

Paint/Graffiti Remover #198-1039, 487-2736(Paint/Graffiti Remover #198-1039, 487-2736)

RS Components Pty Ltd

Chemwatch Hazard Alert Code: 4

Chemwatch: 5232-13

Version No: 2.1.1.1

Safety Data Sheet according to WHS and ADG requirements

Issue Date: 05/12/2016

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L.GHS.AUS.EN

SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	Paint/Graffiti Remover #198-1039, 487-2736 (Paint/Graffiti Remover #198-1039, 487-2736)
Synonyms	Manufacturer's Code: 198-1039, 487-2736
Proper shipping name	AEROSOLS
Other means of identification	Not Available

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

Relevant identified uses	Application is by spray atomisation from a hand held aerosol pack Paint/graffiti remover.
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Details of the supplier of the safety data sheet

Registered company name	RS Components Pty Ltd
Address	25 Pavasi Street NSW 2164 Australia
Telephone	1300 656 636
Fax	1300 656 696
Website	Not Available
Email	Not Available

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	1800 039 008
Other emergency telephone numbers	03 95733112

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

HAZARDOUS CHEMICAL. DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

CHEMWATCH HAZARD RATINGS

	Min	Max
Flammability	4	
Toxicity	1	
Body Contact	2	
Reactivity	1	
Chronic	2	

0 = Minimum
1 = Low
2 = Moderate
3 = High
4 = Extreme

Poisons Schedule	Not Applicable
Classification [1]	Aerosols Category 1, Gas under Pressure (Compressed gas), Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Skin Sensitizer Category 1*, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Acute Aquatic Hazard Category 3, Chronic Aquatic Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

Label elements

GHS label elements	
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SIGNAL WORD	DANGER
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Hazard statement(s)

H222	Extremely flammable aerosol.
H280	Contains gas under pressure; may explode if heated.

Continued...

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H315	Causes skin irritation.
H319	Causes serious eye irritation.
H317	May cause an allergic skin reaction.*
H336	May cause drowsiness or dizziness.
H412	Harmful to aquatic life with long lasting effects.
AUH019	May form explosive peroxides
AUH044	Risk of explosion if heated under confinement

Precautionary statement(s) Prevention

P210	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
P211	Do not spray on an open flame or other ignition source.
P251	Pressurized container: Do not pierce or burn, even after use.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P261	Avoid breathing gas.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P362	Take off contaminated clothing and wash before reuse.
P302+P352	IF ON SKIN: Wash with plenty of soap and 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.
P312	Call a POISON CENTER or doctor/physician if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.
P410+P403	Protect from sunlight. Store in a well-ventilated place.
P410+P412	Protect from sunlight. Do not expose to temperatures exceeding 50 °C/122 °F.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.
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SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
107-98-2	>60	<u>propylene glycol monomethyl ether - alpha isomer</u>
67-63-0	1-5	<u>isopropanol</u>
104-55-2	<1	<u>cinnamaldehyde</u>
8028-48-6	<1	<u>citrus terpenes</u>
74-98-6	10-30	<u>propane</u>
106-97-8.	5-10	<u>butane</u>
75-28-5.	1-5	<u>iso-butane</u>

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	<p>If aerosols come in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh 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. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If solids or aerosol mists are deposited upon the skin:</p> <ul style="list-style-type: none"> ▶ Flush skin and hair with running water (and soap if available). ▶ Remove any adhering solids with industrial skin cleansing cream. ▶ DO NOT use solvents. ▶ Seek medical attention in the event of irritation.

Inhalation	<p>If aerosols, fumes or combustion products are inhaled:</p> <ul style="list-style-type: none"> ▶ Remove to fresh air. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor.
Ingestion	<ul style="list-style-type: none"> ▶ Not considered a normal route of entry.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 FIREFIGHTING MEASURES**Extinguishing media****SMALL FIRE:**

- ▶ Water spray, dry chemical or CO2

LARGE FIRE:

- ▶ Water spray or fog.

Special hazards arising from the substrate or mixture

Fire Incompatibility	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ If safe, switch off electrical equipment until vapour fire hazard removed. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use. <p>-----</p> <p>GENERAL</p> <p>-----</p> <ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves. ▶ Consider evacuation ▶ Fight fire from a safe distance, with adequate cover. ▶ If safe, switch off electrical equipment until vapour fire hazard removed. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ DO NOT approach cylinders suspected to be hot. ▶ Cool fire-exposed cylinders with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. <p>-----</p> <p>FIRE FIGHTING PROCEDURES:</p> <p>-----</p> <ul style="list-style-type: none"> ▶ The only safe way to extinguish a flammable gas fire is to stop the flow of gas. ▶ If the flow cannot be stopped, allow the entire contents of the cylinder to burn while cooling the cylinder and surroundings with water from a suitable distance. ▶ Extinguishing the fire without stopping the gas flow may permit the formation of ignitable or explosive mixtures with air. These mixtures may propagate to a source of ignition. <p>-----</p> <p>SPECIAL HAZARDS</p> <p>-----</p> <ul style="list-style-type: none"> ▶ Excessive pressures may develop in a gas cylinder exposed in a fire; this may result in explosion. ▶ Cylinders with pressure relief devices may release their contents as a result of fire and the released gas may constitute a further source of hazard for the fire-fighter. ▶ Cylinders without pressure-relief valves have no provision for controlled release and are therefore more likely to explode if exposed to fire. <p>-----</p> <p>FIRE FIGHTING REQUIREMENTS:</p> <p>-----</p> <p>The need for proximity, entry and flash-over protection and special protective clothing should be determined for each incident, by a competent fire-fighting safety professional.</p>
Fire/Explosion Hazard	<p>***</p> <ul style="list-style-type: none"> ▶ Liquid and vapour are highly flammable. ▶ Severe fire hazard when exposed to heat or flame. ▶ Vapour forms an explosive mixture with air. ▶ Severe explosion hazard, in the form of vapour, when exposed to flame or spark. ▶ Vapour may travel a considerable distance to source of ignition. ▶ Heating may cause expansion or decomposition with violent container rupture. ▶ Aerosol cans may explode on exposure to naked flames. ▶ Rupturing containers may rocket and scatter burning materials. ▶ Hazards may not be restricted to pressure effects. ▶ May emit acrid, poisonous or corrosive fumes. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). <p>Combustion products include: carbon monoxide (CO) carbon dioxide (CO2) other pyrolysis products typical of burning organic material.</p>

	Contains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions.
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	<ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Wear protective clothing, impervious gloves and safety glasses. ▶ Shut off all possible sources of ignition and increase ventilation. ▶ Wipe up. ▶ If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated. ▶ Undamaged cans should be gathered and stowed safely.
Major Spills	<ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water courses ▶ 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. ▶ Absorb or cover spill with sand, earth, inert materials or vermiculite. ▶ If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated. ▶ Undamaged cans should be gathered and stowed safely. ▶ Collect residues and seal in labelled drums for disposal.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<p>The substance accumulates peroxides which may become hazardous only if it evaporates or is distilled or otherwise treated to concentrate the peroxides. The substance may concentrate around the container opening for example.</p> <p>Purchases of peroxidisable chemicals should be restricted to ensure that the chemical is used completely before it can become peroxidised.</p> <ul style="list-style-type: none"> ▶ A responsible person should maintain an inventory of peroxidisable chemicals or annotate the general chemical inventory to indicate which chemicals are subject to peroxidation. An expiration date should be determined. The chemical should either be treated to remove peroxides or disposed of before this date. ▶ The person or laboratory receiving the chemical should record a receipt date on the bottle. The individual opening the container should add an opening date. ▶ Unopened containers received from the supplier should be safe to store for 18 months. ▶ Opened containers should not be stored for more than 12 months. ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ Avoid smoking, naked lights or ignition sources. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ DO NOT incinerate or puncture aerosol cans. ▶ DO NOT spray directly on humans, exposed food or food utensils. ▶ 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 are maintained.
Other information	<ul style="list-style-type: none"> ▶ Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can ▶ Store in original containers in approved flammable liquid storage area. ▶ DO NOT store in pits, depressions, basements or areas where vapours may be trapped. ▶ No smoking, naked lights, heat or ignition sources. ▶ Keep containers securely sealed. Contents under pressure. ▶ Store away from incompatible materials. ▶ Store in a cool, dry, well ventilated area. ▶ Avoid storage at temperatures higher than 40 deg C. ▶ Store in an upright position. ▶ Protect containers against physical damage. ▶ Check regularly for spills and leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Aerosol dispenser. ▶ Check that containers are clearly labelled.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Avoid reaction with oxidising agents <p>44acidbase44compgas</p>

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	propylene glycol monomethyl ether - alpha isomer	Propylene glycol monomethyl ether	369 mg/m ³ / 100 ppm	553 mg/m ³ / 150 ppm	Not Available	Not Available
Australia Exposure Standards	isopropanol	Isopropyl alcohol	983 mg/m ³ / 400 ppm	1230 mg/m ³ / 500 ppm	Not Available	Not Available
Australia Exposure Standards	propane	Propane	Not Available	Not Available	Not Available	Asphyxiant
Australia Exposure Standards	butane	Butane	1900 mg/m ³ / 800 ppm	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
propylene glycol monomethyl ether - alpha isomer	Propylene glycol monomethyl ether; (Ucar Triol HG-170)	100 ppm	160 ppm	660 ppm
isopropanol	Isopropyl alcohol	400 ppm	2000 ppm	12000 ppm
propane	Propane	Not Available	Not Available	Not Available
butane	Butane	Not Available	Not Available	Not Available
iso-butane	Methylpropane, 2-; (Isobutane)	5500 ppm	17000 ppm	53000 ppm

Ingredient	Original IDLH	Revised IDLH
propylene glycol monomethyl ether - alpha isomer	Not Available	Not Available
isopropanol	12,000 ppm	2,000 [LEL] ppm
cinnamaldehyde	Not Available	Not Available
citrus terpenes	Not Available	Not Available
propane	20,000 [LEL] ppm	2,100 [LEL] ppm
butane	Not Available	Not Available
iso-butane	Not Available	Not Available

MATERIAL DATA

Exposure controls

Appropriate engineering controls	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection.</p> <p>Provide adequate ventilation in warehouse or closed storage areas.</p> <p>Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p>									
	<table border="1"> <thead> <tr> <th>Type of Contaminant:</th> <th>Speed:</th> </tr> </thead> <tbody> <tr> <td>aerosols, (released at low velocity into zone of active generation)</td> <td>0.5-1 m/s</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> </tbody> </table>	Type of Contaminant:	Speed:	aerosols, (released at low velocity into zone of active generation)	0.5-1 m/s	direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)			
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<p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>										

Personal protection	
Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. ▶ 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], [AS/NZS 1336 or national equivalent] ▶ Close fitting gas tight goggles
Skin protection	See Hand protection below
Hands/feet protection	<p>NOTE:</p> <ul style="list-style-type: none"> ▶ 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. ▶ Neoprene gloves ▶ No special equipment needed when handling small quantities. <p>OTHERWISE:</p> <ul style="list-style-type: none"> ▶ For potentially moderate exposures: ▶ Wear general protective gloves, eg. light weight rubber gloves. ▶ For potentially heavy exposures: ▶ Wear chemical protective gloves, eg. PVC. and safety footwear.
Body protection	See Other protection below
Other protection	<p>No special equipment needed when handling small quantities.</p> <p>OTHERWISE:</p> <ul style="list-style-type: none"> ▶ Overalls. ▶ Skin cleansing cream. ▶ Eyewash unit. ▶ Do not spray on hot surfaces. ▶ The clothing worn by process operators insulated from earth may develop static charges far higher (up to 100 times) than the minimum ignition energies for various flammable gas-air mixtures. This holds true for a wide range of clothing materials including cotton. ▶ Avoid dangerous levels of charge by ensuring a low resistivity of the surface material worn outermost. <p>BREITHERICK: Handbook of Reactive Chemical Hazards.</p>
Thermal hazards	Not Available

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:

Paint/Graffiti Remover #198-1039 (Paint/Graffiti Remover #198-1039)

Material	CPI
BUTYL	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVC	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Respiratory protection

Type AX-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*	AX-2 P2	AX-PAPR-2 P2 ^
up to 10 x ES	-	AX-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 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.

Aerosols, in common with most vapours/ mists, should never be used in confined spaces without adequate ventilation. Aerosols, containing agents designed to enhance or mask smell, have triggered allergic reactions in predisposed individuals.

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Colourless highly flammable liquid aerosol with a characteristic odour; slightly soluble in water.		
Physical state	Compressed Gas	Relative density (Water = 1)	Not Available

Paint/Graffiti Remover #198-1039, 487-2736 (Paint/Graffiti Remover #198-1039, 487-2736)

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	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 Available	Oxidising properties	Not Available
Upper Explosive Limit (%)	11.5	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	0.8	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Partly miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ Elevated temperatures. ▶ Presence of open flame. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur. ▶ Presence of elevated temperatures.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	<p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation hazard is increased at higher temperatures.</p> <p>Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure. Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination</p> <p>WARNING: Intentional misuse by concentrating/inhaling contents may be lethal.</p>
Ingestion	<p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>Not normally a hazard due to physical form of product.</p> <p>Considered an unlikely route of entry in commercial/industrial environments</p>
Skin Contact	<p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</p> <p>Spray mist may produce discomfort</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
Eye	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals.</p> <p>Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>

	<p>Direct contact with the eye may not cause irritation because of the extreme volatility of the gas; however concentrated atmospheres may produce irritation after brief exposures..</p> <p>The vapour when concentrated has pronounced eye irritation effects and this gives some warning of high vapour concentrations. If eye irritation occurs seek to reduce exposure with available control measures, or evacuate area.</p>
Chronic	<p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Principal route of occupational exposure to the gas is by inhalation.</p> <p>Repeated oral doses of 3 g/kg for propylene glycol <u>monomethyl</u> ether (PGME) produced minor changes in the liver and kidneys in rats. Repeated doses on the skin over a 90-day period resulted in absorption and anaesthetic death at 7-10 ml/kg/day. Mild narcosis was observed after topical application of 2-4 ml/kg/day. Administration of 2% PGME in drinking water ad libitum to males for 25 days did not elicit significant changes in testes or seminal vesicle and coagulating gland weights or in peripheral leukocyte counts. No significant testicular toxicity was found in rats or rabbits that were exposed at up to 3000 PGME, 6 hours/day, 5 days/week for 13 weeks. Oral and parenteral administration to pregnant rabbits, mice and rats did not induce congenital malformations at concentrations up to 1800 mg/kg/day.</p> <p>In a study on the teratogenic potential of the acetate of the beta-isomer (2-methoxy-1-propyl acetate), a significant increase in the number of litters with abnormal rats and rabbits was found after inhalation exposure by the mothers to 2700 ppm or 550 ppm, respectively, on days 6 to 15, or 6 to 18 of gestation. The rabbit inhalation no-observed-adverse effect concentration was 145 ppm. A similar embryotoxicity profile was seen after inhalation of 2-methoxy-1-propanol (beta-PGMA). In contrast to the alpha-isomer, beta-PGMA is oxidised in rats to 2-methoxypropionic acid.</p> <p>Male dogs exposed to the beta-isomer, developed numerous spermophages in epididymi. Administration of high doses of the beta-isomer to rats, by gavage, caused delayed ossification of the skull of rat foetus.</p> <p>Whilst alpha-PGMA undergoes hepatic O-demethylation as the principal pathway, the beta-isomer is detoxified by alcohol/ aldehyde dehydrogenase.</p> <p>Commercial PGME contains low concentrations of the beta-isomer.</p> <p>Long term or repeated ingestion exposure of isopropanol may produce incoordination, lethargy and reduced weight gain.</p> <p>Repeated inhalation exposure to isopropanol may produce narcosis, incoordination and liver degeneration. Animal data show developmental effects only at exposure levels that produce toxic effects in the adult animals. Isopropanol does not cause genetic damage in bacterial or mammalian cell cultures or in animals.</p> <p>There are inconclusive reports of human sensitisation from skin contact with isopropanol. Chronic alcoholics are more tolerant of systemic isopropanol than are persons who do not consume alcohol; alcoholics have survived as much as 500 ml. of 70% isopropanol.</p> <p>Continued voluntary drinking of a 2.5% aqueous solution through two successive generations of rats produced no reproductive effects.</p> <p>NOTE: Commercial isopropanol does not contain "isopropyl oil". An excess incidence of sinus and laryngeal cancers in isopropanol production workers has been shown to be caused by the byproduct "isopropyl oil". Changes in the production processes now ensure that no byproduct is formed. Production changes include use of dilute sulfuric acid at higher temperatures.</p> <p>Studies with some glycol ethers (principally the monoethylene glycols) and their esters indicate reproductive changes, testicular atrophy, infertility and kidney function changes. The metabolic acetic acid derivatives of glycol ethers (alkoxyacetic acids), not the ether itself, have been found to be the proximal reproductive toxin in animals. The potency of these metabolites decreases significantly as the chain length of the ether increases. Consequently glycol ethers with longer substituents (e.g diethylene glycols, triethylene glycols) have not generally been associated with reproductive effects. One of the most sensitive indicators of toxic effects observed from many of the glycol ethers is an increase in the erythrocytic osmotic fragility in rats Which produces haemolytic anaemia). This appears to be related to the development of haemoglobinuria (blood in the urine) at higher exposure levels or as a result of chronic exposure.</p> <p>Glycol ethers based on propylene oxides, propylene glycol ethers, dipropylene glycol ethers and tripropylene glycol ethers are mainly available, commercially, as alpha-isomers (because of thermodynamic considerations); these are incapable of forming alkoxyacetic or alkoxypropionic acids as metabolites and therefore do not produce erythrocyte fragility unless contaminated by ethylene glycol ethers or to a significant degree by the beta-isomer . beta-Isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).</p>

Paint/Graffiti Remover #198-1039 (Paint/Graffiti Remover #198-1039)	TOXICITY	IRRITATION
	Not Available	Not Available
propylene glycol monomethyl ether - alpha isomer	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit) 230 mg mild
	Inhalation (rat) LC50: 10000 ppm/5 hr ^[2]	Eye (rabbit) 500 mg/24 h. - mild
	Oral (rat) LD50: 5207.2 mg/kg ^[1]	Eye (rabbit): 100 mg SEVERE Skin (rabbit) 500 mg open - mild
isopropanol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 12792 mg/kg ^[1]	Eye (rabbit): 10 mg - moderate
	Inhalation (rat) LC50: 72.6 mg/L/4hr ^[2]	Eye (rabbit): 100 mg - SEVERE
	Oral (rat) LD50: 5000 mg/kg ^[2]	Eye (rabbit): 100mg/24hr-moderate Skin (rabbit): 500 mg - mild
cinnamaldehyde	TOXICITY	IRRITATION
	Oral (rat) LD50: 2220 mg/kg ^[2]	Skin (human): 40 mg/48h SEVERE
citrus terpenes	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	Skin (rabbit): 500mg/24h moderate
propane	TOXICITY	IRRITATION
	Inhalation (mouse) LC50: >15.6-<17.9 mm/2hr ^[1]	Not Available
	Inhalation (rat) LC50: >800000 ppm15 min ^[1]	

	Inhalation (rat) LC50: 1354.944 mg/L15 min ^[1]	
	Inhalation (rat) LC50: 1355 mg/15 min ^[1]	
	Inhalation (rat) LC50: 1442.738 mg/L15 min ^[1]	
	Inhalation (rat) LC50: 1443 mg/15 min ^[1]	
	Inhalation (rat) LC50: 570000 ppm15 min ^[1]	
butane	TOXICITY	IRRITATION
	Inhalation (rat) LC50: 658 mg/L4hr ^[2]	Not Available
iso-butane	TOXICITY	IRRITATION
	Inhalation (rat) LC50: 658 mg/L4hr ^[2]	Not Available

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

<p>PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER</p>	<p>for propylene glycol ethers (PGEs): Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM). Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids. Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product. Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body. As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces. As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating None are skin sensitizers. In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested). Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members. One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. In a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health. In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity. The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. <i>In vitro</i>, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic <i>in vivo</i>. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice. NOTE: For PGE - mixed isomers: Exposure of pregnant rats and rabbits to the substance did not give rise to teratogenic effects at concentrations up to 3000 ppm. Foetotoxic effects were seen in rats but not in rabbits at this concentration; maternal toxicity was noted in both species.</p>
<p>ISOPROPANOL</p>	<p>For isopropanol (IPA): Acute toxicity: Isopropanol has a low order of acute toxicity. It is irritating to the eyes, but not to the skin. Very high vapor concentrations are irritating to the eyes, nose, and throat, and prolonged exposure may produce central nervous system depression and narcosis. Human volunteers reported that exposure to 400 ppm isopropanol vapors for 3 to 5 min. caused mild irritation of the eyes, nose and throat. Although isopropanol produced little irritation when tested on the skin of human volunteers, there have been reports of isolated cases of dermal irritation and/or sensitization. The use of isopropanol as a sponge treatment for the control of fever has resulted in cases of intoxication, probably the result of both dermal absorption and inhalation. There have been a number of cases of poisoning reported due to the intentional ingestion of isopropanol, particularly among alcoholics or suicide victims. These ingestions typically result in a comatose condition. Pulmonary difficulty, nausea, vomiting, and headache accompanied by various degrees of central nervous system depression are typical. In the absence of shock, recovery usually occurred. Repeat dose studies: The systemic (non-cancer) toxicity of repeated exposure to isopropanol has been evaluated in rats and mice by the inhalation and oral</p>

routes. The only adverse effects-in addition to clinical signs identified from these studies were to the kidney.

Reproductive toxicity: A recent two-generation reproductive study characterised the reproductive hazard for isopropanol associated with oral gavage exposure. This study found that the only reproductive parameter apparently affected by isopropanol exposure was a statistically significant decrease in male mating index of the F1 males. It is possible that the change in this reproductive parameter was treatment related and significant, although the mechanism of this effect could not be discerned from the results of the study. However, the lack of a significant effect of the female mating index in either generation, the absence of any adverse effect on litter size, and the lack of histopathological findings of the testes of the high-dose males suggest that the observed reduction in male mating index may not be biologically meaningful.

Developmental toxicity: The developmental toxicity of isopropanol has been characterized in rat and rabbit developmental toxicity studies. These studies indicate that isopropanol is not a selective developmental hazard. Isopropanol produced developmental toxicity in rats, but not in rabbits. In the rat, the developmental toxicity occurred only at maternally toxic doses and consisted of decreased foetal body weights, but no teratogenicity

Genotoxicity: All genotoxicity assays reported for isopropanol have been negative

Carcinogenicity: rodent inhalation studies were conducted to evaluate isopropanol for cancer potential. The only tumor rate increase seen was for interstitial (Leydig) cell tumors in the male rats. Interstitial cell tumors of the testis is typically the most frequently observed spontaneous tumor in aged male Fischer 344 rats. These studies demonstrate that isopropanol does not exhibit carcinogenic potential relevant to humans. Furthermore, there was no evidence from this study to indicate the development of carcinomas of the testes in the male rat, nor has isopropanol been found to be genotoxic. Thus, the testicular tumors seen in the isopropanol exposed male rats are considered of no significance in terms of human cancer risk assessment

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

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.

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. 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. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production. Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A prohaptens is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems. A prohaptens is a chemical that itself is non- or low-sensitising but that 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 as a prohaptens, or both, because air oxidation and bioactivation can often give the same product (geraniol is an example). Some chemicals might act by all three pathways.

Prohaptens

Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohaptens.

In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal.

The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin. These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohaptens can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity.

QSAR prediction: The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptens) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptens. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation

For cinnamyl aldehyde derivatives:

The chemical category designated "cinnamyl derivatives" includes cinnamaldehyde, two alkyl-substituted cinnamaldehydes, and one alkyl-substituted dihydrocinnamaldehyde derivative. The four substances are grouped together because of their close structural relationships and the resulting similarities of their physio-chemical and toxicological properties. Common structural features among members of this chemical category are that they contain either a 3-phenyl-2-propenal or 3-phenylpropanal backbone. The group includes cinnamaldehyde (3-phenyl-2-propenal), *alpha*-amylcinnamaldehyde (2-amyl-3-phenyl-2-propenal), *alpha*-hexylcinnamaldehyde (2-hexyl-3-phenyl-2-propenal) and *p*-*t*-butyl-*alpha*-methylhydrocinnamaldehyde [3-(*p*-*t*-butylphenyl)-2-methylpropanal].

In nature, cinnamaldehyde is the predominant constituent of cassia oil and Ceylon cinnamon bark oil. It is responsible for the spicy aroma strongly reminiscent of cinnamon spice. It is common components of traditional foods. Cinnamaldehyde, *alpha*-amylcinnamaldehyde, and *alpha*-hexylcinnamaldehyde are currently recognized by the U.S. Food and Drug Administration (FDA) as GRAS ("generally regarded as safe") for their intended use as flavoring substances.

Quantitative natural occurrence data for cinnamaldehyde indicates that oral intake occurs predominantly from consumption of cinnamon spice products and cinnamon flavorings

Acute Toxicity: Oral LD50 values have been reported for the four substances in this chemical category. In rats, LD50 values are in the range of 2220-3400 mg/kg, demonstrating that the oral acute toxicity of these substances is extremely low

Cinnamaldehyde, the *alpha*-amyl and *alpha*-hexyl derivatives and its saturated analog (*p*-*t*-butyl-*alpha*-methylhydrocinnamaldehyde) are rapidly absorbed from the gut, metabolised and excreted primarily in the urine and, to a minor extent, in the faeces.

Dermal acute toxicity shows a similar trend for the four substances in this chemical category. Dermal LD50 values range from a low of 590 ul/kg for cinnamaldehyde to more than 2000 mg/kg for *alpha*-amylcinnamaldehyde, more than 3000 mg/kg for *alpha*-hexylcinnamaldehyde, and more than 5000 mg/kg for *p*-*t*-butyl-*alpha*-methylhydrocinnamaldehyde

Genotoxicity: Cinnamaldehyde and its alkyl-substituted derivatives lack direct mutagenic or genotoxic activity, as indicated by the negative results obtained in bacterial test systems. Evidence of genotoxic activity was observed in isolated mammalian cells, with the cinnamyl compounds producing chromosome aberrations and/or mutations in the respective test systems regardless of the presence or absence of metabolic activation; however, the reported *in vitro* activity did not translate into mutagenic, clastogenic, or genotoxic activity *in vivo*.

Reproductive Toxicity: Reproductive studies on cinnamyl derivatives have concentrated on the parent alcohol, aldehyde, and acid. Rats were administered 5, 25, or 250 mg/kg bw/day cinnamaldehyde by gavage in olive oil on days 7 to 17 of gestation. Foetal abnormalities observed included: poor cranial ossification in all dose groups; increased incidences of dilated pelvis/reduced papilla in the kidney as well as dilated urethras in

CINNAMALDEHYDE

the low- and mid-dose groups; and an increase in the number of fetuses with two or more abnormal sternebrae in the mid-dose group. These effects are associated with apparent maternal toxicity as evidenced by a dose related decrease in weight gain at the two highest dose levels.

Developmental Toxicity: In an *in vivo* developmental toxicity assay, 50 time-mated CD-1 female mice received single oral doses of 1200 mg/kg of cinnamaldehyde in corn oil on days 6-13 of gestation. Female body weights were measured on days 6-15 of gestation and 3 days postpartum. Endpoints monitored included litter size, birth weight, neonatal growth, and survival to 3 days postpartum. Based on the measured parameters there was no significant difference between test and control groups

Metabolic disposition: Rodent and humans studies for cinnamaldehyde and *alpha*-substituted cinnamaldehydes indicate that cinnamyl derivatives are absorbed, metabolised and excreted as polar metabolites within 24 hours.

The aromatic cinnamaldehyde derivatives are readily oxidised to cinnamic acid derivatives. Human NAD⁺ dependent alcohol dehydrogenase (ADH) catalyzes oxidation of primary alcohols to aldehydes. Isoenzyme mixtures of NAD⁺ dependent aldehyde dehydrogenase (ALD) catalyse oxidation of aldehydes to carboxylic acids. Aromatic alcohols and aldehydes have been reported to be excellent substrates for ADH and ALD respectively. The urinary metabolites of cinnamyl alcohol and cinnamaldehyde are mainly derived from metabolism of cinnamic acid. The position and size of the substituent do not significantly affect the pathways of metabolic detoxication of cinnamyl derivatives. Cinnamyl derivatives containing *alpha*-alkyl substituents (e.g. *alpha*-methylcinnamaldehyde) are extensively metabolised *via beta*-oxidation followed by cleavage to yield mainly the corresponding hippuric acid derivative.

Ring substituents (e.g. 3-(*p*-isopropylphenyl)propionaldehyde and *p*-methylcinnamaldehyde) do not significantly impact metabolism *via beta*-oxidation. Based on these observations, it may be concluded that the presence of side-chain alkyl substituents and ring substituents do not alter the principal metabolic detoxication pathway for cinnamyl derivatives.

The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis.

Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.

A member or analogue of a group of cinnamyl derivatives generally regarded as safe (GRAS) based in part, on their self-limiting properties as flavouring substances in food; their rapid absorption, metabolic detoxication, and excretion in humans and other animals; their low level of flavour use; the wide margins of safety between the conservative estimates of intake and the no adverse effect levels determined from subchronic and chronic studies and the lack of significant genotoxic and mutagenic potential. This evidence of safety is supported by the fact that the intake of cinnamyl derivatives as natural components of traditional foods is much greater than their intake as intentionally added flavouring substances.

All members of this group are primary alcohols, aldehydes, or carboxylic acids, or their corresponding esters and acetals. The primary oxygenated functional group is located on a three-carbon saturated or unsaturated (i.e. at the 2,3-position) chain with a benzene ring at the 3 position (i.e., a 3-phenylpropyl or 3-phenyl-2-propenyl group). The aromatic ring also may be substituted with alkyl, alkoxy, or hydroxy substituents.

Studies performed for cinnamyl alcohol, the corresponding aldehyde, two cinnamate esters, two *alpha*-alkyl-substituted cinnamaldehyde derivatives, two alkoxy-substituted cinnamaldehyde derivatives and a mixture of five cinnamyl derivatives, show no evidence of any toxicity at dose levels exceeding the estimated daily per capita intake of the respective cinnamyl derivatives by at least three orders of magnitude.

Oral LD50 values have been reported for 39 of the 55 substances in this group. In rats LD50 values are in the range of 1520 to greater than 5000 mg/kg bw. Esters and acetals formed from the parent alcohol, aldehyde or carboxylic acid are hydrolysed prior to or during or after absorption. Once formed cinnamyl alcohol, cinnamaldehyde and cinnamic acid have all been shown to be rapidly absorbed from the gut, metabolised and excreted primarily in the urine and, to a minor extent, in the faeces. Results of numerous studies indicate that cinnamyl derivatives are absorbed, metabolised and excreted as polar metabolites within 24 hours.

In general, esters containing an aromatic ring system are hydrolysed *in vivo* by classes of enzymes recognised as carboxylesterases or esterases. Acetals are rapidly hydrolysed in acidic medium.

The position and size of ring substituents play a role in the metabolism of cinnamyl derivatives. Cinnamyl derivatives containing *alpha*-methyl substituents participate in *beta*-oxidation and cleavage to yield mainly the corresponding hippuric acid derivative.

Substituents greater than C1 located at the *alpha*- or *beta*- position, to some extent, inhibit *beta*-oxidation. In these cases, there may be direct conjugation of the carboxylic acid with glucuronic acid followed by excretion.

Flavor and Extract Manufacturers' Association FEMA

CITRUS TERPENES

The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

d-Limonene is readily absorbed by inhalation and ingestion. Dermal absorption is reported to be lower than by the inhalation route. d-Limonene is rapidly distributed to different tissues in the body, readily metabolised and eliminated primarily through the urine.

Limonene exhibits low acute toxicity by all three routes in animals. Limonene is a skin irritant in both experimental animals and humans. Limited data are available on the potential to cause eye and respiratory irritation. Autooxidised products of d-limonene have the potential to be skin sensitisers. Limited data are available in humans on the potential to cause respiratory sensitisation. Autooxidation of limonene occurs readily in the presence of light and air forming a variety of oxygenated monocyclic terpenes. Risk of skin sensitisation is high in situations where contact with oxidation products of limonene occurs.

Renal tumours induced by limonene in male rats is thought to be sex and species specific and are not considered relevant to humans. Repeated exposure affects the amount and activity of liver enzymes, liver weight, blood cholesterol levels and bile flow in animals. Increase in liver weight is considered a physiological adaptation as no toxic effects on the liver have been reported. From available data it is not possible to identify a NOAEL for these effects. Limonene is neither genotoxic or teratogenic nor toxic to the reproductive system.

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 constituents, some of which have the potential to cause toxic effects; for example, bergapten (aka 5-methoxysporalen or 5-MOP) is a naturally occurring furanocoumarin (psoralen) in bergamot oil that causes phototoxicity. Under the rules governing cosmetic products in the European Union, citrus-derived ingredients must have furocoumarin content below 1 mg/kg in sun-protection and bronzing products.

Acute toxicity:

The dermal LD50 of undiluted either bitter orange or citrus *reticulata* (tangerine) leaf oil (described as "petitgrain bigarade oil") was reported as greater than 2000 mg/kg in rabbits. The dermal LD50 of undiluted mandarin peel oil (*Citrus reticulata*) was greater than 5000 mg/kg in rabbits

Dermal irritation:

Varying degrees of irritation were observed in animals treated with undiluted citrus *aurantium amara* (bitter orange) flower wax, unreported concentrations of either bitter orange or citrus *reticulata* (tangerine) leaf oil (described as "petitgrain bigarade oil"), or unreported concentrations of mandarin peel oil. In human subjects, no irritation was observed after topical exposure to citrus *aurantium dulcis* (orange) peel wax (100%), bergamot oil (up to 15%), either bitter orange or citrus *reticulata* (tangerine) leaf oil (described as "petitgrain bigarade oil"; up to 8%), lemon oil (up to 20%), or mandarin peel oil (8%).

Ocular irritation:

The eye tolerance of citrus *aurantium amara* (bitter orange) flower wax (> 50%) was tested *in vitro* using the SIRC cell strain. Tolerance was evaluated by measuring cytotoxicity. Negative controls solutions were physiological serum or sample diluent and the positive control solutions were 0.01% to 0.2% SDS. Negligible cytotoxicity was observed.

Sensitisation:

Bitter orange or citrus *reticulata* (tangerine) leaf oil (described as "petitgrain bigarade oil") and mandarin peel oil were not sensitising in human maximization tests. In studies of 250 dermatitic patients, less than 2.5% had positive reactions to bergamot oil, bitter orange oil, lemon oil, or sweet orange oil tested at 2% in paraffin.

In a retrospective study (2001-2010) of professional food handlers in Denmark, 8.5% (16/188) of the patients had positive reactions to orange peel and 7.9% (15/191) of the patients had positive reactions to lemon peel

Phototoxicity and Photosensitisation:

Citrus *aurantium dulcis* (orange) peel wax (100%) was not photosensitising in a human study. Mixed results were observed in non-human and human phototoxicity and photosensitisation studies of diluted and undiluted bergamot oil, either bitter orange or citrus *reticulata* (tangerine) leaf oil (described as "petitgrain bigarade oil"), lime oil, lemon oil, lemon fruit and peel juice, grapefruit oil, mandarin oil, tangerine oil, bitter orange oil, bitter orange peel oil, orange peel, orange mesocarp, and orange fruit. Many of the citrus-derived ingredients contain constituents that are photoactive agents, although those noted to be furocoumarin free tended not to induce photosensitisation.

Phototoxicity and photosensitisation were noted in several patients exposed to bergamot oil or limes/lime juice

Carcinogenicity:

Tumour-promoting activity was observed in mouse skin exposed to essential oils of orange (sweet), lemon, grapefruit, or lime.

Groups of mice received weekly applications of 0.25 ml of the test substances 3 weeks after the application of 9,10-dimethyl-1,2-benzanthracene (DMBA) a tumour initiator. By the fifth week, papillomas were observed in mice exposed to lemon oil, grapefruit oil, and lime oil. Papillomas were observed in the orange oil group by the 12th week. After 33 weeks, 10/20 mice in the lemon oil and lime oil treatment groups and 13/20 mice in the grapefruit oil and orange oil groups had papillomas.

No malignant skin tumours were observed in the orange oil group; treatment was stopped after 42 weeks. Squamous cell carcinomas of the skin were observed in 2 mice from the lemon oil group and 2 mice of the grapefruit oil group between weeks 36 and 55.

Non-dermal tumors during the treatment period were observed in 1 mouse of the orange oil group (a haemangioma of the subcutaneous tissue starting at week 7) and in 1 mouse of the grapefruit oil group (a spindle cell sarcoma of the subcutaneous tissues). No tumours of the internal organs were observed. The survival of all the mice in this experiment was poor due to a very high incidence of renal disease.

Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A **prehapten** is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems.

In the case of prehapten, it is possible to prevent activation outside the body to a certain extent by different measures, e.g. prevention of air exposure during handling and storage of the ingredients and the final product, and by the addition of suitable antioxidants. When antioxidants are used, care should be taken that they will not be activated themselves and thereby form new sensitisers.

Prehapten

Most terpenes with oxidisable allylic positions can be expected to autoxidise on air exposure due to their inherent properties. Depending on the stability of the oxidation products that are formed, a difference in the sensitisation potency of the oxidised terpenes can be seen

Autoxidation is a free radical chain reaction in which hydrogen atom abstraction in combination with addition of oxygen forms peroxy radicals. The reaction shows selectivity for positions where stable radicals can be formed. So far, all fragrance substances that have been investigated with regard to the influence of autoxidation on the allergenic potential, including identification of formed oxidation products, have oxidisable allylic positions that are able to form hydroperoxides and/or hydrogen peroxide as primary oxidation products upon air exposure. Once the hydroperoxides have been formed outside the skin they form specific antigens and act as skin sensitisers. Secondary oxidation products such as aldehydes and epoxides can also be allergenic, thus further increasing the sensitisation potency of the autoxidation mixture. The process of photoactivation may also play a role, but further research is required to establish whether this activation route is currently underestimated in importance due to insufficient knowledge of the true haptens in this context.

It should be noted that activation of substances via air oxidation results in various haptens that might be the same or cross-reacting with other haptens (allergens). The main allergens after air oxidation of linalool and linalyl acetate are the hydroperoxides. If linalyl acetate is chemically hydrolysed outside the skin it can thereafter be oxidised to the same haptens as seen for linalool. A corresponding example is citronellol and citronellyl acetate. In clinical studies, concomitant reactions to oxidised linalool and oxidised linalyl acetate have been observed. Whether these reactions depend on cross-reactivity or are due to exposure to both fragrance substances cannot be elucidated as both have an allergenic effect themselves. Linalool and linalyl acetate are the main components of lavender oil. They autoxidise on air exposure also when present in the essential oil, and form the same oxidation products found in previous studies of the pure synthetic terpenes. Experimental sensitisation studies showed that air exposure of lavender oil increased the sensitisation potency. Patch test results in dermatitis patients showed a connection between positive reactions to oxidised linalool, linalyl acetate and lavender oil.

Prohapten

Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohapten.

In the case of prohapten, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal.

The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin. These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohapten can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity.

QSAR prediction: The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohapten) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohapten. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation.

for cold-pressed oil Citrus terpenes possess low toxicity following ingestion, dermal contact or inhalation. * Florida Chemical Company MSDS

CINNAMALDEHYDE & CITRUS TERPENES

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.

CINNAMALDEHYDE & CITRUS TERPENES

Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and contact dermatitis occur. Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes.

Cases of occupational asthma induced 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.

Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water.

Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16% of patients patch tested for suspected allergic contact dermatitis.

Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a sufficient degree of fragrance contact allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the allergen. As a consequence, symptoms, i.e. allergic contact dermatitis, may occur upon re-exposure to the

fragrance allergen(s) in question. Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or hands. It may affect fitness for work and the quality of life of the individual. Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease. Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure.

Hands: Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema may often be complicated by sensitisation. Fragrance allergy may be a relevant problem in patients with hand eczema; perfumes are present in consumer products to which their hands are exposed. A significant relationship between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe) chronic hand eczema may not be clear.

Axillae Bilateral axillary (underarm) dermatitis 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 dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy.

Face Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of being fragrance allergic.

Irritant reactions (including contact urticaria): Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this. Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported. The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested, but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen.

Pigmentary anomalies: The term "pigmented cosmetic dermatitis" was introduced in 1973 for what had previously been known as melanosia faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil.

Photo-reactions Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon. Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. 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 naso-respiratory tract. 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. In an epidemiological investigation, a significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.

CITRUS TERPENES & PROPANE	No significant acute toxicological data identified in literature search.		
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 available but does not fill the criteria for classification
 ✔ – Data required to make classification available
 ☒ – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
propylene glycol monomethyl ether - alpha isomer	LC50	96	Fish	1005.858mg/L	3
propylene glycol monomethyl ether - alpha isomer	EC50	48	Crustacea	>500mg/L	1
propylene glycol monomethyl ether - alpha isomer	EC50	96	Algae or other aquatic plants	7152.973mg/L	3
propylene glycol monomethyl ether - alpha isomer	EC50	384	Crustacea	227.843mg/L	3
propylene glycol monomethyl ether - alpha isomer	NOEC	96	Fish	=4600mg/L	1
isopropanol	LC50	96	Fish	183.844mg/L	3
isopropanol	EC50	48	Crustacea	12500mg/L	5
isopropanol	EC50	96	Algae or other aquatic plants	993.232mg/L	3

Continued...

Paint/Graffiti Remover #198-1039, 487-2736 (Paint/Graffiti Remover #198-1039, 487-2736)

isopropanol	EC50	384	Crustacea	42.389mg/L	3
isopropanol	NOEC	5760	Fish	0.02mg/L	4
cinnamaldehyde	LC50	96	Fish	12.616mg/L	3
cinnamaldehyde	EC50	96	Algae or other aquatic plants	94.713mg/L	3
citrus terpenes	LC50	96	Fish	1.279mg/L	3
citrus terpenes	EC50	96	Algae or other aquatic plants	0.060mg/L	3
propane	LC50	96	Fish	10.307mg/L	3
propane	EC50	96	Algae or other aquatic plants	32.252mg/L	3
propane	EC50	384	Crustacea	2.462mg/L	3
butane	LC50	96	Fish	5.862mg/L	3
butane	EC50	96	Algae or other aquatic plants	15.346mg/L	3
butane	EC50	384	Crustacea	1.416mg/L	3
iso-butane	LC50	96	Fish	6.706mg/L	3
iso-butane	EC50	96	Algae or other aquatic plants	18.064mg/L	3
iso-butane	EC50	384	Crustacea	1.617mg/L	3

Legend:

Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 - Aquatic Toxicity Data (Estimated) 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

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
propylene glycol monomethyl ether - alpha isomer	LOW (Half-life = 56 days)	LOW (Half-life = 1.7 days)
isopropanol	LOW (Half-life = 14 days)	LOW (Half-life = 3 days)
cinnamaldehyde	LOW	LOW
citrus terpenes	HIGH	HIGH
propane	LOW	LOW
butane	LOW	LOW
iso-butane	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
propylene glycol monomethyl ether - alpha isomer	LOW (BCF = 2)
isopropanol	LOW (LogKOW = 0.05)
cinnamaldehyde	LOW (BCF = 10)
citrus terpenes	HIGH (LogKOW = 5.6842)
propane	LOW (LogKOW = 2.36)
butane	LOW (LogKOW = 2.89)
iso-butane	LOW (BCF = 1.97)

Mobility in soil

Ingredient	Mobility
propylene glycol monomethyl ether - alpha isomer	HIGH (KOC = 1)
isopropanol	HIGH (KOC = 1.06)
cinnamaldehyde	LOW (KOC = 102.4)
citrus terpenes	LOW (KOC = 2899)
propane	LOW (KOC = 23.74)
butane	LOW (KOC = 43.79)
iso-butane	LOW (KOC = 35.04)

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.

Continued...

Paint/Graffiti Remover #198-1039, 487-2736 (Paint/Graffiti Remover #198-1039, 487-2736)

- ▶ 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.
- ▶ Consult State Land Waste Management Authority for disposal.
- ▶ Discharge contents of damaged aerosol cans at an approved site.
- ▶ Allow small quantities to evaporate.
- ▶ **DO NOT incinerate or puncture aerosol cans.**
- ▶ Bury residues and emptied aerosol cans at an approved site.

SECTION 14 TRANSPORT INFORMATION

Labels Required

	
Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG)

UN number	1950
UN proper shipping name	AEROSOLS
Transport hazard class(es)	Class : 2.1 Subrisk : Not Applicable
Packing group	Not Applicable
Environmental hazard	Not Applicable
Special precautions for user	Special provisions : 63 190 277 327 344 Limited quantity : 1000ml

Air transport (ICAO-IATA / DGR)

UN number	1950
UN proper shipping name	Aerosols, flammable; Aerosols, flammable (engine starting fluid)
Transport hazard class(es)	ICAO/IATA Class : 2.1 ICAO / IATA Subrisk : Not Applicable ERG Code : 10L
Packing group	Not Applicable
Environmental hazard	Not Applicable
Special precautions for user	Special provisions : A145A167A802; A1A145A167A802 Cargo Only Packing Instructions : 203 Cargo Only Maximum Qty / Pack : 150 kg Passenger and Cargo Packing Instructions : 203; Forbidden Passenger and Cargo Maximum Qty / Pack : 75 kg; Forbidden Passenger and Cargo Limited Quantity Packing Instructions : Y203; Forbidden Passenger and Cargo Limited Maximum Qty / Pack : 30 kg G; Forbidden

Sea transport (IMDG-Code / GGVSee)

UN number	1950
UN proper shipping name	AEROSOLS
Transport hazard class(es)	IMDG Class : 2.1 IMDG Subrisk : Not Applicable
Packing group	Not Applicable
Environmental hazard	Not Applicable
Special precautions for user	EMS Number : F-D, S-U Special provisions : 63 190 277 327 344 959 Limited Quantities : 1000ml

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER(107-98-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	

ISOPROPANOL(67-63-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

CINNAMALDEHYDE(104-55-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

CITRUS TERPENES(8028-48-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

PROPANE(74-98-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	International Air Transport Association (IATA) Dangerous Goods Regulations - Prohibited List Passenger and Cargo Aircraft

BUTANE(106-97-8.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	International Air Transport Association (IATA) Dangerous Goods Regulations - Prohibited List Passenger and Cargo Aircraft

ISO-BUTANE(75-28-5.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists	International Air Transport Association (IATA) Dangerous Goods Regulations - Prohibited List Passenger and Cargo Aircraft
Australia Inventory of Chemical Substances (AICS)	

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (propylene glycol monomethyl ether - alpha isomer; butane; propane; citrus terpenes; isopropanol; iso-butane)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	N (citrus terpenes)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	N (citrus terpenes)
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Other information

Ingredients with multiple cas numbers

Name	CAS No
cinnamaldehyde	104-55-2, 14371-10-9, 57194-69-1, 132200-89-6, 3839-82-5, 7148-78-9
citrus terpenes	8028-48-6, 94266-47-4

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.

A list of reference resources used to assist the committee may be found at:

www.chemwatch.net

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
 OSF: Odour Safety Factor
 NOAEL :No Observed Adverse Effect Level

Continued...

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

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