredients, such as citral, are known to be irritant. Fragrances may cause a dose-related contact urticaria (hives) which is not allergic; cinnamal, cinnamic alcohol and Myroxylon pereirae are known to cause hives, but others,

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including menthol, vanillin and benzaldehyde have also been reported.

Pigmentary anomalies: Type IV allergy is responsible for "pigmented cosmetic dermatitis", referring to increased pigmentation on the face and neck. Testing showed a number of fragrance ingredients were associated, including jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol and geranium oil.

Light reactions: Musk ambrette produced a number of allergic reactions mediated by light and was later banned from use in Europe.

Furocoumarins (psoralens) in some plant-derived fragrances have caused phototoxic reactions, with redness. There are now limits for the amount of furocoumarins in fragrances. Phototoxic reactions still occur, but are rare.

General/respiratory: Fragrances are volatile, and therefore, in addition to skin exposure, a perfume also exposes the eyes and the nose / airway. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma. Asthma-like symptoms can be provoked by sensory mechanisms. A significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients and hand eczema.

D-LIMONENE & LEMON OIL

Monomethyltin chloride, thioglycolate esters, and tall oil ester reaction product:

Monomethyltin trichloride (MMTC, CAS RN: 993-16-8), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG); MMT (2-EHMA), CAS RN: 57583-34-3), monomethyltin tris[isooctylmercaptoacetate (MMT(IOTG), CAS RN: 54849-38-6) and methyltin reverse ester tallate reaction product (TERP, CAS RNs: 201687-58-3, 201687-57-2, 68442-12-6, 151436-98-5) are considered one category of compounds for mammalian studies via the oral route. The justification for this category is based on structural similarities and the demonstrated rapid conversion of all of the esters to the MMTC when placed in simulated mammalian gastric contents [0.07M HCl] under physiological conditions. For the MMT(EHTG) >90% conversion to MMTC occurred within 0.5 hours. For TERP, 68% of the monomethyltin portion of the compound was converted to MMTC within 1 hour. Thus, MMTC is the appropriate surrogate for mammalian toxicology studies via the oral route.

TERP is a reaction product of MMTC and dimethyltin dichloride (DMTC), Na₂S, and tall oil fatty acid [a mixture of carboxylic acids, predominantly C-18]. The reaction product is a mixture of carboxylic esters and includes short oligomers of mono/dimethyltins bridged by sulfide groups. Although the tall oil component of TERP is not structurally similar to EHTG, TERP's conversion to MMTC justifies its inclusion. While the contribution of the various ligands to the overall toxicity may vary, the contribution is expected to be small relative to that of the MMTC. Further, the EHTG ligand from MMT(EHTG) is likely to be more toxic than the oleic or linoleic acid from TERP so inclusion of TERP in the category is a rather conservative approach. The other possible degradate of tall oil and EHTG is 2-mercaptoethanol (2-ME), and it is common to both ligands.

Data for MMT(EHTG) and MMT(IOTG) are used interchangeably because they are isomers differing only slightly in the structure of the C-8 alcohol of the mercaptoester ligand. In addition, the breakdown products of MMT(EHTG) and MMT(IOTG) are the thioglycolate esters (EHTG and IOTG), which have the common degradates, thioglycolic acid and C-8 alcohols (either 2-ethylhexanol or isooctanol). EHTG and IOTG also have similar physicochemical and toxicological properties.

The chemistry of the alkyl organotin has been well studied. For organotin, like MMT(EHTG), the alkyl groups are strongly bound to tin and remain bound to tin under most reaction conditions. However, other ligands, such as carboxylates or sulfur based ligands (EHTG), are more labile and are readily replaced under mild reaction conditions. To assess the reactivity of MMT(EHTG) under physiological conditions simulating the mammalian stomach, an in-vitro hydrolysis test was performed. This in vitro test provides chemical information that strongly suggests both the probable in vivo metabolic pathway and the toxicokinetics of the MMT(EHTG) substance. This result verifies that under physiological conditions MMT(EHTG) is rapidly and essentially completely converted to the corresponding monomethyltin chloride, MMTC.

Acute toxicity:

The majority of toxicology studies were conducted with commercial mixtures having high monoalkyltin to dialkyltin ratios.

Gastric hydrolysis studies were conducted with TERP and MMT(EHTG) in which simulated gastric fluid [0.07M HCl under physiological conditions] converted these substances to methyltin chloride and the respective organic acids. Based on data for DMTC and DMT esters the dermal penetration of MMTC and its esters is expected to be low.

Oral:

Acute oral LD50 values for MMTC, MMT(EHTG), MMT(IOTG), and TERP indicated low to moderate toxicity; the most reliable data place the LD50s in the range of 1000 mg/kg.

The acute oral LD50 of MMT(2-EHMA) was 880 mg/kg in rats. Clinical observations included depression, comatose, piloerection, eye squinting, hunched posture, laboured breathing, ataxia, faecal/urine stains, and masticatory movement. No gross pathological changes were reported in surviving animals.

Dermal

Acute dermal LD50 values were =1000 mg/kg bw, and inhalation LC50 was >200 mg/L. MMTC was corrosive to skin and assumed corrosive to eyes.

The acute dermal LD50 of MMT(2-EHMA) in rabbits was 1000 (460 to 2020) mg/kg for females and 2150 (1000 to 4620) mg/kg for males.

There were no deaths at 215 and 464 mg/kg, 0/2 males and 1/2 females died at 1000 mg/kg and 1/2 males and 2/2 females died at 2150 mg/kg. All animals died at 4640 and 10 000 mg/kg. A variety of clinical abnormalities were observed and disappeared in surviving animals by the end of the exposure period. Clinical signs included death, uncoordinated movements, shaking, and hypersensitivity to external stimuli.

Gross necropsy results for animals that died during the study included irritated intestines; blanched stomach; reddened lungs; pale or congested kidneys; and oral, ocular and/or nasal discharges

Inhalation:

The acute inhalation LC50 of MMT(2-EHMA) was 240 mg/L.

The study reported an acute inhalation LC50 of 240 (212 to 271) mg/L in a 1-hr aerosol exposure to male and female rats. The mortality rate was 2/10, 6/10, 9/10 and 10/10 animals at dose levels of 200, 250, 300 and 250 mg/L/hr, respectively. Gross findings included blood in lungs, dark spleen, pale kidneys, fluid in the chest cavity, and heart failure. The slope of the dose-response curve was 1.22 (1.04 to 1.43).

Irritation:

MMT(IOTG)/(EHTG) are irritating to skin, but not to eyes.

Sensitisation:

No data on sensitization are available on MMT(EHTG)/(IOTG), but the hydrolysis products EHTG or IOTG are sensitizers. No primary irritation data were available for TERP, but it was a sensitizer in the mouse Local Lymph Node Assay.

Topical application with 5, 25 and 50 % v/v MMT(2-EHMA) elicited a stimulation index (SI) of 2.13, 7.25 and 9.05, respectively in a local lymph node assay (OECD 429), thus the material is a sensitiser.

Repeat dose toxicity:

There are no repeated-dose studies for the category members via the dermal or inhalation routes.

In a 90-day repeated dose oral study of MMTC, treatment-related changes were limited to the high dose group (750 ppm in diet; 50 mg/kg bw/d with some gender-related variation). Organ weight changes (adrenal, kidney, thymus, spleen, brain, epididymides), haematology, clinical chemistry, and urinalysis changes were noted, but histopathology only confirmed effects in the thymus and brain. The critical toxic effects were neurotoxicity and thymic atrophy. Both sexes had decreased cortex/medulla ratios in the thymus. In the brain there was loss of perikarya of neuronal cells in the pyramidal layer of the Hippocampus CA1/2 in both sexes, and in males there was loss of perikarya in the piriform cortex. The NOAEL was 150 ppm (10 mg/kg bw/d). Another 90-day dietary study using MMTC showed increased relative kidney weights and slight to moderate epithelial hyperplasia of the bladder in females at the lowest dose (NOAEL <20 ppm in diet [$<1-3.6$ mg/kg bw/d]) and additional effects including increased relative thymus weights in females and urinalysis results in both sexes at higher doses.

A 90-day dietary study with dose levels of 30, 100, 300, and 1000 ppm TERP in the diet resulted in slightly decreased food intake, body and organ weight changes, and decreased specific gravity of the urine at the highest dose. The NOAEL was 300 ppm in diet (equivalent to 15 mg/kg bw/d). A 28-day gavage study using TERP showed changes in clinical chemistry and slight differences in haematology at 150 mg/kg bw/d and higher. The NOAEL was 50 mg/kg bw/d.

The effects of MMT(IOTG) were evaluated in a 90-day dietary study using doses of 100, 500, and 1500 ppm (decreased from 2500 ppm) in the diet. Based on clinical chemistry effects at 500 ppm and other effects at higher doses, the NOAEL was 100 ppm in diet (approximately 6-21 mg/kg bw/d).

Neurotoxicity:

In a guideline 90-day subchronic dietary study conducted in Wistar rats, effects occurred at the high dose of 750 ppm MMT(2-EHMA, (equivalent to 49.7 mg/kg bw/day in males and 53.6 mg/kg bw/day in females), which consisted of changes in neurobehavioral parameters and associated brain histopathology. The NOAEL was the next lower dose of 150 ppm (equivalent to 9.8 mg/kg bw/day in males and 10.2 mg/kg bw/day in females)

Immunotoxicity:

Immune function was assessed in male Sprague-Dawley rats exposed to the mixture of organotin used in PVC pipe production.

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	Adult male rats were given drinking water for 28 d containing a mixture of dibutyltin dichloride (DBTC), dimethyltin dichloride (DMTC), monobutyltin trichloride (MBT), and monomethyltin trichloride (MMT) in a 2:2:1:1 ratio, respectively, at 3 different concentrations (5:5:2.5:2.5, 10:10:5:5, or 20:20:10:10 mg organotin/L). Rats were also exposed to MMT alone (20 or 40 mg MMT/L) or plain water as a control. Delayed-type hypersensitivity, antibody synthesis, and natural killer cell cytotoxicity were evaluated in separate endpoint groups immediately after exposure ended. The evaluated immune functions were not affected by the mixture or by MMT alone. The data suggest that immunotoxicity is unlikely to result from the concentration of organotin present in drinking water delivered via PVC pipes, as the concentrations used were several orders of magnitude higher than those expected to leach from PVC pipes Genotoxicity: In a guideline 90-day subchronic dietary study in rats, with MMT(2-EHMA), based on the changes in neurobehavioral parameters and associated brain histopathology that occurred at the high dose of 750 ppm (equivalent to 49.7 mg/kg bw/day in males and 53.6 mg/kg bw/day in females), as well as changes in haematology, clinical chemistry, urinalysis, organ weights, and pathology of the thymus at the same dose, the NOAEL was the next lower dose of 150 ppm (equivalent to 9.8 mg/kg bw/day in males and 10.2 mg/kg bw/day in females). The monomethyltin compounds as a class are not mutagenic in the Ames test. TERP was positive in a human lymphocyte assay. MMTC was equivocal for induction of micronucleated polychromatic erythrocytes (MPEs) in an in vivo rat micronucleus test (OECD 474). In this study a statistically significant increase in MPE was observed only at 24 h and not at 48 h after treatment and there was no dose-response. Based on these observations the overall conclusion is that MMTC does not have genotoxic potential. From the results obtained in a micronucleus test with MMT(2-EHMA), it was demonstrated that the substance was weakly genotoxic to bone marrow cells of rats and that the substance has the potential to induce damage to the mitotic spindle apparatus of the bone marrow target cells. Carcinogenicity: In a limited carcinogenicity study, MMT(EHTG) produced no compound-related macroscopic or microscopic changes in rats fed 100 ppm in the diet for two years. Toxicity to reproduction: In the reproductive satellite portion of the 90-day study using MMTC (with dose levels of 30, 150, and 750 ppm in the diet), post-implantation loss, decreased litter size and increased neonatal mortality occurred at 750 ppm (26-46 mg/kg bw/d for females). Maternal gestational body weights were transiently suppressed and other maternal toxicity was inferred from the repeated dose results at this dose. There were no malformations observed at any dose. The NOAEL for maternal toxicity, and reproductive, and foetotoxic effects was 150 ppm in the diet (6-12 mg/kg bw/d). SIDS Initial Assessment Profile (SIAM 23 2006) ECHA Registration Dossier for MMT(2-EHMA) (ethylhexyl 10-ethyl-4-[[2-[(2-ethylhexyl)oxy]-2-oxoethyl]thio]-4-methyl-7-oxo-8-oxa-3,5-dithia-4-stannatetradecanoate) d-Limonene is readily absorbed by inhalation and swallowing. Absorption through the skin is reported to be lower than by inhalation. It is rapidly distributed to different tissues in the body, readily metabolized and eliminated, primarily 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 sensitize 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.
D-LIMONENE & ALPHA-TERPINEOL & DIHYDROMYRCENOL & CITRAL & LEMON OIL	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 prehaptens 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 sensitizers. 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 show 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 as prohaptens. The possibility of a prohaptens 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.
ALPHA-TERPINEOL & CITRIC ACID, MONOHYDRATE & BETA-CITRONELLOL & CITRAL & P-CYMENTH & ISOTHIAZOLINONES, MIXED & LEMON OIL & C.I. ACID YELLOW 23 & C.I. FOOD YELLOW 3	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.
ALPHA-TERPINEOL & DIHYDROMYRCENOL	For terpenoid tertiary alcohols and their related esters: These substances are metabolised in the liver and excreted primarily in the urine and faeces. A portion is also excreted unchanged. They have low short term toxicity when ingested or applied on the skin. However, repeated and long term use may cause dose dependent harm to both the foetus and mother.
ALPHA-TERPINEOL & BETA-CITRONELLOL	With few exceptions* (see below), there are no safety concerns regarding certain cyclic and non-cyclic terpene alcohols **, as fragrance ingredients, under present declared levels of use and exposure, because - They have low acute toxicity - No significant toxicity was observed in repeat dose toxicity tests - They were not found to cause mutations or genetic toxicity - Substances in this group are processed similarly in the body - There is no indication of persistent breakdown products causing severe toxicity - They practically do not irritate the skin - They have a generally low potential for sensitization - The margin of safety is more than 100 times the maximum daily exposure. *Safety concerns exist for the following substances for the following reasons: - 6,7-dihydrogeraniol, hydroabietyl alcohol and 2-isopropyl-2-decahydronaphthalenol are potent skin sensitizers. - Farnesol is a weak sensitizer. - Scalrol and linalool may contain impurities and/or oxidation products that are strong sensitizers. - No sensitization test results were available for 2(10)-pinen-3-ol, 2,6-dimethyloct-3,5-dien-2-ol, and 3,7-dimethyl-4,6-octadien-3-ol. These materials should be regarded as potential sensitizers until tested. ** The common characteristic structural element of acyclic -noncyclic- and cyclic terpene alcohols is the typically branched isoprene unit 2-methyl-1,3-butadiene
DIHYDROMYRCENOL & BETA-CITRONELLOL	Current opinion holds that there are no safety concerns regarding the branched chain unsaturated non-cyclic alcohols, as fragrance ingredients, at current declared levels of use and exposure; however, use of these materials at higher maximum levels of skin or whole-body exposure requires re-evaluation.

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	At current declared levels of use, there was no evidence or only minimal evidence of skin irritation in humans. Sensitising hydroperoxides may be formed by contact with air. It should be ensured that oxidation reactions are prevented in the end product. The use of these materials under the declared levels of use and exposure will not induce sensitization. These compounds generally have low acute toxicity. The branched chain, unsaturated alcohols tested had low whole-body toxicity after repeated application. In animals, repeated exposure at high doses caused liver changes and kidney damage. There was little or no evidence of adverse effects on fertility or development. Data on cancer-causing potential is not available, but they are not of primary concern. Alkyl alcohols of chain length C6-13 are absorbed from skin, when inhaled or swallowed but show evidence of little harm. They are broken down and rapidly excreted by the body.
CITRIC ACID, MONOHYDRATE & ISOTHIAZOLINONES, MIXED	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
ALPHA-HEXYLCINNAMALDEHYDE & BETA-CITRONELLOL	Fragrance allergens act as haptens, low molecular weight chemicals that cause an immune response only when attached to a carrier protein. However, not all sensitizing fragrance chemicals are directly reactive, but require previous activation. A prohaptens is a chemical that itself causes little or no sensitization, but is transformed into a hapten in the skin (bioactivation), usually via enzyme catalysis. It is not always possible to know whether a particular allergen that is not directly reactive acts as a prohaptens or a hapten , or both. Prohaptens: Compounds that are bioactivated in the skin and thereby form haptens are referred to prohaptens. The possibility of a prohaptens 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.
ALPHA-HEXYLCINNAMALDEHYDE & CITRAL	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.
C.I. ACID YELLOW 3 & C.I. FOOD YELLOW 3	There has been public concern that food colourings may cause ADHD-like behavior in children. These concerns have led the FDA and other food safety authorities to regularly review the scientific literature, and led the UK FSA to commission a study by researchers at Southampton University of the effect of a mixture of six food dyes (Tartrazine, Allura Red AC, Ponceau 4R, Quinoline Yellow WS, Sunset Yellow and Carmoisine (dubbed the "Southampton 6")) and sodium benzoate (a preservative) on children in the general population, who consumed them in beverages; the study published in 2007. The study found "a possible link between the consumption of these artificial colours and a sodium benzoate preservative and increased hyperactivity" in the children; the advisory committee to the FSA that evaluated the study also determined that because of study limitations, the results could not be extrapolated to the general population, and further testing was recommended". The European regulatory community, with a stronger emphasis on the precautionary principle, required labelling and temporarily reduced the acceptable daily intake (ADI) for the food colourings; the UK FSA called for voluntary withdrawal of the colourings by food manufacturers. However, in 2009 the EFSA re-evaluated the data at hand and determined that "the available scientific evidence does not substantiate a link between the colour additives and behavioural effects". There is no evidence to support broad claims that food colouring causes food intolerance and ADHD-like behaviour in children. It is possible that certain food colouring may act as a trigger in those who are genetically predisposed, but the evidence is weak.

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

Legend: ✗ – Data either not available or does not fill the criteria for classification
✓ – Data available to make classification

SECTION 12 Ecological information

Toxicity

PowerHouse - Lemon Cleaner 5in1 Action 28 fl oz	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
sodium linear-(C12-14)alkyl ether sulfate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	7.4mg/l	2
	EC50	72h	Algae or other aquatic plants	27.7mg/l	2
	LC50	96h	Fish	>1<10mg/l	2
	NOEC(ECx)	672h	Fish	0.14mg/l	2
D-glucose, decyl octyl ethers, oligomeric	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	31.62mg/l	2
	EC50	72h	Algae or other aquatic plants	12.43mg/l	2
	LC50	96h	Fish	96.64mg/l	2
	NOEC(ECx)	672h	Fish	1mg/l	2
ethanol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	2mg/L	4
	EC50	72h	Algae or other aquatic plants	275mg/l	2
	LC50	96h	Fish	42mg/L	4
	EC50	96h	Algae or other aquatic plants	<0.001mg/L	4
	EC50(ECx)	96h	Algae or other aquatic plants	<0.001mg/L	4

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castor oil, hydrogenated, ethoxylated	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	48h	Crustacea	>1mg/l	2
	EC50	48h	Crustacea	>1mg/l	2
	EC50	72h	Algae or other aquatic plants	>1mg/l	2
	LC50	96h	Fish	>1mg/l	2
d-limonene	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	0.46mg/l	2
	EC50	48h	Crustacea	0.307mg/l	2
	EC50	72h	Algae or other aquatic plants	0.214mg/l	2
	NOEC(ECx)	0h	Algae or other aquatic plants	<0.05-1.5mg/L	4
alpha-terpineol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	73mg/l	2
	EC50	72h	Algae or other aquatic plants	~17mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	~3.9mg/l	2
	LC50	96h	Fish	6.3mg/l	4
dihydromyrcenol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	65mg/l	2
	NOEC(ECx)	96h	Fish	<3.5mg/l	2
	LC50	96h	Fish	27.8mg/l	2
	EC50	48h	Crustacea	38mg/l	2
citric acid, monohydrate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC10(ECx)	24h	Algae or other aquatic plants	>1000mg/l	4
alpha-hexylcinnamaldehyde	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	>0.36<0.59mg/l	2
	EC50	72h	Algae or other aquatic plants	>0.065mg/l	2
	NOEC(ECx)	504h	Crustacea	0.063mg/L	2
	LC50	96h	Fish	~1.7mg/l	2
beta-citronellol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	17.48mg/l	2
	EC50	72h	Algae or other aquatic plants	2.4mg/l	2
	EC20(ECx)	72h	Algae or other aquatic plants	1.1mg/l	2
	LC50	96h	Fish	14.66mg/l	2
citral	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	4.6mg/l	1
	EC50	48h	Crustacea	6.8mg/l	2
	EC50	72h	Algae or other aquatic plants	16mg/l	1
	EC10(ECx)	96h	Algae or other aquatic plants	1.9mg/l	1
	EC50	96h	Algae or other aquatic plants	19mg/l	1
p-cymene	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	1.9mg/l	2
	EC50	72h	Algae or other aquatic plants	2.01mg/l	2
	EC50	96h	Algae or other aquatic plants	22mg/L	4
	NOEC(ECx)	72h	Algae or other aquatic plants	<0.623mg/l	2
	LC50	96h	Fish	2mg/l	2
isothiazolinones, mixed	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	0.129mg/l	2
	EC50	48h	Crustacea	0.007mg/l	2
	EC50	72h	Algae or other aquatic plants	0.006mg/L	2
	NOEC(ECx)	48h	Algae or other aquatic plants	<0.001mg/L	2
	EC50	96h	Algae or other aquatic plants	0.036mg/L	2
lemon oil	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
C.I. Acid Yellow 23	Endpoint	Test Duration (hr)	Species	Value	Source

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	BCF	1008h	Fish	<0.29	7
	EC50	48h	Crustacea	>120mg/l	2
	EC50	72h	Algae or other aquatic plants	>125mg/l	2
	EC50(ECx)	48h	Crustacea	>120mg/l	2
	LC50	96h	Fish	>120mg/l	2
C.I. Acid Yellow 3	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	202.6mg/l	2
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	NOEC(ECx)	504h	Crustacea	5.992mg/l	2
	LC50	96h	Fish	>100mg/l	2
C.I. Food Yellow 3	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	>100mg/l	2
	EC50	72h	Algae or other aquatic plants	113.2mg/l	2
	EC50	96h	Algae or other aquatic plants	44524mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	15.729mg/l	2
citral diethyl acetal	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	12.35mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	1.5mg/l	2
	EC50	72h	Algae or other aquatic plants	1.6-19mg/l	2
	LC50	96h	Fish	7.2mg/l	2
Legend:		Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data			

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ethanol	LOW (Half-life = 2.17 days)	LOW (Half-life = 5.08 days)
d-limonene	HIGH	HIGH
alpha-terpineol	HIGH	HIGH
dihydromyrcenol	HIGH	HIGH
citric acid, monohydrate	LOW	LOW
alpha-hexylcinnamaldehyde	LOW	LOW
beta-citronellol	LOW	LOW
citral	LOW	LOW
p-cymene	HIGH	HIGH
C.I. Acid Yellow 23	HIGH	HIGH
citral diethyl acetal	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
sodium linear-(C12-14)alkyl ether sulfate	LOW (LogKOW = 1.62)
D-glucose, decyl octyl ethers, oligomeric	LOW (LogKOW = -0.58)
ethanol	LOW (LogKOW = -0.31)
d-limonene	HIGH (LogKOW = 4.8275)
alpha-terpineol	LOW (LogKOW = 3.28)
dihydromyrcenol	LOW (LogKOW = 3.47)
citric acid, monohydrate	LOW (LogKOW = -1.64)
alpha-hexylcinnamaldehyde	HIGH (LogKOW = 4.82)
beta-citronellol	MEDIUM (LogKOW = 3.91)
citral	LOW (LogKOW = 3.45)
p-cymene	MEDIUM (LogKOW = 3.9963)
lemon oil	MEDIUM (LogKOW = 4.38)
C.I. Acid Yellow 23	LOW (BCF = 3)
C.I. Acid Yellow 3	LOW (LogKOW = 1.06)
citral diethyl acetal	HIGH (LogKOW = 4.82)

Mobility in soil

Continued...

Ingredient	Mobility
ethanol	HIGH (Log KOC = 1)
d-limonene	LOW (Log KOC = 1324)
alpha-terpineol	LOW (Log KOC = 57.85)
dihydromyrcenol	LOW (Log KOC = 54.78)
citric acid, monohydrate	LOW (Log KOC = 10)
alpha-hexylcinnamaldehyde	LOW (Log KOC = 4025)
beta-citronellol	LOW (Log KOC = 70.79)
citral	LOW (Log KOC = 147.7)
p-cymene	LOW (Log KOC = 1324)
C.I. Acid Yellow 23	LOW (Log KOC = 79.38)
citral diethyl acetal	LOW (Log KOC = 136)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority. ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Authority for disposal. ▶ Bury or incinerate residue at an approved site. ▶ Recycle containers if possible, or dispose of in an authorised landfill.
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SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

14.7. 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
sodium linear-(C12-14)alkyl ether sulfate	Not Available
D-glucose, decyl octyl ethers, oligomeric	Not Available
ethanol	Not Available
castor oil, hydrogenated, ethoxylated	Not Available
d-limonene	Not Available
alpha-terpineol	Not Available
dihydromyrcenol	Not Available
citric acid, monohydrate	Not Available
alpha-hexylcinnamaldehyde	Not Available
beta-citronellol	Not Available
citral	Not Available
p-cymene	Not Available
isothiazolinones, mixed	Not Available
lemon oil	Not Available
C.I. Acid Yellow 23	Not Available
C.I. Acid Yellow 3	Not Available
C.I. Food Yellow 3	Not Available
citral diethyl acetal	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
sodium linear-(C12-14)alkyl ether sulfate	Not Available
D-glucose, decyl octyl ethers, oligomeric	Not Available
ethanol	Not Available
castor oil, hydrogenated, ethoxylated	Not Available
d-limonene	Not Available
alpha-terpineol	Not Available
dihydromyrcenol	Not Available
citric acid, monohydrate	Not Available
alpha-hexylcinnamaldehyde	Not Available
beta-citronellol	Not Available
citral	Not Available
p-cymene	Not Available
isothiazolinones, mixed	Not Available
lemon oil	Not Available
C.I. Acid Yellow 23	Not Available
C.I. Acid Yellow 3	Not Available
C.I. Food Yellow 3	Not Available
citral diethyl acetal	Not Available

SECTION 15 Regulatory information

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

- sodium linear-(C12-14)alkyl ether sulfate is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)
- D-glucose, decyl octyl ethers, oligomeric is found on the following regulatory lists**

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

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

Australian Inventory of Industrial Chemicals (AIIC)
- castor oil, hydrogenated, ethoxylated is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)
- d-limonene is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
- alpha-terpineol is found on the following regulatory lists**

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

Australian Inventory of Industrial Chemicals (AIIC)
- citric acid, monohydrate is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)
- alpha-hexylcinnamaldehyde is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)
- beta-citronellol is found on the following regulatory lists**

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

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australian Inventory of Industrial Chemicals (AIIC)
- p-cymene is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)
- isothiazolinones, mixed is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
- lemon oil is found on the following regulatory lists**

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australian Inventory of Industrial Chemicals (AIIC)

C.I. Acid Yellow 23 is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australian Inventory of Industrial Chemicals (AIIC)

C.I. Acid Yellow 3 is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)

C.I. Food Yellow 3 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

citral diethyl acetal is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (isothiazolinones, mixed)
Canada - DSL	Yes
Canada - NDSL	No (sodium linear-(C12-14)alkyl ether sulfate; D-glucose, decyl octyl ethers, oligomeric; ethanol; castor oil, hydrogenated, ethoxylated; d-limonene; alpha-terpineol; dihydromyrcenol; citric acid, monohydrate; alpha-hexylcinnamaldehyde; beta-citronellol; citral; p-cymene; isothiazolinones, mixed; lemon oil; C.I. Acid Yellow 23; C.I. Acid Yellow 3; C.I. Food Yellow 3; citral diethyl acetal)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (isothiazolinones, mixed; lemon oil)
Japan - ENCS	No (lemon oil)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	TSCA Inventory 'Active' substance(s) (sodium linear-(C12-14)alkyl ether sulfate; D-glucose, decyl octyl ethers, oligomeric; ethanol; castor oil, hydrogenated, ethoxylated; d-limonene; alpha-terpineol; dihydromyrcenol; citric acid, monohydrate; alpha-hexylcinnamaldehyde; beta-citronellol; citral; p-cymene; lemon oil; C.I. Acid Yellow 23; C.I. Acid Yellow 3; C.I. Food Yellow 3; citral diethyl acetal); No (isothiazolinones, mixed)
Taiwan - TCSI	Yes
Mexico - INSQ	No (sodium linear-(C12-14)alkyl ether sulfate; D-glucose, decyl octyl ethers, oligomeric; castor oil, hydrogenated, ethoxylated; alpha-hexylcinnamaldehyde; isothiazolinones, mixed; lemon oil; C.I. Food Yellow 3; citral diethyl acetal)
Vietnam - NCI	Yes
Russia - FBEPH	No (lemon oil; citral diethyl acetal)
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	20/06/2025
Initial Date	20/06/2025

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	20/06/2025	Identification of the substance / mixture and of the company / undertaking - Synonyms

Other information

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

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

Definitions and abbreviations

- ▶ PC - TWA: Permissible Concentration-Time Weighted Average
- ▶ PC - STEL: Permissible Concentration-Short Term Exposure Limit
- ▶ IARC: International Agency for Research on Cancer
- ▶ ACGIH: American Conference of Governmental Industrial Hygienists
- ▶ STEL: Short Term Exposure Limit
- ▶ TEEL: Temporary Emergency Exposure Limit,
- ▶ IDLH: Immediately Dangerous to Life or Health Concentrations
- ▶ ES: Exposure Standard
- ▶ OSF: Odour Safety Factor
- ▶ NOAEL: No Observed Adverse Effect Level

- LOAEL: Lowest Observed Adverse Effect Level
- TLV: Threshold Limit Value
- LOD: Limit Of Detection
- OTV: Odour Threshold Value
- BCF: BioConcentration Factors
- BEI: Biological Exposure Index
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration
- MARPOL: International Convention for the Prevention of Pollution from Ships
- IMSBC: International Maritime Solid Bulk Cargoes Code
- IGC: International Gas Carrier Code
- IBC: International Bulk Chemical Code

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

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TEL (+61 3) 9572 4700.