

8361 Label and Adhesive Remover (Aerosol) MG Chemicals UK Limited

Version No: A-2.00

Safety data sheet according to REACH Regulation (EC) No 1907/2006, as amended by UK REACH Regulations SI 2019/758

Issue Date: 11/08/2021 Revision Date: 11/08/2021 L.REACH.GB.EN

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

1.1. Product Identifier

Product name 8361			
Synonyms	SDS Code: 8361-a; 8361-140G, 8361-140GCA UFI:UHJ0-40F4-R001-CSAK		
Other means of identification	Label and Adhesive Remover (Aerosol)		

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

Relevant identified uses	Label and adhesive remover
Uses advised against	Not Applicable

1.3. Details of the supplier of the safety data sheet

Registered company name	MG Chemicals UK Limited	MG Chemicals (Head office)	
Address	Heame House, 23 Bilston Street, Sedgely Dudley DY3 1JA United Kingdom	9347 - 193 Street Surrey V4N 4E7 British Columbia Canada	
Telephone	+(44) 1663 362888	+(1) 800-201-8822	
Fax	Not Available	+(1) 800-708-9888	
Website	Not Available	www.mgchemicals.com	
Email	sales@mgchemicals.com	Info@mgchemicals.com	

1.4. Emergency telephone number

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Association / Organisation	Verisk 3E (Access code: 335388)		
Emergency telephone numbers	+(44) 20 35147487		
Other emergency telephone numbers	+(0) 800 680 0425		

SECTION 2 Hazards identification

2.1. Classification of the substance or mixture

Classified according to
GB-CLP Regulation, UK SI
2019/720 and UK SI 2020/1567
[1]

H336 - Specific target organ toxicity - single exposure Category 3 (narcotic effects), H411 - Chronic Aquatic Hazard Category 2, H315 - Skin Corrosion/Irritation Category 2, H317 - Skin Sensitizer Category 1, H304 - Aspiration Hazard Category 1, H222+H229 - Aerosols Category 1

Legend:

1. Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567

2.2. Label elements

Hazard pictogram(s)









Signal word

Danger

Hazard statement(s)

H336	May cause drowsiness or dizziness.
H411	Toxic to aquatic life with long lasting effects.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H304	May be fatal if swallowed and enters airways.
H222+H229	Extremely flammable aerosol. Pressurized container: may burst if heated.

Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P211	Do not spray on an open flame or other ignition source.
P251	Do not pierce or burn, even after use.
P271	Use only a well-ventilated area.
P280	Wear protective gloves and protective clothing.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P331	Do NOT induce vomiting.
P302+P352	IF ON SKIN: Wash with plenty of water.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.
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 to authorised hazardous or special waste collection point in accordance with any local regulation.

2.3. Other hazards

Inhalation and/or ingestion may produce health damage*.

Cumulative effects may result following exposure $\!\!\!\!^\star$.

May produce discomfort of the eyes, respiratory tract and skin*.

Limited evidence of a carcinogenic effect*.

C14-20 aliphatics (<=2% aromatics)	Listed in the Europe Regulation (EU) 2018/1881 Specific Requirements for Endocrine Disruptors
d-limonene	Listed in the Europe Regulation (EC) No 1907/2006 - Annex XVII (Restrictions may apply)

SECTION 3 Composition / information on ingredients

3.1.Substances

See 'Composition on ingredients' in Section 3.2

3.2.Mixtures

1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567	Nanoform Particle Characteristics
1.64742-47-8. 2.265-093-4 265-148-2 265-149-8 3.649-214-00-1 649-221-00- X 649-422-00-2 4.Not Available	54	C14-20 aliphatics (<=2% aromatics) [e]	Specific target organ toxicity - single exposure Category 3 (narcotic effects), Aspiration Hazard Category 1; H336, H304, EUH066 [1]	Not Available
1.29118-24-9 2.Not Available 3.Not Available 4.Not Available	25	1.3.3.3- tetrafluoropropene	Gas under Pressure (Liquefied gas); H280, EUH044 [1]	Not Available
1.5989-27-5 2.227-813-5 3.601-029-00-7 4.Not Available	15	d-limonene	Flammable Liquid Category 3, Skin Corrosion/Irritation Category 2, Skin Sensitizer Category 1, Acute Aquatic Hazard Category 1, Chronic Aquatic Hazard Category 1; H226, H315, H317, H400, H410 [2]	Not Available

1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classified according to GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567	Nanoform Particle Characteristics
1.99-85-4 2.202-794-6 3.Not Available 4.Not Available	2	gamma-terpinene	Flammable Liquid Category 3, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, Skin Sensitizer Category 1, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Specific target organ toxicity - single exposure Category 3 (narcotic effects), Chronic Aquatic Hazard Category 2; H226, H315, H319, H317, H335, H336, H411 [1]	Not Available
1.127-91-3 2.204-872-5 3.Not Available 4.Not Available	0.9	beta-pinene	Flammable Liquid Category 3, Acute Toxicity (Oral, Dermal and Inhalation) Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, Skin Sensitizer Category 1, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Specific target organ toxicity - single exposure Category 3 (narcotic effects), Chronic Aquatic Hazard Category 1; H226, H302+H312+H332, H315, H319, H317, H335, H336, H410, EUH019 [1]	Not Available
1.123-35-3 2.204-622-5 3.Not Available 4.Not Available	0.7	myrcene	Flammable Liquid Category 3, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, Skin Sensitizer Category 1, Reproductive Toxicity Category 2, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Specific target organ toxicity - single exposure Category 3 (narcotic effects), Chronic Aquatic Hazard Category 1; H226, H315, H319, H317, H361f, H335, H336, H410, EUH001, EUH019 [1]	Not Available
1.586-62-9 2.209-578-0 3.Not Available 4.Not Available	0.7	terpinolene	Flammable Liquid Category 3, Skin Sensitizer Category 1, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Aspiration Hazard Category 1, Chronic Aquatic Hazard Category 1; H226, H317, H336, H304, H410, EUH001, EUH019 [1]	Not Available
1.80-56-8 2.201-291-9 3.Not Available 4.Not Available	0.7	alpha-pinene	Flammable Liquid Category 3, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2, Skin Sensitizer Category 1, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Specific target organ toxicity - single exposure Category 3 (narcotic effects), Chronic Aquatic Hazard Category 1; H226, H315, H319, H317, H335, H336, H410, EUH019 [1]	Not Available
1.99-86-5 2.202-795-1 3.Not Available 4.Not Available	0.3	alpha-terpinene	Flammable Liquid Category 3, Acute Toxicity (Oral) Category 4, Skin Sensitizer Category 1, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Chronic Aquatic Hazard Category 1; H226, H302, H317, H336, H410, EUH019 [1]	Not Available
Le	•	Classified by Chemwatch; 2. Classification drawn from GB-CLP Regulation, UK SI 2019/720 and UK SI 2020/1567; 3. Classification drawn from C&L * EU IOELVs available; [e] Substance identified as having endocrine disrupting properties		

SECTION 4 First aid measures

4.1. Description of first aid measures

Eye Contact	If aerosols come in contact with the eyes: 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	If solids or aerosol mists are deposited upon the skin: 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	If aerosols, fumes or combustion products are inhaled: 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	 Not considered a normal route of entry. If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

4.2 Most important symptoms and effects, both acute and delayed

See Section 11

4.3. Indication of any immediate medical attention and special treatment needed

For petroleum distillates

- In case of ingestion, gastric lavage with activated charcoal can be used promptly to prevent absorption decontamination (induced emesis or lavage) is controversial and should be considered on the merits of each individual case; of course the usual precautions of an endotracheal tube should be considered prior to lavage, to prevent aspiration.
- Individuals intoxicated by petroleum distillates should be hospitalized immediately, with acute and continuing attention to neurologic and cardiopulmonary function.
- Positive pressure ventilation may be necessary.
- Acute central nervous system signs and symptoms may result from large ingestions of aspiration-induced hypoxia.
- After the initial episode, individuals should be followed for changes in blood variables and the delayed appearance of pulmonary oedema and chemical pneumonitis. Such patients should be followed for several days or weeks for delayed effects, including bone marrow toxicity, hepatic and renal impairment Individuals with chronic pulmonary disease will be more seriously impaired, and recovery from inhalation exposure may be complicated.

- · Gastrointestinal symptoms are usually minor and pathological changes of the liver and kidneys are reported to be uncommon in acute intoxications.
- · Chlorinated and non-chlorinated hydrocarbons may sensitize the heart to epinephrine and other circulating catecholamines so that arrhythmias may occur.Careful consideration of this potential adverse effect should precede administration of epinephrine or other cardiac stimulants and the selection of bronchodilators.

BP America Product Safety & Toxicology Department

Treat symptomatically.

for intoxication due to Freons/ Halons;

- A: Emergency and Supportive Measures
- ▶ Maintain an open airway and assist ventilation if necessary
- Treat coma and arrhythmias if they occur. Avoid (adrenaline) epinephrine or other sympathomimetic amines that may precipitate ventricular arrhythmias. Tachyarrhythmias caused by increased myocardial sensitisation may be treated with propranolol, 1-2 mg IV or esmolol 25-100 microgm/kg/min IV.
- Monitor the ECG for 4-6 hours
- B: Specific drugs and antidotes:
- ▶ There is no specific antidote

C: Decontamination

- Inhalation; remove victim from exposure, and give supplemental oxygen if available.
- Ingestion; (a) Prehospital: Administer activated charcoal, if available. DO NOT induce vomiting because of rapid absorption and the risk of abrupt onset CNS depression. (b) Hospital: Administer activated charcoal, although the efficacy of charcoal is unknown. Perform gastric lavage only if the ingestion was very large and recent (less than 30 minutes)
- D: Enhanced elimination:
- ▶ There is no documented efficacy for diuresis, haemodialysis, haemoperfusion, or repeat-dose charcoal.

POISONING and DRUG OVERDOSE, Californian Poison Control System Ed. Kent R Olson; 3rd Edition

- ▶ Do not administer sympathomimetic drugs unless absolutely necessary as material may increase myocardial irritability.
- No specific antidote
- Because rapid absorption may occur through lungs if aspirated and cause systematic effects, the decision of whether to induce vomiting or not should be made by an attending physician.
- If lavage is performed, suggest endotracheal and/or esophageal control.
- ▶ Danger from lung aspiration must be weighed against toxicity when considering emptying the stomach.
- ▶ Treatment based on judgment of the physician in response to reactions of the patient

SECTION 5 Firefighting measures

5.1. Extinguishing media

SMALL FIRE

Water spray, dry chemical or CO2

LARGE FIRE:

► Water spray or fog.

5.2. Special hazards arising from the substrate or mixture

Fire Incompatibility

Fire Fighting

Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

5.3. Advice for firefighters

- 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.
- b 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.

Combustion products include:

carbon dioxide (CO2)

- 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).

carbon monoxide (CO)

hydrogen fluoride

other pyrolysis products typical of burning organic material.

Contains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions.

WARNING: Long standing in contact with air and light may result in the formation

of potentially explosive peroxides.

Vented gas is more dense than air and may collect in pits, basements.

SECTION 6 Accidental release measures

Fire/Explosion Hazard

6.1. Personal precautions, protective equipment and emergency procedures

See section 8

6.2. Environmental precautions

See section 12

6.3. Methods and material for containment and cleaning up

Minor Spills

Environmental hazard - contain spillage.

- 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.

Environmental hazard - contain spillage.

Chemical Class: aliphatics, halogenated

For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
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LAND SPILL - SMALL

cross-linked polymer - particulate	1	shovel	shovel	R, W, SS
cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT
wood fiber - pillow	2	throw	pitchfork	R, P, DGC, RT
treated wood fibre - particulate	2	shovel	shovel	R, W, DGC
sorbent clay - particulate	3	shovel	shovel	R, I, P
foamed glass - pillow	3	throw	pitchfork	R, P, DGC, RT

LAND SPILL - MEDIUM

cross-linked polymer - particulate	1	blower	skiploader	R,W, SS
cross-linked polymer - pillow	2	throw	skiploader	R, DGC, RT
sorbent clay - particulate	3	blower	skiploader	R, I, P
polypropylene - particulate	3	blower	skiploader	W, SS, DGC
foamed glass - pillow	3	throw	skiploader	R, P, DGC, RT
expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC

Legend

Major Spills

DGC: Not effective where ground cover is dense

R: Not reusable

I: Not incinerable

P: Effectiveness reduced when rainy

RT:Not effective where terrain is rugged

SS: Not for use within environmentally sensitive sites

W: Effectiveness reduced when windy

Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;

R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988

CARE: Absorbent materials wetted with occluded oil must be moistened with water as they may auto-oxidize, become self heating and ignite. Some oils slowly oxidise when spread in a film and oil on cloths, mops, absorbents may autoxidise and generate heat, smoulder, ignite and burn. In the workplace oily rags should be collected and immersed in water.

- Clear area of all unprotected personnel and move upwind.
- Alert Emergency Authority and advise them of the location and nature of hazard.
- May be violently or explosively reactive.
- Wear full body clothing with breathing apparatus.
- Prevent by any means available, spillage from entering drains and water-courses.
- ► Consider evacuation.
- Shut off all possible sources of ignition and increase ventilation.
- No smoking or naked lights within area.
- ▶ Use extreme caution to prevent violent reaction.
- Stop leak only if safe to so do.
- ▶ Water spray or fog may be used to disperse vapour.
- ► DO NOT enter confined space where gas may have collected.
- ▶ Keep area clear until gas has dispersed.
- Remove leaking cylinders to a safe place.
- Fit vent pipes. Release pressure under safe, controlled conditions
- Burn issuing gas at vent pipes.
- ▶ DO NOT exert excessive pressure on valve; DO NOT attempt to operate damaged valve. 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.

6.4. Reference to other sections

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

SECTION 7 Handling and storage

Safe handling

7.1. Precautions for safe handling

The conductivity of this material may make it a static accumulator., A liquid is typically considered nonconductive if its conductivity is below 100 pS/m and is considered semi-conductive if its conductivity is below 10 000 pS/m., Whether a liquid is nonconductive or semi-conductive, the precautions are the same., A number of factors, for example liquid temperature, presence of contaminants, and anti-static additives can greatly influence the conductivity of a liquid.

- 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.
- DO NOT allow clothing wet with material to stay in contact with skin

Fire and explosion protection

See section 5

Consider storage under inert gas

- ▶ 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.

7.2. Conditions for safe storage, including any incompatibilities

Suitable container

Storage incompatibility

Other information

- Aerosol dispenser.
- Check that containers are clearly labelled.

d-Limonene:

- forms unstable peroxides in storage, unless inhibited; may polymerise
- reacts with strong oxidisers and may explode or combust
- 🕨 is incompatible with strong acids, including acidic clays, peroxides, halogens, vinyl chloride and iodine pentafluoride
- flow or agitation may generate electrostatic charges due to low conductivity

As a general rule, hydrofluorocarbons tend to be flammable unless they contain more fluorine atoms than hydrogen atoms.

- The various oxides of nitrogen and peroxyacids may be dangerously reactive in the presence of alkenes. BRETHERICK L.: Handbook of Reactive Chemical Hazards
- Avoid reaction with strong Lewis or mineral acids
- ▶ Reaction with halogens requires carefully controlled conditions.
- Free radical initiators should be avoided.

HAZARD

- ▶ Although anti-oxidants may be present, in the original formulation, these may deplete over time as they come into contact with air.
- Rags wet / soaked with unsaturated hydrocarbons / drying oils may auto-oxidise; generate heat and, in-time, smoulder and ignite. This is especially the case where oil-soaked materials are folded, bunched, compressed, or piled together this allows the heat to accumulate or even accelerate the reaction
- Oily cleaning rags should be collected regularly and immersed in water, or spread to dry in safe-place away from direct sunlight.or stored, immersed, in solvents in suitably closed containers.
- ► Haloalkenes are highly reactive.
- Some of the more lightly substituted lower members are highly flammable; many members of the group are peroxidisable and polymerisable.
- Avoid reaction or contact with potassium or its alloys although apparently stable on contact with a wide rage of halocarbons, reaction products may be shock-sensitive and may explode with great violence on light impact. Severity generally increases with the degree of halocarbon substitution and potassium-sodium alloys give extremely sensitive mixtures.

BRETHERICK L.: Handbook of Reactive Chemical Hazards

- Avoid reaction with metal halides and active metals, eg. sodium (Na), potassium (K), calcium (Ca), zinc (Zn), powdered aluminium (Al), magnesium (Mg) and magnesium alloys.
- Avoid contact with rubber, and plastics such as methacrylate polymers, polyethylene and polystyrene

Terpenoids and terpenes, are generally unsaturated, are thermolabile, are often volatile and may be easily oxidised or hydrolysed depending on their respective structure.

Terpenoids are subject to autoxidation. Autoxidation is any oxidation that occurs in open air or in presence of oxygen (and sometimes UV radiation) and forms peroxides and hydroperoxides.

Though autoxidation has been particularly investigated in the field of fatty oils, it also plays a most crucial part for terpenoid deterioration. Although virtually all types of organic materials can undergo air oxidation, certain types are particularly prone to autoxidation, including

unsaturated compounds that have allylic or benzylic hydrogen atoms (C6H5CH2-); these materials are converted to hydroperoxides by autoxidation. Promoted by heat, catalytic quantities of redox-reactive metals, and exposure to light, autoxidation may result in the formation of explosive peroxides which may become explosive upon concentration.

As a rule, however, primary autoxidation products such as hydroperoxides eventually break down during advanced stages of oxidation depending on their individual stability. Thereby they give rise to a range of stable oxidised secondary products such as mono- to polyvalent alcohols, aldehydes, ketones, epoxides, peroxides, or acids as well as highly viscous, often oxygen-bearing polymers. Light, heat, or increasing acidity often promote this breakdown.

Compounds rich in allylic hydrogen atoms (2HC=CHCH2-R), found in most terpenoids, make up the most probable targets for autoxidation. Several terpenoids (typically oxygen containing derivatives) are saturated and do not react in a similar fashion to their unsaturated congeners. Thermolabile terpenoids, especially mere terpenes and aldehydes, are susceptible to rearrangement processes at elevated temperatures. Terpenic conversion reactions, upon heating, have been reported both for isolated compounds as well as for essential oils.(which tend to be rich in mono-, and sesqui-terpenes.

Mono-, bi-, or tricyclic mono- terpenoids (those containing two isoprene units, dienes) and sesquiterpenoids (with three isoprene units, trienes) of different chemical classes, such as hydrocarbons, ketones, alcohols, oxides, aldehydes, phenols, or esters, make up the major part in essential oils.

Electron-donating groups and increasing alkyl substitution contribute to a stronger carbon-peroxide bond through a hyperconjugative effect, thus leading to more stable and subsequently built-up hydroperoxides.

Some oxygen-bearing terpenoids such as menthol, eucalyptol (1,8-cineol), and menthone do not form hydroperoxides upon oxidation but are directly converted into ketones, acids, and aldehydes. None of these are unsaturated compounds.

Due to their low volatility, diterpenes (with four isoprenes, tetraenes) are barely encountered in genuine essential oils obtained by distillation, while tri- and higher terpenoids such as sterols or carotenoids are only present in the nonvolatile fractions such as plant resins or gums and will remain in the residue

Aging processes generally come along with a more or less pronounced quality loss In addition to the frequent development of unpleasant and often pungent flavours, shifting colors such as the formation of a yellow staining or changes in consistency up to resinification have been reported both upon degradation of single terpenoids as well as of essential oils.

- The interaction of alkenes and alkynes with nitrogen oxides and oxygen may produce explosive addition products; these may form at very low temperatures and explode on heating to higher temperatures (the addition products from 1,3-butadiene and cyclopentadiene form rapidly at -150 C and ignite or explode on warming to -35 to -15 C). These derivatives ('pseudo- nitrosites') were formerly used to characterise terpene hydrocarbons.
- Exposure to air must be kept to a minimum so as to limit the build-up of peroxides which will concentrate in bottoms if the product is distilled. The product must not be distilled to dryness if the peroxide concentration is substantially above 10 ppm (as active oxygen) since explosive decomposition may occur. Distillate must be immediately inhibited to prevent peroxide formation. The effectiveness of the antioxidant is limited once the peroxide levels exceed 10 ppm as active oxygen. Addition of more inhibitor at this point is generally ineffective. Prior to distillation it is recommended that the product should be washed with aqueous ferrous ammonium sulfate to destroy peroxides; the washed product should be immediately re-inhibited.
- A range of exothermic decomposition energies for double bonds is given as 40-90 kJ/mol. The relationship between energy of decomposition and processing hazards has been the subject of discussion; it is suggested that values of energy released per unit of mass, rather than on a molar basis (J/g) be used in the assessment. For example, in 'open vessel processes' (with man-hole size openings, in an industrial setting), substances with exothermic decomposition energies below 500 J/g are unlikely to present a danger, whilst those in 'closed vessel processes' (opening is a safety valve or bursting disk) present some danger where the decomposition energy exceeds 150 J/g. BRETHERICK: Handbook of Reactive Chemical Hazards. 4th Edition
- The reaction of ozone with alkenes is believed to proceed *via* the formation of a vibrationally excited Primary Ozonide (POZ) which falls apart to give a vibrationally excited Criegee Intermediate (CI) The CI can decompose to give OH radicals, or be stabilised. This may be of relevance in atmospheric chemistry.
- · Violent explosions at low temperatures in ammonia synthesis gas units have been traced to the addition products of dienes and nitrogen dioxide
 - Compressed gases may contain a large amount of kinetic energy over and above that potentially available from the energy of reaction produced by the gas in chemical reaction with other substances

7.3. Specific end use(s)

See section 1.2

SECTION 8 Exposure controls / personal protection

8.1. Control parameters

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
C14-20 aliphatics (<=2% aromatics)	Dermal 2.91 mg/kg bw/day (Systemic, Chronic) Inhalation 16.4 mg/m³ (Systemic, Chronic) Inhalation 5 002.67 mg/m³ (Systemic, Acute) Dermal 1.25 mg/kg bw/day (Systemic, Chronic) * Inhalation 4.85 mg/m³ (Systemic, Chronic) * Oral 1.25 mg/kg bw/day (Systemic, Chronic) * Inhalation 3 001.6 mg/m³ (Systemic, Acute) *	17 g/kg food (Oral)
1,3,3,3-tetrafluoropropene	Inhalation 3 902 mg/m³ (Systemic, Chronic) Inhalation 830 mg/m³ (Systemic, Chronic) *	0.1 mg/L (Water (Fresh)) 1 mg/L (Water (Marine))
d-limonene	Dermal 9.5 mg/kg bw/day (Systemic, Chronic) Inhalation 66.7 mg/m³ (Systemic, Chronic) Dermal 4.8 mg/kg bw/day (Systemic, Chronic) * Inhalation 16.6 mg/m³ (Systemic, Chronic) * Oral 4.8 mg/kg bw/day (Systemic, Chronic) *	14 μg/L (Water (Fresh)) 1.4 μg/L (Water - Intermittent release) 3.85 mg/kg sediment dw (Sediment (Fresh Water)) 0.385 mg/kg sediment dw (Sediment (Marine)) 0.763 mg/kg soil dw (Soil) 1.8 mg/L (STP) 133 mg/kg food (Oral)
gamma-terpinene	Dermal 0.833 mg/kg bw/day (Systemic, Chronic) Inhalation 2.939 mg/m³ (Systemic, Chronic) Dermal 0.417 mg/kg bw/day (Systemic, Chronic) * Inhalation 0.725 mg/m³ (Systemic, Chronic) * Oral 0.417 mg/kg bw/day (Systemic, Chronic) *	0.003 mg/L (Water (Fresh)) 0 mg/L (Water - Intermittent release) 0.49 mg/kg sediment dw (Sediment (Fresh Water)) 0.049 mg/kg sediment dw (Sediment (Marine)) 0.423 mg/kg soil dw (Soil) 10 mg/L (STP)
beta-pinene	Dermal 0.8 mg/kg bw/day (Systemic, Chronic) Inhalation 5.69 mg/m³ (Systemic, Chronic) Dermal 54 μg/cm² (Local, Chronic)	1.004 μg/L (Water (Fresh)) 0.1 μg/L (Water - Intermittent release) 5.02 (Water (Marine))

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
	Dermal 0.3 mg/kg bw/day (Systemic, Chronic) * Inhalation 1 mg/m³ (Systemic, Chronic) * Oral 0.3 mg/kg bw/day (Systemic, Chronic) * Dermal 27 µg/cm² (Local, Chronic) *	0.337 mg/kg sediment dw (Sediment (Fresh Water)) 0.034 mg/kg sediment dw (Sediment (Marine)) 0.067 mg/kg soil dw (Soil) 3.26 mg/L (STP) 13.1 mg/kg food (Oral)
terpinolene	Dermal 0.52 mg/kg bw/day (Systemic, Chronic) Inhalation 3.6 mg/m³ (Systemic, Chronic) Dermal 44 µg/cm² (Local, Chronic) Dermal 0.26 mg/kg bw/day (Systemic, Chronic) * Inhalation 0.9 mg/m³ (Systemic, Chronic) * Oral 0.26 mg/kg bw/day (Systemic, Chronic) *	0.001 mg/L (Water (Fresh)) 0 mg/L (Water - Intermittent release) 0.013 mg/L (Water (Marine)) 0.145 mg/kg sediment dw (Sediment (Fresh Water)) 0.015 mg/kg sediment dw (Sediment (Marine)) 0.016 mg/kg soil dw (Soil) 0.2 mg/L (STP) 10.31 mg/kg food (Oral)
alpha-pinene	Dermal 0.132 mg/kg bw/day (Systemic, Chronic) Inhalation 0.933 mg/m³ (Systemic, Chronic) Dermal 161 µg/cm² (Local, Chronic) Dermal 0.134 mg/kg bw/day (Systemic, Chronic) * Inhalation 0.467 mg/m³ (Systemic, Chronic) * Oral 0.134 mg/kg bw/day (Systemic, Chronic) *	0 mg/L (Water (Fresh)) 0 mg/L (Water - Intermittent release) 0.003 mg/L (Water (Marine)) 0.03 mg/kg sediment dw (Sediment (Fresh Water)) 0.003 mg/kg sediment dw (Sediment (Marine)) 0.003 mg/kg soil dw (Soil) 0.2 mg/L (STP) 8.76 mg/kg food (Oral)
alpha-terpinene	Dermal 0.833 mg/kg bw/day (Systemic, Chronic) Inhalation 2.939 mg/m³ (Systemic, Chronic) Dermal 0.417 mg/kg bw/day (Systemic, Chronic) * Inhalation 0.725 mg/m³ (Systemic, Chronic) * Oral 0.417 mg/kg bw/day (Systemic, Chronic) *	0.002 mg/L (Water (Fresh)) 0 mg/L (Water - Intermittent release) 0.017 mg/L (Water (Marine)) 0.196 mg/kg sediment dw (Sediment (Fresh Water)) 0.02 mg/kg sediment dw (Sediment (Marine)) 0.023 mg/kg soil dw (Soil) 0.1 mg/L (STP) 8.333 mg/kg food (Oral)

^{*} Values for General Population

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Not Available						

Not Applicable

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
C14-20 aliphatics (<=2% aromatics)	1,100 mg/m3	1,800 mg/m3	40,000 mg/m3
1,3,3,3-tetrafluoropropene	1,400 ppm	Not Available	Not Available
d-limonene	15 ppm	67 ppm	170 ppm
alpha-pinene	60 ppm	120 ppm	1,500 ppm

Ingredient	Original IDLH	Revised IDLH
C14-20 aliphatics (<=2% aromatics)	2,500 mg/m3	Not Available
1,3,3,3-tetrafluoropropene	Not Available	Not Available
d-limonene	Not Available	Not Available
gamma-terpinene	Not Available	Not Available
beta-pinene	Not Available	Not Available
myrcene	Not Available	Not Available
terpinolene	Not Available	Not Available
alpha-pinene	Not Available	Not Available
alpha-terpinene	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
d-limonene	E	≤ 0.1 ppm
gamma-terpinene	E	≤ 0.1 ppm
beta-pinene	E	≤ 0.1 ppm
myrcene	E	≤ 0.1 ppm
terpinolene	D	> 0.1 to ≤ 1 ppm
alpha-pinene	E	≤ 0.1 ppm

Notes:

Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit	
alpha-terpinene	Е	≤ 0.1 ppm	
Notes:	Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.		

MATERIAL DATA

Fragrance substance with is an established contact allergen in humans.

Scientific Committee on Consumer Safety SCCS OPINION on Fragrance allergens in cosmetic products 2012

IFRA Restricted Fragrance Substance

The International Fragrance Association (IFRA) Standards form the basis for the globally accepted and recognized risk management system for the safe use of fragrance ingredients and are part of the IFRA Code of Practice. This is the self-regulating system of the industry, based on risk assessments carried out by an independent Expert Panel. for d-Limonene:

CEL TWA: 30 ppm, 165.6 mg/m3 (compare WEEL-TWA*)

(CEL = Chemwatch Exposure Limit)

A Workplace Environmental Exposure Level* has been established by AIHA (American Industrial Hygiene Association) who have produced the following rationale:

d-Limonene is not acutely toxic. In its pure form it is not a sensitiser but is irritating to the skin. Although there is clear evidence of carcinogenicity in male rats, the effect has been attributed to an alpha-2u-globin (a2u-G) renal toxicity which is both species and gender specific. Humans do not synthesise a2u-G, and metabolism studies indicate that 75% to 95% of d-limonene is excreted in 2-3 days with different metabolites identified between humans and rats. In a 2-year study, liver effects were noted in male mice at 500 mg/kg and reduced survival was noted in female rats at 600 mg/kg. The no observable effect levels (NOELs) were 250 and 300 mg/kg, respectively. A WEEL of 30 ppm is recommended to protect against these effects.

May act as a simple asphyxiants; these are gases which, when present in high concentrations, reduce the oxygen content in air below that required to support breathing, consciousness and life; loss of consciousness, with death by suffocation may rapidly occur in an oxygen deficient atmosphere.

CARE: Most simple asphyxiants are odourless or possess low odour and there is no warning on entry into an oxygen deficient atmosphere. If there is any doubt, oxygen content can be checked simply and quickly. It may not be appropriate to only recommend an exposure standard for simple asphyxiants rather it is essential that sufficient oxygen be maintained. Air normally has 21 percent oxygen by volume, with 18 percent regarded as minimum under normal atmospheric pressure to maintain consciousness / life. At pressures significantly higher or lower than normal atmospheric pressure, expert guidance should be sought.

NOTE N: The classification as a carcinogen need not apply if the full refining history is known and it can be shown that the substance from which it is produced is not a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI.

European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

8.2. Exposure controls

Care: Atmospheres in bulk storages and even apparently empty tanks may be hazardous by oxygen depletion. Atmosphere must be checked before entry.

Requirements of State Authorities concerning conditions for tank entry must be met. Particularly with regard to training of crews for tank entry; work permits; sampling of atmosphere; provision of rescue harness and protective gear as needed

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are:

Process controls which involve changing the way a job activity or process is done to reduce the risk.

Enclosure and/or isolation of emission source which keeps a selected hazard 'physically' away from the worker and ventilation that strategically 'adds' and 'removes' air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.

Employers may need to use multiple types of controls to prevent employee overexposure.

General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection.

Provide adequate ventilation in warehouse or closed storage areas.

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.

8.2.1. Appropriate engineering controls

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.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 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.

8.2.2. Personal protection











Eve and face protection

- 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 DO NOT wear contact lens Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lens 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] Skin protection See Hand protection below NOTE: The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. No special equipment needed when handling small quantities. Hands/feet protection OTHERWISE: 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 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. BRETHERICK: Handbook of Reactive Chemical Hazards. No special equipment needed when handling small quantities. Other protection

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:

OTHERWISE:

• Overalls.

Skin cleansing cream.Evewash unit.

► Do not spray on hot surfaces.

8361 Label and Adhesive Remover (Aerosol)

Material	СРІ
NITRILE	A
PVA	A
VITON	A

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

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

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	A-AUS / Class1	-
up to 50	1000	-	A-AUS / Class 1
up to 50	5000	Airline *	-
up to 100	5000	-	A-2
up to 100	10000	-	A-3
100+			Airline**

- * Continuous Flow ** Continuous-flow or positive pressure demand A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)
- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used
- Generally not applicable.

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.

8.2.3. Environmental exposure controls

See section 12

SECTION 9 Physical and chemical properties

9.1. Information on basic physical and chemical properties

Appearance	Colourless		
Physical state	Liquid	Relative density (Water = 1)	0.83
Odour	Not Available	Partition coefficient n-octanol / water	4.2
Odour threshold	Not Available	Auto-ignition temperature (°C)	237
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	<20.5
Initial boiling point and boiling range (°C)	>177	Molecular weight (g/mol)	Not Available
Flash point (°C)	48	Taste	Not Available
Evaporation rate	Not Available BuAC = 1	Explosive properties	Not Available
Flammability	Flammable.	Oxidising properties	Not Available
Upper Explosive Limit (%)	6.1	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	0.7	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	0.2	Gas group	Not Available
Solubility in water	Partly miscible	pH as a solution (%)	Not Available
Vapour density (Air = 1)	4.7	VOC g/L	Not Available
Nanoform Solubility	Not Available	Nanoform Particle Characteristics	Not Available
Particle Size	Not Available		

9.2. Other information

Not Available

SECTION 10 Stability and reactivity

10.1.Reactivity	See section 7.2
10.2. Chemical stability	Elevated temperatures. Presence of open flame. Product is considered stable. Hazardous polymerisation will not occur.
10.3. Possibility of hazardous reactions	See section 7.2
10.4. Conditions to avoid	See section 7.2
10.5. Incompatible materials	See section 7.2
10.6. Hazardous decomposition products	See section 5.3

SECTION 11 Toxicological information

11.1. Information on toxicological effects

The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.

Inhaled

Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.

Exposure to high concentrations of fluorocarbons may produce cardiac arrhythmias or cardiac arrest due sensitisation of the heart to adrenalin or noradrenalin. Deaths associated with exposures to fluorocarbons (specifically halogenated aliphatics) have occurred in occupational settings and in inhalation of bronchodilator drugs.

Bronchospasm consistently occurs in human subjects inhaling fluorocarbons. At a measured concentration of 1700 ppm of one of the commercially available aerosols there is a biphasic change in ventilatory capacity, the first reduction occurring within a few minutes and the second delayed up to 30 minutes. Most subjects developed bradycardia (reduced pulse rate).

Bradycardia is encountered in dogs when administration is limited to upper respiratory tract (oropharyngeal and nasal areas). Cardiac arrhythmias can be experimentally induced in animals (species dependency is pronounced with dogs and monkeys requiring lesser amounts of fluorocarbon FC-11 than rats or mice). Sensitivity is increased by injection of adrenalin or cardiac ischaemia/necrosis or pulmonary thrombosis/bronchitis. The cardiotoxic effects of the fluorocarbons originate from irritation of the respiratory tract which in turn reflexively influences the heart rate (even prior to absorption of the fluorocarbon) followed by direct depression of the heart after absorption. Exposure to fluorocarbon thermal decomposition products may produce flu-like symptoms including chills, fever, weakness, muscular aches, headache, chest discomfort, sore throat and dry cough. Complete recovery usually occurs within 24 hours of exposure.

High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.

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.

Symptoms of asphyxia (suffocation) may include headache, dizziness, shortness of breath, muscular weakness, drowsiness and ringing in the ears. If the asphyxia is allowed to progress, there may be nausea and vomiting, further physical weakness and unconsciousness and, finally, convulsions, coma and death. Significant concentrations of the non-toxic gas reduce the oxygen level in the air. As the amount of oxygen is reduced from 21 to 14 volume %, the pulse rate accelerates and the rate and volume of breathing increase. The ability to maintain attention and think clearly is diminished and muscular coordination is somewhat disturbed. As oxygen decreases from 14-10% judgement becomes faulty; severe injuries may cause no pain. Muscular exertion leads to rapid fatigue. Further reduction to 6% may produce nausea and vomiting and the ability to move may be lost. Permanent brain damage may result even after resuscitation at exposures to this lower oxygen level. Below 6% breathing is in gasps and convulsions may occur. Inhalation of a mixture containing no oxygen may result in unconsciousness from the first breath and death will follow in a few minutes.

WARNING: Intentional misuse by concentrating/inhaling contents may be lethal.

Accidental ingestion of the material may be damaging to the health of the individual.

Not normally a hazard due to physical form of product.

Considered an unlikely route of entry in commercial/industrial environments

Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosal ulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea and vomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injury may produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Light aromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration into the lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage.

Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.

Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).

Ingestion

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.

The material may accentuate any pre-existing dermatitis condition

Spray mist may produce discomfort

Skin Contact

In common with other halogenated aliphatics, fluorocarbons may cause dermal problems due to a tendency to remove natural oils from the skin causing irritation and the development of dry, sensitive skin. They do not appear to be appreciably absorbed.

Open cuts, abraded or irritated skin should not be exposed to this material

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.

The liquid may be miscible with fats or oils and may degrease the skin, producing a skin reaction described as non-allergic contact dermatitis. The material is unlikely to produce an irritant dermatitis as described in EC Directives.

Eye

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. 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.

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...

Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may also result. The aromatic fraction may produce irritation and lachrymation.

Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population.

Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.

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.

Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.

Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers. Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.

Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.

Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.

Halogenated oxiranes may arise following epoxidation of haloalkenes.

The metabolism of haloethylenes by microsomal oxidation leading to epoxide formation across the double bond has been proposed. The resulting oxiranes are highly reactive and may covalently bind to nucleic acids leading to mutations and possible cancers A measure of such potential carcinogenicity is the development of significant preneoplastic foci in livers of treated rats.

The carcinogenicity of halogenated oxiranes may lie in the reactivity of an epoxide intermediate. It is reported that 1,1-dichloroethylene, vinyl chloride, trichloroethylene, tetrachloroethylene and chloroprene, for example, are carcinogens in vivo - this may be a consequence of oxirane formation.

Symmetrically substituted oxiranes such as 1,2-dichloroethylene and 1,1,2-2-tetrachloroethylene are more stable and less mutagenic than unsymmetrical chlorinated oxiranes such as 1,1-dichloroethylene, 1,1,2-trichloroethylene and monochloroethylene (vinyl chloride). The carcinogenicity of 1,1-dichloroethylene has primarily been associated with inhalation exposure while that of vinyl chloride, trichloroethylene and tetrachloroethylene occurs following exposure by both inhalation and oral routes. National Toxicology Program Toxicity Report Series Number 55: April 2002

Various studies report an association between cancer and industrial exposure to tetrachloroethylene; IARC concluded that this evidence is sufficient to assign appropriate warnings. Similar warnings have been issued by IARC for vinyl fluoride. Similarly vinyl bromide exhibited neoplastic and tumourigenic activity in rats exposed by inhalation and is classified by various bodies as potentially carcinogenic. Substances such as chloroprene (2-chloro-1,3-butadiene), are reported to produce an increased frequency of chromosomal aberrations in the lymphocytes of Russian workers. Russian epidemiological studies also suggest an increased incidence of skin and lung cancer following exposure to chloroprene, a result which is not supported by other studies.

Generally speaking, the monohalogenated substances exhibit higher carcinogenic potential than their dihalogenated counterparts. Whether additional substitution lessens such hazard is conjectural. Tetrafluoroethylene, for example, produced clear evidence of carcinogenic activity in a two-year inhalation study in rats and mice. National Toxicology Program Technical Report Series 450, April 1997

Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses.

Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.

Hydrocarbon solvents are liquid hydrocarbon fractions derived from petroleum processing streams, containing only carbon and hydrogen atoms, with carbon numbers ranging from approximately C5-C20 and boiling between approximately 35-370 deg C. Many of the hydrocarbon solvents have complex and variable compositions with constituents of 4 types, alkanes (normal paraffins, isoparaffins, and cycloparaffins) and aromatics (primarily alkylated one- and two-ring species). Despite the compositional complexity, most hydrocarbon solvent constituents have similar toxicological properties, and the overall toxicological hazards can be characterized in generic terms. Hydrocarbon solvents can cause chemical pneumonitis if aspirated into the lung, and those that are volatile can cause acute CNS effects and/or ocular and respiratory irritation at exposure levels exceeding occupational recommendations. Otherwise, there are few toxicologically important effects. The exceptions, n-hexane and naphthalene, have unique toxicological properties

Animal studies:

No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) was observed in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar

naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of a alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human. Principal route of occupational exposure to the gas is by inhalation.

On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

In the presence of air, a number of common flavour and fragrance chemicals can form peroxides surprisingly fast. Antioxidants can in most cases minimise the oxidation.

Fragrance terpenes are generally easily oxidised in air. Non-oxidised limonene, linalool and caryophyllene turned out to be very weak sensitizers, however after oxidation limonene hydroperoxide and linalool hydroperoxide are strong sensitizers. Of the patients tested 2.6% showed positive reaction to oxidised limonene, 1.3% to oxidised linalool, 1.1% to linalool hydroperoxide, 0.5% to oxidised caryophyllene, while testing with caryophyllene oxide and oxidised myrcene resulted in few positive patch tests. 2/3 of the patients reacting positive to oxidised terpenes had fragrance related contact allergy and/or positive history for adverse reactions to fragrances.

Chronic

As well as the hydroperoxides produced by linalol, limonene and delta-3-carene other oxidation and resinification effects progressively causes other fairly major changes in essential oil quality over time. Autoxidation of fragrance terpenes contributes greatly to fragrance allergy, which emphasizes the need of testing with compounds that patients are actually exposed to and not only with the ingredients originally applied in commercial formulations.

Hydroperoxides of d-limonene are potent contact allergens when studied in guinea pigs. They may result when d-limonene is unstabilised against oxidation, or upon prolonged standing at room temperature and/ or upon exposure to light, or when stabiliser levels diminish. The two major hydroperoxides in auto-oxidised d-limonene, are cis- and trans- limonene-2-hydroperoxide (2-hydroperoxy-p-mentha-6,8-diene). In photo-oxidised d-limonene, they represent a minor fraction. Hydroperoxides may bind to proteins of the skin to make antigens either via a radical mechanism or after reactions to give epoxides. The cross-reactivity between the epoxide limonene-1,2-oxide, a potent contact allergen, and the hydroperoxides is NOT significant, indicating different mechanisms of sensitisation.

d-Limonene was considered to be weakly carcinogenic for the mouse fore-stomach epithelium, but not tumour producing. In 13-week and 2-year gavage-studies, male rats showed a range of compound-related kidney lesions including exacerbation of age-related nephropathy, mineralisation in the renal medulla, hyperplasia of the transitional epithelium overlying the renal papilla and proliferation of the renal tubular epithelium. Neoplasms were believed to be caused by progression to tubular cell hyperplasia to tubular cell adenomas and, with increasing size, to adenocarcinomas or carcinomas. The similarity of the nephrotoxicity caused by trichloroethylene and N-(4'-fluoro-4-biphenyl)acetamide, tris(2,3-dibromopropyl)phosphate in rats and the species specific nature of the response suggests that degeneration and necrosis of convoluted tubules may be associated with the accumulation of alpha-2u-globin (a2u-G). Since a2u-G is a species and gender-specific protein that is causal for both the cytotoxic and carcinogenic response in male rats, extrapolation of d-limonene carcinogenicity data from rat studies to other species (including humans) is probably not warranted. Humans do not synthesise a2u-G; they do however produce other related low molecular weight proteins capable of binding chemicals that cause a2u-G nephropathy in rats but this does not necessarily connote human risk. The Risk Assessment Forum of the USA EPA concluded:

- Male renal rat tumours arising as a result of a process involving a2u-G accumulation do not contribute to the qualitative weight-of-evidence that the chemical poses a human carcinogenic hazard. Such tumours are included in dose-response extrapolations for the estimation of human carcinogenic risk.
- If the chemical induces a2u-G accumulation in male rats, the associated nephropathy is not to be used as an end-point for determining non-carcinogenic hazard.

Peroxidisable terpenes and terpenoids should only be used when the level of peroxides is kept to the lowest practicable level, for instance by adding antioxidants at the time of production. Such products should have a peroxide value of less than 10 millimoles peroxide per liter. This requirement is based on the published literature mentioning sensitising properties when containing peroxides.

361 Label and Adhesive	TOXICITY		IRRITATION	
Remover (Aerosol)	Not Available		Not Available	
	TOXICITY	IRRITA	TION	
C14-20 aliphatics (<=2% aromatics)	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Eye : Not irritating (OECD 405) *		5) *
	Inhalation(Rat) LC50; 4.6 mg/l4h ^[2]	Eye: no	o adverse effect observ	ved (not irritating) ^[1]
a. oaoo,	Oral(Rat) LD50; 7400 mg/kg ^[2]	Skin : 1	Not irritating (OECD 40	04)*
		Skin: a	dverse effect observed	d (irritating) ^[1]
	TOXICITY			IRRITATION
3,3,3-tetrafluoropropene	Inhalation(Rat) LC50; >1157.752 ppm4h ^[2]			Not Available
	TOXICITY	IRRITATIO	N	
d-limonene	Dermal (rabbit) LD50: >2 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]		(not irritating) ^[1]
	Oral(Rat) LD50; >2000 mg/kg ^[1]	Skin (rabbit): 500mg/24h moderate		te
	Skin: no adverse effect observed (not irritating) ^[1]			
	TOXICITY		IRRITATION	
gamma-terpinene	dermal (rat) LD50: >2000 mg/kg ^[1]		Skin (rabbit): 500	mg/24h mod.
	Oral(Rat) LD50; >2000 mg/kg ^[1]			
	TOXICITY	IRRITATIO	N	
	Oral(Rabbit) LD50; 4700 mg/kg ^[2]	Eye: no ad	verse effect observed	(not irritating)[1]
beta-pinene		Skin (rabbit):500 mg/24h-moderate		te
	Skin: no adverse effect observed (not in			(not irritating) ^[1]
	TOXICITY	IR	RITATION	
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	Ey	ye: adverse effect obse	erved (irritating) ^[1]
myrcene	Oral(Mouse) LD50; >3380 mg/kg ^[1]		kin (rabbit): 500 mg/24	

TOXICITY

terpinolene

IRRITATION

	dermal (rat) LD50: >2000 mg/kg ^[1]			Not Available	
	Oral(Rat) LD50; >2000 mg/kg ^[1]				
	TOXICITY		ITATION	[4]	
	dermal (rat) LD50: >2000 mg/kg ^[1]		e: no adverse effect observed (not in	ritating) ^[1]	
alpha-pinene	Oral(Rat) LD50; >500 mg/kg ^[1]		n (man): 100% - SEVERE		
			n (rabbit): 500 mg/24h - mod		
		SKI	n: adverse effect observed (irritating	3)	
	TOXICITY		IRRITATION		
alpha-terpinene	dermal (rat) LD50: >2000 mg/kg ^[1]		Eye: adverse effect observed (irrit	ating)[1]	
	Oral(Rat) LD50; ~1680 mg/kg ^[1]		Skin: adverse effect observed (irri		
			,	<u> </u>	
Legend:	Nalue obtained from Europe ECHA Registered Substant specified data extracted from RTECS - Register of Toxic E			n manufacturer's SDS. Unless otherwise	
8361 Label and Adhesive Remover (Aerosol)	allergen with specific antibodies of the IgE class and belon allergen-specific potential for causing respiratory sensitisat disposition of the exposed person are likely to be decisive. person to allergy. They may be genetically determined or a Immunologically the low molecular weight substances become (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis we asthma and atopic eczema (neurodermatitis) which is asso Exogenous allergic alveolitis is induced essentially by aller lymphocytes) may be involved. Such allergy is of the delay Epoxidation of double bonds is a common bioactivation pacapacity in vivo and in vitro and to chemically reactive tower untermore, a SAR study of potentially prohaptenic alkering are prohaptens, whereas related alkenes containing compounds. This difference in sensitizing capacity of the delay to the high reactivity and sensitizing capacity of the Allergic Contact Dermatitis—Formation, Structural Require Ann-Therese Karlberg et al: Chem. Res. Toxicol. 2008, 21, http://ftp.cdc.gov/pub/Documents/OEL/06.%20Dotson/Reference	rection, the Factor acquired one countries of thich is ociated gen sport through a cones dening isolate onjugate the allylements, pp 53-	e amount of the allergen, the exposis which increase the sensitivity of the form of the fo	ure period and the genetically determined the mucosa may play a role in predisposing a exposure to irritant substances. The by binding to peptides or proteins expetibility to allergic rhinitis, allergic bronchia type; cell-mediated reactions (Tang exposure. Formed, were found to possess sensitising emost common nucleophilic amino acids. The or in conjunction with a six-membered agated diene were weak or nonsensitizing with isolated double bonds was found to be	
C14-20 ALIPHATICS (<=2% AROMATICS)	*Exxsol D 100 SDS				
1,3,3,3- TETRAFLUOROPROPENE	Inhalation (rat) NOEL (28 days): >1.5 mg/l** Vendor HFO-1234ze is not likely to accumulate in the bodies of humans or animals HFO-1234ze i practically non-toxic. Short-term exposures at levels higher than 10% have not induced cardiac sensitization to adrenalin nor induced serious toxic effects. Rats and rabbits did not exhibit any serious toxic, developmental or reproductive effects even with exposures to high levels of HFO-1234ze. Based on a series of mutagenicity and genomics studies, the cancer risk for HFO-1234ze is low, no cardiac sensitisation was observed in dogs with exposures up to 120,000 ppm; repeated dose toxicity in rats (13-wk) found mild effects on the heart (NOEL 5,000ppm); in vitro genotoxicity findings include negative Ames Test and negative human lymphocyte chromosome aberration test; in vivo genotoxicity findings in the mouse micronucleus test were negative (inhalation, mammalian bone-marrow cytogenic test with chromosomal analysis).				
D-LIMONENE	The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing. Tumorigenic by RTECS criteria				
MYRCENE	NOTE: beta-Myrcene above 0.25 g/kg was found to be det during pregnancy by gavage	rimenta	al to the fertility and progeny numbe	r and development in the rat when given	
TERPINOLENE	Terpinolene was not irritating to human skin when applied at a concentration of 20% in petrolatum for 48 hours under a closed patch in 24 volunteers, and it was not a sensitiser in the maximization test. However, in a case report and was reported that a 49-year old woman developed eczematous lesions of the hands and forearms using a machine cleaner containing terpinolene. Upon patch testing, terpinolene gave a positive reaction. Terpinolene was not irritating in rabbits when applied to intact or abraded skin with an occluded patch for 24 hours				
ALPHA-PINENE	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 unlikely, given the severity of response, but repeated exposures may produce severe ulceration.				
8361 Label and Adhesive Remover (Aerosol) & D-LIMONENE & GAMMA- TERPINENE & BETA-PINENE & MYRCENE & TERPINOLENE & ALPHA-PINENE & ALPHA- TERPINENE	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				
8361 Label and Adhesive Remover (Aerosol) & 1,3,3,3- TETRAFLUOROPROPENE	clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. The fluoroalkenes vary widely in acute inhalation toxicity. Those, such as perfluoroisobutylene, PFIB, the most highly toxic member, attacks the pulmonary epithelium of rats eventuating in edema and death after a delay of about one day. Other fluoroalkenes, such as hexafluoropropylene (HFP) or chlorotrifluoroethylene (CTFE), also cause pulmonary injury but at lower concentrations produce concentration dependent changes in the renal concentrating mechanism of the rat. Changes in the CNS of rats and rabbits have also been reported for CTFE. CTFE, in repeated exposures, has produced blood pressure changes in dogs, CNS effects and changes in the erythropoietic system. Mechanisms of action research for fluoroalkenes is an important area of need. The nucleophilic sensitivity of the fluoroalkenes and the potential carcinogenic effects stemming are the subject of speculation.				

carcinogenic effects stemming are the subject of speculation.

Fluoralkanes, in contrast, are amongst the least toxic of all substances.

Disinfection by products (DBPs) re formed when disinfectants such as chlorine, chloramine, and ozone react with organic and inorganic matter in water. The observations that some DBPs such as trihalomethanes (THMs), di-/trichloroacetic acids, and 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX) are carcinogenic in animal studies have raised public concern over the possible adverse health effects of DBPs. To date, several hundred DBPs have been identified.

Numerous haloalkanes and haloalkenes have been tested for carcinogenic and mutagenic activities. n general, the genotoxic potential is dependent on the nature, number, and position of halogen(s) and the molecular size of the compound. Short-chain monohalogenated (excluding fluorine) alkanes and alkenes are potential direct-acting alkylating agents, particularly if the halogen is at the terminal end of the carbon chain or at an allylic position. Dihalogenated alkanes are also potential alkylating or cross-linking agents (either directly or after GSH conjugation), particularly if they are vicinally substituted (e.g., 1,2-dihaloalkane) or substituted at the two terminal ends of a short to medium-size

conjugation), particularly it they are vicinally substituted (e.g., 1,2-dinaloalikane) or substituted at the two terminal ends of a short to medium-size (e.g., 2-7) alkyl moiety (i.e., alpha, omega-dihaloalikane). Fully halogenated haloalikanes tend to act by free radical or nongenotoxic mechanisms (such as generating peroxisome-proliferative intermediates) or undergo reductive dehalogenation to yield haloalikenes that in turn could be activated to epoxides.

Haloalkenes are of concern because of potential to generate genotoxic intermediates after epoxidation. The concern for haloalkenes may be diminished if the double bond is internal or sterically hindered.

The cancer concern levels of the 14 haloalkanes and haloalkenes, have been rated based on available screening cancer bioassay (pulmonary adenoma assay) and genotoxicity data. Five brominated and iodinated methane and ethane derivatives are given a moderate rating. Beyond the fact that bromine and iodine are better leaving groups than chlorine, there is also evidence that brominated THMs may be preferentially activated by a theta-class glutathione S-transferase (GSTT1-1) to mutagens in Salmonella even at low substrate concentrations Furthermore, there are human carcinogenicity implications because of polymorphism in GSTT1-1. Human subpopulations with expressed GSTT1-1 may be at a greater risk to brominate THMs than humans who lack the gene.

Six, two, and one haloalkanes/ haloalkene(s) are given low-moderate, marginal, and low concern, respectively.

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 connubial 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 suffcient 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.

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 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 perierae 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 melanosis 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

8361 Label and Adhesive Remover (Aerosol) & D-LIMONENE & BETA-PINENE & MYRCENE & TERPINOLENE & ALPHA-PINENE

ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.

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 prehaptens, 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 peroxyl 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 clincal 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.

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.

8361 Label and Adhesive Remover (Aerosol) & C14-20 ALIPHATICS (<=2% AROMATICS) Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo-paraffins.

The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption, is known as the 'hydrocarbon continuum hypothesis', and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.

8361 Label and Adhesive Remover (Aerosol) & D-LIMONENE 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 turnours induced by limonene in male rats is though 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 adaption as no toxic effects on the liver have been reported. From available data it is not possible to identify an NOAEL for these effects. Limonene is neither genotoxic or teratogenic nor toxic to the reproductive system.

D-LIMONENE & GAMMA-TERPINENE & BETA-PINENE & MYRCENE & TERPINOLENE & AI PHA-PINENE & AI PHA-

TERPINENE

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

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

TERP is a reaction product of MMTC and dimethyltin dichloride (DMTC), Na2S, and tall oil fatty acid [a mixture of carboxylic acids, predominantly C-18]. The reaction product is a mixture of carboxylic esters and includes short oligomers of mono/dimethyltins bridged by sulfide groups.

Although the tall oil component of TERP is not structurally similar to EHTG, TERP's conversion to MMTC justifies its inclusion. While the contribution of the various ligands to the overall toxicity may vary, the contribution is expected to be small relative to that of the MMTC. Further, the EHTG ligand from MMT(EHTG) is likely to be more toxic than the oleic or linoleic acid from TERP so inclusion of TERP in the category is a rather conservative approach. The other possible degradate of tall oil and EHTG is 2-mercaptoethanol (2-ME), and it is common to both ligands. Data for MMT(EHTG) and MMT(IOTG) are used interchangeably because they are isomers differing only slightly in the structure of the C-8 alcohol of the mercaptoester ligand. In addition, the breakdown products of MMT(EHTG) and MMT(IOTG) are the thioglycolate esters (EHTG and IOTG), which have the common degradates, thioglycolic acid and C-8 alcohols (either 2-ethylhexanol or isooctanol). EHTG and IOTG also have similar physicochemical and toxicological properties.

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

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

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

Oral:

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

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

Dermal

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

The acute dermal LD50 of MMT(2-EHMA) in rabbits was 1000 (460 to 2020) mg/kg for females and 2150 (1000 to 4620) mg/kg for males. There were no deaths at 215 and 464 mg/kg, 0/2 males and 1/2 females died at 1000 mg/kg and 1/2 males and 2/2 females died at 2150 mg/kg. All animals died at 4640 and 10 000 mg/kg. A variety of clinical abnormalities were observed and disappeared in surviving animals by the end of the exposure period. Clinical signs included death, uncoordinated movements, shaking, and hypersensitivity to external stimuli.

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

Inhalation:

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

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

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

Sensitisation:

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

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

Repeat dose toxicity:

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

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

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

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

Neurotoxicity:

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

Immunotoxicity:

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

Adult male rats were given drinking water for 28 d containing a mixture of dibutyltin dichloride (DBTC), dimethyltin dichloride (DMTC), monobutyltin trichloride (MBT), and monomethyltin trichloride (MMT) in a 2:2:1:1 ratio, respectively, at 3 different concentrations (5:5:2.5:2.5.2.5), or 20:20:10:10 mg organotin/L). Rats were also exposed to MMT alone (20 or 40 mg MMT/L) or plain water as a control. Delayed-type hypersensitivity, antibody synthesis, and natural killer cell cytotoxicity were evaluated in separate endpoint groups immediately after exposure ended.

The evaluated immune functions were not affected by the mixture or by MMT alone. The data suggest that immunotoxicity is unlikely to result from the concentration of organotins present in drinking water delivered via PVC pipes, as the concentrations used were several orders of magnitude higher than those expected to leach from PVC pipes

In a guideline 90-day subchronic dietary study in rats,with MMT(2-EHMA), based on the changes in neurobehavioral parameters and associated brain histopathology that occurred at the high dose of 750 ppm (equivalent to 49.7 mg/kg bw/day in males and 53.6 mg/kg bw/day in females), as well as changes in haematology, clinical chemistry, urinalysis, organ weights, and pathology of the thymus at the same dose, the NOAEL was the next lower dose of 150 ppm (equivalent to 9.8 mg/kg bw/day in males and 10.2 mg/kg bw/day in females).

The monomethyltin compounds as a class are not mutagenic in the Ames test. TERP was positive in a human lymphocyte assay. MMTC was equivocal for induction of micronucleated polychromatic erythrocytes (MPEs) in an in vivo rat micronucleus test (OECD 474). In this study a statistically significant increase in MPE was observed only at 24 h and not at 48 h after treatment and there was no dose-response. Based on these observations the overall conclusion is that MMTC does not have genotoxic potential.

From the results obtained in a micronucleus test with MMT(2-EHMA), it was demonstrated that the substance was weakly genotoxic to bone

marrow cells of rats and that the substance has the potential to induce damage to the mitotic spindle apparatus of the bone marrow target cells. Carcinogenicity:

In a limited carcinogenicity study, MMT(EHTG) produced no compound-related macroscopic or microscopic changes in rats fed 100 ppm in the diet for two years.

Toxicity to reproduction:

In the reproductive satellite portion of the 90-day study using MMTC (with dose levels of 30, 150, and 750 ppm in the diet), post-implantation loss, decreased litter size and increased neonatal mortality occurred at 750 ppm (26-46 mg/kg bw/d for females). Maternal gestational body weights were transiently suppressed and other maternal toxicity was inferred from the repeated dose results at this dose. There were no malformations observed at any dose. The NOAEL for maternal toxicity, and reproductive, and foetotoxic effects was 150 ppm in the diet (6-12 mg/kg bw/d). SIDS Initial Assessment Profile (SIAM 23 2006)

ECHA Registration Dossier for MMT(2-EHMA) (ethylhexyl 10-ethyl-4-[[2-[(2-ethylhexyl)oxy]-2-oxoethyl]thio]-4-methyl-7-oxo-8-oxa-3,5-dithia-4-stannatetradecanoate)

GAMMA-TERPINENE & BETA-PINENE & MYRCENE & TERPINOLENE & ALPHAPINENE

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

For monoterpenes:

The chemical category designated terpenoid hydrocarbons includes three simple C10 isomeric monocyclic terpene hydrocarbons (*d*-limonene, *dl*-limonene, and terpinolene) two simple C10 acyclic terpene hydrocarbons (*beta*-myrcene and dihydromyrcene) and mixtures composed primarily of *d*-limonene, *dl*-limonene (dipentene), terpinolene, myrcene, and *alpha* deta-pinene

Monoterpene hydrocarbons are mainly released by coniferous woodland such as pine trees, cedars, redwood and firs. To a lesser extent, they are also produced and released by deciduous plants. They are common components of traditional foods occurring in essentially all fruits and vegetables.

Members of this chemical category are of very low acute toxicity

Studies of terpene hydrocarbons indicate that they are rapidly absorbed, distributed, metabolised and excreted. The principal metabolic pathway involves side chain oxidation to yield monocyclic terpene alcohols and carboxylic acids. These metabolites are mainly conjugated with glucuronic acid and excreted in the urine, or to a lesser extent in the feces. A secondary pathway involves epoxidation of either the exocyclic or endocyclic double bond yielding an epoxide that is subsequently detoxicated *via* formation of the corresponding diol or conjugation with gluctahione. Although some species- and sex-specific differences exist, studies for *d*-limonene and *beta*-myrcene indicate that the monoterpene hydrocarbons in this chemical category will participate in common pathways of absorption, distribution, metabolism and excretion.

Genotoxicity: Based on the results of this *in vivo* genotoxicity assay and the numerous *in vitro* genotoxicity assays, it is unlikely that any of these materials would exhibit a significant genotoxic potential *in vivo*.

Carcinogenicity: Under the conditions of 2-year gavage studies, conducted by NTP, there was clear evidence of carcinogenic activity of d-limonene for male F344/N rats as shown by increased incidences in tubular cell hyperplasia, adenomas, and adenocarcinomas of the kidney. There was no evidence of carcinogenic activity of d-limonene for female rats receiving 300 or 600 mg/kg bw/d. It has been demonstrated that renal lesions, which were observed in the NTP study, resulted from the accumulation of aggregates of alpha-2 microglobulin (a low molecular-weight protein synthesised in the liver) and limonene-1,2-epoxide in the P2 segment of the renal proximal tubule. While humans produce low molecular weight serum proteins, which are reabsorbed by the kidney, there is no evidence that a similar alpha-2 microglobulin is produced. The kidney changes seen in male rats administered limonene have been well characterized, and are known to be specific to the male rat and of no significance in human risk assessment.

Reproductive toxicity: Substances within this chemical category exhibit low reproductive toxicity potential. This is based on the results of three reproductive toxicity assays. using sweet orange peel oil predominantly composed of *d*-limonene and *beta*-myrcene.

Developmental toxicity: Given the results of six developmental toxicity assays using limonene, sweet orange oil and beta-myrcene, it may be concluded that the substances within this chemical category exhibit low developmental toxicity potential

GAMMA-TERPINENE & MYRCENE

GAMMA-TERPINENE &

ALPHA-TERPINENE

MYRCENE & TERPINOLENE &

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 the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

For bicyclic terpenes:

Acute toxicity: The literature abounds with clinical reports of accidental and intentional acute poisoning with pinene-based turpentine. Rat oral LD50 values are available for *alpha*-pinene, *beta*-pinene, camphene and turpentine oil and indicate these materials to be very low in oral acute toxicity with LD50 values in the range from 3388 mg/kg to greater than 5000 mg/kg. Rabbit dermal LD50 values similarly indicate very low toxicities with values greater than the limit doses of 2000 or 5000 mg/kg.

Acute inhalation toxicity has been measure in different animal species. The acute LC50 was reported to be 13,500 mg/m3 in rats, 13,500 mg/m3 in guinea pigs, and 9000 mg/m3 in mice. The acute inhalation LC50 of commercial grade turpentine in Wistar rats is reported to be in the range of 12,000-20,000 mg/m3 for 1 to 6 hour exposures and the LC50 for a 2-hour exposure in Swiss-Webster mice is 29,000 mg/m3. Based on these results the acute oral, dermal, and inhalation toxicities of bicyclic terpene hydrocarbons is concluded to be low.

Repeat dose toxicity: A 28-day repeat dose study has been performed with camphene according to an OECD Guideline 407 in both sexes of Wistar rats. Animals of both sexes at the 1000 mg/kg bw/day dose exhibited vacuolization of hepatocytes and increase liver weights. Male rats also exhibited alpha-2-microglobulin-type nephrotoxicity at all dose levels.

Subsequent investigations have shown that the *alpha-2*-microglobulin nephropathy found in the F344/N male rat does not develop in mammals that do not express the hepatic form of *alpha-2*-microglobulin (e.g. other strains of rats, mice, dogs, humans). Therefore, the nephrotoxicity observed in the camphene study in male F344 rats is not relevant to the human health risk assessment. Based on liver toxicity, the NOAEL for this study is concluded to be 250 mg/kg bw/day

Reproductive toxicity: In the a-animal species study, no reproductive effects were observed when dose levels of up to 260 to 600 mg/kg bw of an essential oil predominantly composed of bicyclic terpene hydrocarbons (alpha-pinene, beta-pinene, and sabinene) was administered daily to mice, rats, or hamsters during gestation. When this data is combined with the fact that no adverse effects were observed to the reproductive organs in a 28-day study with camphene at dose levels up to 250 mg/kg bw/day, it is concluded that bicyclic terpene hydrocarbons including alpha-pinene and beta-pinene are not reproductive toxicants

Two ninety day inhalation studies have been performed for alpha-pinene in which a full complement of male and female sex organs and tissues were subjected to histopathological examination. Both studies reported no microscopic changes that could be associated with exposure to the test substance. Taking into account the lack of any effects to females in a earlier teratology study, the absence of any maternal or developmental effects in a reproductive/developmental study of a pinene-based oil and for a structurally related monoterpene hydrocarbon, myrcene, it can be concluded that the members of this category show no significant reproductive or developmental toxicity

Developmental toxicity:. Based on the NOAELs for maternal and developmental toxicity in studies with camphene (250 and 1000 mg/kg bw/day) and a terpene hydrocarbon mixture containing *alpha*- and *beta*-pinene and camphene (688 mg/kg bw/day), and the lack of any signs of maternal or developmental toxicity in a mice,

rats, or hamsters given 260 to 600 mg/kg bw/day of a mixture composed primarily (>80%) of *alpha*- and *beta*-pinene and sabinene, it is concluded that bicyclic terpene hydrocarbons are not maternal or developmental toxicants.

BETA-PINENE & ALPHA-PINENE

Genotoxicity:

In vitro: In vitro genotoxicity assays available for *alpha*-pinene, *beta*-pinene and camphene demonstrate that these substances have a little, if any, genotoxic potential. In standard Ames assays of *alpha*-pinene, *beta*-pinene and camphene, *Salmonella typhimurium* strains TA97, TA98, TA100, TA1535, TA1537, and TA1538 provided no evidence of mutagenicity at any dose tested.

In vivo: Based on the lack of any evidence of genotoxicity in numerous in vitro assays with and without metabolic activation, it is unlikely that any of these bicyclic terpenes would exhibit a significant genotoxic potential in vivo.

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:

X - Data either not available or does not fill the criteria for classification

Data available to make classification

11.2.1. Endocrine Disruption Properties

Many chemicals may mimic or interfere with the body's hormones, known as the endocrine system. Endocrine disruptors are chemicals that can interfere with endocrine (or hormonal) systems. Endocrine disruptors interfere with the synthesis, secretion, transport, binding, action, or elimination of natural hormones in the body. Any system in the body controlled by hormones can be derailed by hormone disruptors. Specifically, endocrine disruptors may be associated with the development of learning disabilities, deformations of the body various cancers and sexual development problems. Endocrine disrupting chemicals cause adverse effects in animals. But limited scientific information exists on potential health problems in humans. Because people are typically exposed to multiple endocrine disruptors at the same time, assessing public health effects is difficult.

SECTION 12 Ecological information

12.1. Toxicity

8361 Label and Adhesive	Endpoint	Test Duration (hr)		Species	Value		Source	
Remover (Aerosol)	Not Available	Not Available		Not Available Not Available			Not Available	
	Endpoint	Test Duration (hr)	Spe	ecies		Value	Source	
C14-20 aliphatics (<=2%	NOEC(ECx)	72h		ae or other aquatic plai	nts	<0.03mg/		
aromatics)	NOEC(ECx)	3072h	Fish			1mg/l	1	
	Endpoint	Test Duration (hr)	Spe	cies		Value	Source	
	EC50	72h	Alga	ae or other aquatic plar	nts	>170mg/	1 2	
,3,3,3-tetrafluoropropene	EC50	48h	Crus	stacea		>160mg/	1 2	
	EC50(ECx)	48h	Crus	stacea		>160mg/	1 2	
	Endpoint	Test Duration (hr)	Spe	ecies		Value	Source	
	NOEC(ECx)	504h	Cru	stacea		0.05mg/l	2	
d-limonene	EC50	72h	Alga	Algae or other aquatic plants		0.214mg/	1 2	
	LC50	96h	Fish	า		0.46mg/l	2	
	EC50	48h	Cru	stacea		0.307mg/	1 2	
	Endpoint	Test Duration (hr)	Specie	es		Value	Source	
	EC50(ECx)	96h	Fish			2.792mg/l	2	
gamma-terpinene	EC50	72h	Algae	or other aquatic plants		>10.82mg/l	2	
	EC50	48h	Crusta	cea		2.99-4.07mg/l	4	
	Endpoint	Test Duration (hr)	Spe	cies		Value	Source	
	EC50	72h	Alga	e or other aquatic plan	ts	0.7mg/l	2	
beta-pinene	LC50	96h	Fish			0.557mg/	1 2	
	EC50	48h	Crus	stacea		1.09mg/l	2	
	EC10(ECx)	48h	Alga	e or other aquatic plan	ts	0.378mg/	2	
							,	
	Endpoint	Test Duration (hr)	Spe	ecies		Value	Source	
myrcene	EC50	72h	Alga	ae or other aquatic plar	nts	0.31mg/	1 2	
,	EC50	48h	Cru	stacea		1.47mg/	1 2	
	EC50(ECx)	72h	1	ae or other aquatic plai		0.31mg/	1 2	

	Endpoint	Test I	Ouration (hr)	Species			Value	Source
	EC10(ECx)	72h		Algae or other a	aquatic plants		0.054mg/l	2
terpinolene	EC50	72h		Algae or other a	aquatic plants		0.302mg/l	2
	LC50	96h		Fish			0.805mg/l	2
	EC50	48h		Crustacea			0.634mg/l	2
alpha-pinene	NOEC(ECx) LC50 EC50	48h 96h 48h		Algae or other Fish Crustacea	aquatic plants		0.131mg/l 0.303mg/l 0.475mg/l	2 2 2
	Endpoint		Test Duration (hr)		Species	Valu	le	Source
alpha-terpinene	EC50(ECx)		48h		Crustacea	1.7m	ng/l	2
			48h		Crustacea	1.7m		2

Toxic 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

Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment

of equipment wash-waters.

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

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.

For petroleum distillates:

Environmental fate:

When petroleum substances are released into the environment, four major fate processes will take place: dissolution in water, volatilization, biodegradation and adsorption. These processes will cause changes in the composition of these UVCB substances. In the case of spills on land or water surfaces, photodegradation-another fate process-can also be significant.

As noted previously, the solubility and vapour pressure of components within a mixture will differ from those of the component alone. These interactions are complex for complex UVCBs such as petroleum hydrocarbons.

Each of the fate processes affects hydrocarbon families differently. Aromatics tend to be more water-soluble than aliphatics of the same carbon number, whereas aliphatics tend to be more volatile. Thus, when a petroleum mixture is released into the environment, the principal water contaminants are likely to be aromatics, whereas aliphatics will be the principal air contaminants. The trend in volatility by component class is as follows: alkenes = alkanes > aromatics = cycloalkanes.

The most soluble and volatile components have the lowest molecular weight; thus there is a general shift to higher molecular weight components in residual materials. Biodegradation:

Biodegradation is almost always operative when petroleum mixtures are released into the environment. It has been widely demonstrated that nearly all soils and sediments have populations of bacteria and other organisms capable of degrading petroleum hydrocarbons. Degradation occurs both in the presence and absence of oxygen. Two key factors that determine degradation rates are oxygen supply and molecular structure. In general, degradation is more rapid under aerobic conditions. Decreasing trends in degradation rates according to structure are as follows:

- (1) n-alkanes, especially in the C10–C25 range, which are degraded readily;
- (2) isoalkanes;
- (3) alkenes;
- (4) benzene, toluene, ethylbenzene, xylenes (BTEX) (when present in concentrations that are not toxic to microorganisms);
- (5) monoaromatics;
- (6) polynuclear (polycyclic) aromatic hydrocarbons (PAHs); and

Legend:

(7) higher molecular weight cycloalkanes (which may degrade very slowly.

Three weathering processes-dissolution in water, volatilization and biodegradation-typically result in the depletion of the more readily soluble, volatile and degradable compounds and the accumulation of those most resistant to these processes in residues.

When large quantities of a hydrocarbon mixture enter the soil compartment, soil organic matter and other sorption sites in soil are fully saturated and the hydrocarbons will begin to form a separate phase (a non-aqueous phase liquid, or NAPL) in the soil. At concentrations below the retention capacity for the hydrocarbon in the soil, the NAPL will be immobile this is referred to as residual NAPL. Above the retention capacity, the NAPL becomes mobile and will move within the soil

Bioaccumulation potential was characterized based on empirical and/or modelled data for a suite of petroleum hydrocarbons expected to occur in petroleum substances. Bioaccumulation factors (BAFs) are the preferred metric for assessing the bioaccumulation potential of substances, as the bioconcentration factor (BCF) may not adequately account for the bioaccumulation potential of substances via the diet, which predominates for substances with log Kow > ~4.5

In addition to fish BCF and BAF data, bioaccumulation data for aquatic invertebrate species were also considered. Biota-sediment/soil accumulation factors (BSAFs), trophic magnification factors and biomagnification factors were also considered in characterizing bioaccumulation potential.

Overall, there is consistent empirical and predicted evidence to suggest that the following components have the potential for high bioaccumulation, with BAF/BCF values greater than 5000: C13–C15 isoalkanes, C12 alkenes, C12–C15 one-ring cycloalkanes, C12 and C15 two-ring cycloalkanes, C14 polycycloalkanes, C15 one-ring aromatics, C15 and C20 cycloalkane monoaromatics, C12–C13 diaromatics, C20 cycloalkane diaromatics, and C14 and C20 three-ring PAHs

These components are associated with a slow rate of metabolism and are highly lipophilic. Exposures from water and diet, when combined, suggest that the rate of uptake would exceed that of the total elimination rate. Most of these components are not expected to biomagnify in aquatic or terrestrial foodwebs, largely because a combination of metabolism, low dietary assimilation efficiency and growth dilution allows the elimination rate to exceed the uptake rate from the diet; however,

one study suggests that some alkyl-PAHs may biomagnify. While only BSAFs were found for some PAHs, it is possible that BSAFs will be > 1 for invertebrates, given that they do not have the same metabolic competency as fish.

In general, fish can efficiently metabolize aromatic compounds. There is some evidence that alkylation increases bioaccumulation of naphthalene but it is not known if this can be generalized to larger PAHs or if any potential increase in bioaccumulation due to alkylation will be sufficient to exceed a BAF/BCF of 5000.

Some lower trophic level organisms (i.e., invertebrates) appear to lack the capacity to efficiently metabolize aromatic compounds, resulting in high bioaccumulation potential for some aromatic components as compared to fish.

This is the case for the C14 three-ring PAH, which was bioconcentrated to a high level (BCF > 5000) by invertebrates but not by fish. There is potential for such bioaccumulative components to reach toxic levels in organisms if exposure is continuous and of sufficient magnitude, though this is unlikely in the water column following a spill scenario due to relatively rapid dispersal

Bioaccumulation of aromatic compounds might be lower in natural environments than what is observed in the laboratory. PAHs may sorb to organic material suspended in the water column (dissolved humic material), which decreases their overall bioavailability primarily due to an increase in size. This has been observed with fish

Diesel fuel studies in salt water are available. The values varied greatly for aquatic species such as rainbow trout and Daphnia magna, demonstrating the inherent variability of diesel fuel compositions and its effects on toxicity. Most experimental acute toxicity values are above 1 mg/L. The lowest 48-hour LC50 for salmonids was 2.4 mg/L. Daphnia magna had a

24-hour LC50 of 1.8 mg/. The values varied greatly for aquatic species such as rainbow trout and Daphnia magna, demonstrating the inherent variability of diesel fuel compositions and its effects on toxicity. Most experimental acute toxicity values are above 1 mg/L. The lowest 48-hour LC50 for salmonids was 2.4 mg/L. Daphnia magna had a 24-hour LC50 of 1.8 mg/L

The tropical mysid Metamysidopsis insularis was shown to be very sensitive to diesel fuel, with a 96-hour LC50 value of 0.22 mg/L this species has been shown to be as sensitive as temperate mysids to toxicants. However, However this study used nominal concentrations, and therefore was not considered acceptable. In another study involving diesel fuel, the effect on brown or common shrimp (Crangon crangon) a 96-hour LC50 of 22 mg/L was determined. A "gas oil" was also tested and a 96-hour LC50 of 12 mg/L was determined The steady state cell density of marine phytoplankton decreased with increasing concentrations of diesel fuel, with different sensitivities between species . The diatom Phaeodactylum tricornutum showed a 20% decrease in cell density in 24 hours following a 3 mg/L exposure with a 24-hour no-observed effect concentration (NOEC) of 2.5 mg/L. The microalga Isochrysis galbana was more tolerant to diesel fuel, with a 24-hour lowest-observed-effect concentration (LOEC) of 26 mg/L (14% decrease in cell density), and a NOEC of 25 mg/L. Finally, the green algae Chlorella salina was relatively insensitive to diesel fuel contamination, with a 24-hour LOEC of 170 mg/L (27% decrease in cell density), and a NOEC of 160 mg/L . All populations of phytoplankton returned to a steady state within 5 days of exposure

In sandy soils, earthworm (Eisenia fetida) mortality only occurred at diesel fuel concentrations greater than 10 000 mg/kg, which was also the concentration at which sub-lethal weight

Nephrotoxic effects of diesel fuel have been documented in several animal and human studies. Some species of birds (mallard ducks in particular) are generally resistant to the toxic effects of petrochemical ingestion, and large amounts of petrochemicals are needed in order to cause direct mortality

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

Monomethyltin trichloride (MMTC, CAS RN: 993-16-8), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA)), CAS RN: 57583-34-3), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA)), CAS RN: 57583-34-3), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA)), CAS RN: 57583-34-3), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA)), CAS RN: 57583-34-3), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA)), CAS RN: 57583-34-3), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA)), CAS RN: 57583-34-3), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA)), CAS RN: 57583-34-3), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA)), CAS RN: 57583-34-3), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA)), CAS RN: 57583-34-3), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA)), CAS RN: 57583-34-3), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA)), CAS RN: 57583-34-3), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA)), CAS RN: 57583-34-3), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA)), CAS RN: 57583-34-3), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA)), CAS RN: 57583-34-3), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA)), CAS RN: 57583-34-3), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA)), CAS RN: 57583-34-3), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA)), CAS RN: 57583-34-3), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA)), CAS RN: 57583-34-3), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (EHTG; MMT (2-EHMA)), CAS RN: 57583-34-3), monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (2-EHMA)), CAS RN: 57583-34-30, monomethyltin tris[2-ethylhexylmercaptoacetate (MMT (2-EHMA)), monomethyltin tris[2-ethylhexylmercaptoace tris[isooctylmercaptoacetate (MMT(IOTG), CAS RN: 54849-38-6), CAS RN: 57583-34-3) and methyltin reverse ester tallate reaction product (TERP, CAS RNs: 201687-58-3, 201687-57-2, 68442-12-6, 151436-98-5) are considered as a single category of compounds for the purpose of an environmental assessment. All share a MMTC as a building block.

Environmental fate

MMT(IOTG), MMT(EHTG), and TERP are sparingly soluble in water (0.6-10.7 mg/L). In water, these monomethyltin compounds undergo rapid degradation by hydrolysis. Although there is no stability data for MMT(EHTG)/(IOTG) or TERP, data for other organotins [DOTC, DBTL and DBT(EHTG)] indicate that the monomethyltin compounds are expected to hydrolyze within minutes to hours in water. The thioester ligands on MMT(EHTG)/(IOTG) will be rapidly displaced to form mono-methyltin hydroxide which eventually precipitates as the oxide. It is also possible that the labile ligands can be displaced by other anions in the medium. The displaced thioester ligands, EHTG/IOTG, can also undergo further hydrolysis of the ester linkage to form thioglycolic acid and ethylhexanol or isooctanol, respectively.

MMTC is a solid at room temperature and melts at 43 deg C, boils at 171 deg C, has a calculated vapour pressure of 1.7 hPa at 25 deg C, and is soluble in water (1038 g/L at 20 deg C). The measured log Kow is -0.9 and MMTC is not readily biodegradable. Atmospheric degradation occurs by photochemical induced hydroxyl radicals, with a half-life of 15.7 days. A Henry's Law constant of 3.83 x 10-7 atm-m3/mol predicts MMTC will volatilize from surface water (t1/2 = 99 days and 3 years for model river and lake, respectively). If released to the environment, MMTC is expected to partition primarily into water (54%) and soil (43%).

In water, MMTC undergoes rapid degradation by hydrolysis and is expected to hydrolyze within minutes. It is expected that the chlorines in MMTC will be displaced to form mono-methyltin hydroxide which eventually precipitates as the oxide (the alkyltin moiety (MMT) was hydrolytically stable at pH 4, 7, and 9 (t1/2 > 1 year at 25 deg C)).

TERP is a liquid at room temperature, boils at 216 deg C, and has a calculated vapour pressure of 0.2 hPa at 25 deg C. TERP is slightly soluble in water (4.4 mg/L), highly hydrophobic (log Kow = 25.5), has low potential for bioaccumulation (log BCF = 2.0), and is readily biodegradable. It is degraded atmospherically by hydroxyl radicals and ozone, with a half-life of 0.5 hours. If released to the environment, TERP is predicted to partition primarily to sediment (99%).

MMT(EHTG) is a liquid at room temperature and has a freezing point of -85 to -65 deg C, decomposes at 260 deg C has a derived vapour pressure of 0.02 hPa at 25 deg C, a calculated log Kow of 10.98, is slightly soluble in water (1.8-6 mg/L), and is readily biodegradable. MMT(EHTG) is also degraded atmospherically, with a half-life of 6.3 hours. A Henry's Law constant of 3.18×10+4 atm-m3/mol predicts MMT(EHTG) will volatilize from surface water (t1/2 = 8 hours and 11 days for a model river and lake, respectively). If released to the environment, MMT(EHTG) is expected to partition primarily into sediment (71%) and soil (25%).

Bioavailability:

The considerable difference in the structures of the labile ligands causes differences in water solubility between the alkyltin chloride and thioesters affecting their respective bioavailabilities and distribution in the environment. Furthermore, MMT(EHTG) and MMT(IOTG) will degrade in aqueous solution such that organisms will be exposed to the parent material and their different degradation products. MMTC is not an appropriate surrogate for the thioesters or TERP for the ecotoxicity and environmental fate endpoints.

In the ecotoxicity tests the organisms were most likely exposed to parent substance as well as hydrolysis/degradation products.

MMTC was not acutely toxic to zebra fish (Brachydanio rerio) (96-h LC50 > 102 mg/L) or Daphnia magna (48-h EC50 > 101 mg/L). MMTC inhibited the growth (72-h EC50 = 0.03 mg/L) and biomass (72-h EC50 = 0.02 mg/L) of the green alga Scenedesmus subspicatus (NOEC = 0.007 mg/L). MMTC was not acutely toxic to earthworms at nominal concentrations up to 1000 mg/kg

TERP was not acutely toxic to rainbow trout (Oncorhynchus mykiss) (96-hr LC50 >4.4 mg/L), inhibited D. magna survival and mobility (48-h EC50 = 0.27 mg/L), and inhibited growth of the freshwater green alga Pseudokirchneriella subcapitata was (72-h EC50 = 0.64 mg/L; NOEC = 0.28 mg/L).

MMT(EHTG) was not acutely toxic to B. rerio (LC50 > 6 mg/L; NOEC = 3.6 mg/L) and did not inhibit the growth of S. subspicatus (72-h EC50 > 1.84 mg/L; NOEC = 0.6 mg/L). The 21-d EC50 for reproduction in a chronic Daphnia magna study was > 0.134 mg/L (NOEC = 0.134 mg/L).

Substances containing unsaturated carbons are ubiquitous in indoor environments. They result from many sources (see below). Most are reactive with environmental ozone and many produce stable products which are thought to adversely affect human health. The potential for surfaces in an enclosed space to facilitate reactions should be considered. Source of unsaturated substances Unsaturated substances (Reactive Emissions) Major Stable Products produced following reaction with ozone.

Formaldehyde

Formaldehyde, benzaldehyde

oxidation products Soft woods, wood flooring, including Isoprene, limonene, alpha-pinene, other terpenes and

Isoprene, nitric oxide, squalene, unsaturated sterols,

Methacrolein, methyl vinyl ketone, nitrogen dioxide, acetone, 6MHQ, geranyl acetone, Occupants (exhaled breath, ski oils, oleic acid and other unsaturated fatty acids, unsaturated 4OPA, formaldehyde, nonanol, decanal, 9-oxo-nonanoic acid, azelaic acid, nonanoic acid.

cypress, cedar and silver fir boards, sesquiterpenes houseplants

4-Phenylcyclohexene, 4-vinylcyclohexene, styrene,

Formaldehyde, 4-AMC, pinoaldehyde, pinic acid, pinonic acid, formic acid, methacrolein, methyl vinyl ketone, SOAs including ultrafine particles

Carpets and carpet backing Linoleum and paints/polishes containing linseed oil

2-ethylhexyl acrylate, unsaturated fatty acids and esters Linoleic acid, linolenic acid

Formaldehyde, acetaldehyde, benzaldehyde, hexanal, nonanal, 2-nonenal Propanal, hexanal, nonanal, 2-heptenal, 2-nonenal, 2-decenal, 1-pentene-3-one, propionic acid, n-butyric acid

Latex paint waxes, air fresheners

Residual monomers Limonene, alpha-pinene, terpinolene, alpha-terpineol, and other sesquiterpenes

Formaldehyde, acetaldehyde, glycoaldehyde, formic acid, acetic acid, hydrogen and Certain cleaning products, polishes, linalool, linalyl acetate and other terpenoids, longifolene organic peroxides, acetone, benzaldehyde, 4-hydroxy-4-methyl-5-hexen-1-al, 5-ethenyldihydro-5-methyl-2(3H)-furanone, 4-AMC, SOAs including ultrafine particles Formaldehyde, methacrolein, methyl vinyl ketone

Natural rubber adhesive Photocopier toner, printed paper, styrene polymers

Styrene

Isoprene, terpenes

Styrene, acrolein, nicotine

Formaldehyde, benzaldehyde, hexanal, glyoxal, N-methylformamide, nicotinaldehyde,

Environmental tobacco smoke Soiled clothing, fabrics, bedding

Squalene, unsaturated sterols, oleic acid and other saturated fatty acids

Acetone, geranyl acetone, 6MHO, 40PA, formaldehyde, nonanal, decanal, 9-oxononanoic acid, azelaic acid, nonanoic acid Formaldehyde, nonanal, and other aldehydes; azelaic acid; nonanoic acid; 9-oxo-

Soiled particle filters

other vegetative debris; soot; diesel particles

Unsaturated fatty acids from plant waxes, leaf litter, and nonanoic acid and other oxo-acids; compounds with mixed functional groups (=0, -OH,

Unsaturated fatty acids and esters, unsaturated oils, Ventilation ducts and duct liners

Polycyclic aromatic hydrocarbons

Perfumes, colognes, essential oils Limonene, alpha-pinene, linalool, linalyl acetate,

(e.g. lavender, eucalyptus, tea tree) terpinene-4-ol, gamma-terpinene

Overall home emissions Limonene, alpha-pinene, styrene C5 to C10 aldehydes

Oxidized polycyclic aromatic hydrocarbons

Formaldehyde, 4-AMC, acetone, 4-hydroxy-4-methyl-5-hexen-1-al, 5-ethenyl-dihydro-5-methyl-2(3H) furanone, SOAs including ultrafine particles

Formaldehyde, 4-AMC, pinonaldehyde, acetone, pinic acid, pinonic acid, formic acid, benzaldehyde, SOAs including ultrafine particles

Abbreviations: 4-AMC, 4-acetyl-1-methylcyclohexene; 6MHQ, 6-methyl-5-heptene-2-one, 4OPA, 4-oxopentanal, SOA, Secondary Organic Aerosols

Reference: Charles J Weschler; Environmental Helath Perspectives, Vol 114, October 2006

In addition to carbon dioxide (CO2), methane (CH4) and nitrous oxide (N2O), the greenhouse gases mentioned in the Kyoto Protocol include synthetic substances that share the common feature of being highly persistent in the atmosphere and exhibiting very high specific radiative forcing (radiative forcing is the change in the balance between radiation coming into the atmosphere and radiation out; a positive radiative forcing tends on average to warm the surface of the earth). These synthetic substances include hydrocarbons that are partially fluorinated (HCFs) or totally fluorinated (PFCs) as well as sulfur hexafluoride (SF6).

The greenhouse potential of these substances, expressed as multiples of that of CO2, are within the range of 140 to 11,700 for HFCs, from 6500 to 9,200 for PFCs and 23,900 for SF6. Once emitted into the atmosphere, these substances have an impact on the environment for decades, centuries, or in certain instances, for thousands of years

Many of these substances have only been commercialised for a few years, and still only contribute only a small percentage of those gases released to the atmosphere by humans (anthropogenic) which increase the greenhouse effect. However, a rapid increase can be seen in their consumption and emission, and therefore in their contribution to the anthropogenic increase in the greenhouse effect.

Since the adoption of the Kyoto Protocol, new fluorinated substances have appeared on the market, which are stable in air and have a high greenhouse potential; these include nitrogen trifluoride (NF3) and fluoroethers.

'Urban grime

Atmospheric fate: Due to the high volatility of limonene the atmosphere is expected to be the major environmental sink for this chemical where it is expected to undergo gas-phase reactions with photochemically produced hydroxyl radicals, ozone and nitrate radicals. Calculated lifetimes for the reaction of d-limonene with photochemically produced hydroxyl radicals range from 0.3-2 h based on experimentally determined rate constants. The oxidation of limonene may contribute to aerosol and photochemical smog formation. Calculated lifetimes for the night-time reaction of d-limonene with nitrate radicals range form 0.9 to 9 minutes. The daytime atmospheric lifetime of d-limonene is estimated to range from 12 to 48 min. depending upon local hydroxyl rate and ozone concentrations. Products produced from hydroxy radical reaction with limonene are 4-acetyl-1-methylcyclohexene, a keto-aldehyde, formaldehyde, 3-oxobutanal, glyoxal and a C10 dicarbonyl. The same carbonyls, along with formic acid and C8 and C9 carboxylic acids, may form in reactions with ozone. Ozonolysis of limonene may also lead to the formation of hydrogen peroxide and organic peroxides, which have various toxic effects on plant cells and may damage forests. Products of ozonolysis include bis(hydroxmethyl)peroxide, a precursor to hydroxymethyl hydroperoxide and hydrogen peroxide. The reaction of d-limonene with ozone in the dark results in the formation of 4-acetyl-1-methylcyclohexene and formaldehyde. Reactions with nitrogen oxides produce aerosol formation as well as lower molecular weight products such as formaldehyde, acetaldehyde, formic acid, acetone and peroxyacetyl nitrate.

Terrestrial fate: When released to the ground limonene is expected to have low to very low mobility in soil based on its physicochemical properties. The soil adsorption coefficient (Koc) calculated on the basis of solubility (13.8 mg/l, 25 C) and the log octanol/ water partition coefficient (4.23) ranges from 1030 and 4780. The Henry's law constant indicates that limonene will rapidly volatilise from both dry and moist soil; however its absorption to soil may slow the process.

Aquatic fate: In the aquatic environment, limonene is expected to evaporate to a significant extent owing to its high volatility. The estimated half-life for volatilisation of limonene from a model river (1 m deep, flow 1 m/s and wind speed 3 m/s) is 3.4 h. Some limonene is expected to absorb to sediment and suspended organic matter.

Biodegradation and bioaccumulation: Limonene does not have functional groups for hydrolysis and its cyclohexene ring and ethylene group are known to resist hydrolysis Therefore, hydrolysis of limonene is not expected in terrestrial or in aquatic environments. The hydrolytic half-life of d-limonene is estimated to be >1000 days. Biotic degradation of

limonene has been shown with some species of microorganisms such as Penicillium digitatum, Corynespora cassiicola, Diplodia gossyppina and a soil strain of Pseudomonans sp (SL strain). Limonene is readily biodegradable (41-98% degradation by biological oxygen demand in 14 d) under aerobic conditions in a standard test (OECD 301 C 'Modified MITI Test (1)', OECD, 1981a; MITI, 1992). Also in a test simulating aerobic sewage treatment (OECD 303 A 'Simulation Test - Aerobic Sewage Treatment: Coupled Units Test'; OECD, 1981b), limonene disappeared almost completely (>93.8%) during 14 days of incubation.

Biodegradation has been assessed under anaerobic conditions; there was no indication of any metabolisms, possibly because of the toxicity to micro-organisms.

The bioconcentration factor, calculated on the basis of water solubility and the log octanol/ water partition coefficient (log Kow) is 246-262, suggesting that limonene may bioaccumulate in fish and other aquatic species

Ecotoxicity: Technical limonene is practically nontoxic to birds on a subacute dietary basis, and is slightly toxic to freshwater fish and invertebrates on an acute basis. for d-limonene:

LD50 Colinus virginianus (Bobwhite quail, 16 weeks old) oral >2000 mg/kg

LC50 Colinus virginianus (Bobwhite quail, 10 day old) dietary >5620 ppm/8 days

LC50 Colinus virginianus (Bobwhite quail, 14 day old) dietary >5000 ppm/8 days

LC50 Anas platyrhynchos (Mallard duck, 14 day old) dietary >5000 ppm/8 days

LC50 Oncorhynchus mykiss (Rainbow trout) 80 ppm/96 hr (95% confidence limit: 71.4-88.7 ppm); static /92% Al formulated product

LC50 Oncorhynchus mykiss (Rainbow trout) 568 ppm/96 hr (95% confidence limit: 437-852 ppm); static /4.0% Al formulated product

EC50 Daphnia magna (Water flea, <24 hr old; intoxication, immobilization) 17 ppm/48 hr (95% confidence limit: 11-33 ppm); static /4.0% Al formulated product

LC50 Pimephales promelas (Fathead minnow) 966 ppm/96 hr (95% confidence limit: 740-1652 ppm); static /4.0% Al formulated product

LC50 Pimephales promelas (Fathead minnow) 38.5 mg/L/96 hr; flow through /from table/ LC50

Leuciscus idus (Golden orfe) 32 mg/L/48 hr /Conditions of bioassay not specified in source examined

The acute toxicity of d-limonene ranges from slight to high for aquatic organisms. The lowest acute toxicity values (EC50 or LC50) identified were approximately 0.4 mg/litre for Daphnia (US EPA, 1990b) and 0.7 mg/litre for fish (US EPA, 1990a,b). The no-observed-effect concentration (NOEC) for

green algae is approximately 4 mg/litre (US EPA, 1990a). The acute toxicity (EC50 or LC50) of dipentene to Daphnia and fish is about 50-70 times lower than that for d-limonene (US EPA, 1990b). No studies were identified on the chronic toxicity of limonene to aquatic organisms.

DO NOT discharge into sewer or waterways

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
d-limonene	HIGH	HIGH
gamma-terpinene	HIGH	HIGH
beta-pinene	HIGH	HIGH
myrcene	HIGH	HIGH
terpinolene	HIGH	HIGH
alpha-pinene	HIGH	HIGH
alpha-terpinene	HIGH	HIGH

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
C14-20 aliphatics (<=2% aromatics)	LOW (BCF = 159)
d-limonene	HIGH (LogKOW = 4.8275)
gamma-terpinene	MEDIUM (LogKOW = 4.5)
beta-pinene	MEDIUM (LogKOW = 4.16)

Ingredient	Bioaccumulation
myrcene	MEDIUM (LogKOW = 4.17)
terpinolene	MEDIUM (LogKOW = 4.47)
alpha-pinene	MEDIUM (LogKOW = 4.44)
alpha-terpinene	MEDIUM (LogKOW = 4.25)

12.4. Mobility in soil

Ingredient	Mobility
d-limonene	LOW (KOC = 1324)
gamma-terpinene	LOW (KOC = 1324)
beta-pinene	LOW (KOC = 1204)
myrcene	LOW (KOC = 1269)
terpinolene	LOW (KOC = 1324)
alpha-pinene	LOW (KOC = 1204)
alpha-terpinene	LOW (KOC = 1324)

12.5. Results of PBT and vPvB assessment

	P	В	Т	
Relevant available data	Not Available	Not Available	Not Available	
PBT	×	×	×	
vPvB	X	×	×	
PBT Criteria fulfilled?				
vPvB			No	

12.6. Endocrine Disruption Properties

The evidence linking adverse effects to endocrine disruptors is more compelling in the environment than it is in humans. Endocrine distruptors profoundly alter reproductive physiology of ecosystems and ultimately impact entire populations. Some endocrine-disrupting chemicals are slow to break-down in the environment. That characteristic makes them potentially hazardous over long periods of time. Some well established adverse effects of endocrine disruptors in various wildlife species include; eggshell-thinning, displayed of characteristics of the opposite sex and impaired reproductive development. Other adverse changes in wildlife species that have been suggested, but not proven include; reproductive abnormalities, immune dysfunction and skeletal deformaties.

12.7. Other adverse effects

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

SECTION 13 Disposal considerations

13.1. Waste treatment methods

Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.

A Hierarchy of Controls seems to be common - the user should investigate:

- ► Reduction
- ► Reuse
- ► Recycling
- Disposal (if all else fails)

Product / Packaging disposal

This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.

- DO NOT allow wash water from cleaning or process equipment to enter drains.
- It may be necessary to collect all wash water for treatment before disposal.
- In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
- Where in doubt contact the responsible authority.
- 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.

Waste treatment options Not Available

Sewage disposal options Not Available

Not Available

SECTION 14 Transport information

Labels Required



Land transport (ADR-RID)

14.1. UN number	1950	1950		
14.2. UN proper shipping name	AEROSOLS	AEROSOLS		
14.3. Transport hazard class(es)	Class			
14.4. Packing group	Not Applica	Not Applicable		
14.5. Environmental hazard	Environmer	Environmentally hazardous		
	Hazard id	lentification (Kemler)	Not Applicable	
	Classifica	ition code	5F	
14.6. Special precautions for	Hazard Label		2.1	
user	Special provisions		190 327 344 625	
	Limited q	uantity	1 L	
	Tunnel R	estriction Code	2 (D)	

Air transport (ICAO-IATA / DGR)

14.1. UN number	1950					
14.2. UN proper shipping name	Aerosols					
	ICAO/IATA Class	TA Class 2.1				
14.3. Transport hazard class(es)	ICAO / IATA Subrisk	Not Applicable				
0.000(00)	ERG Code	ERG Code 10L				
14.4. Packing group	Not Applicable	Not Applicable				
14.5. Environmental hazard	Environmentally hazardo	Environmentally hazardous				
	Special provisions		A145 A167 A802; A1 A145 A167 A802			
14.6. Special precautions for user	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)

14.1. UN number	1950		
14.2. UN proper shipping name	AEROSOLS		
14.3. Transport hazard class(es)	IMDG Class 2.1 IMDG Subrisk Not Applicable		
14.4. Packing group	Not Applicable		
14.5. Environmental hazard	Marine Pollutant		
14.6. Special precautions for user		, S-U 90 277 327 344 381 959 0 ml	

Inland waterways transport (ADN)

14.1. UN number	1950		
14.2. UN proper shipping name	AEROSOLS		
14.3. Transport hazard class(es)	2.1 Not Applicable		
14.4. Packing group	Not Applicable		
14.5. Environmental hazard	Environmentally hazardous		
14.6. Special precautions for user	Classification code 5F Special provisions 190; 327; 344; 625 Limited quantity 1 L		

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

Not Applicable

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

Hot Transport in Bank in accordance with month of Junior V and the imobe code		
Product name	Group	
C14-20 aliphatics (<=2% aromatics)	Not Available	
1,3,3,3-tetrafluoropropene	Not Available	
d-limonene	Not Available	
gamma-terpinene	Not Available	
beta-pinene	Not Available	
myrcene	Not Available	
terpinolene	Not Available	
alpha-pinene	Not Available	
alpha-terpinene	Not Available	

14.9. Transport in bulk in accordance with the ICG Code

Product name	Ship Type
C14-20 aliphatics (<=2% aromatics)	Not Available
1,3,3,3-tetrafluoropropene	Not Available
d-limonene	Not Available
gamma-terpinene	Not Available
beta-pinene	Not Available
myrcene	Not Available
terpinolene	Not Available
alpha-pinene	Not Available
alpha-terpinene	Not Available

SECTION 15 Regulatory information

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

C14-20 aliphatics (<=2% aromatics) is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

EU REACH Regulation (EC) No 1907/2006 - Annex XVII (Appendix 2) Carcinogens: category 1B (Table 3.1)/category 2 (Table 3.2)

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

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

1,3,3,3-tetrafluoropropene is found on the following regulatory lists

Europe EC Inventory

d-limonene is found on the following regulatory lists

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

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

gamma-terpinene is found on the following regulatory lists

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

beta-pinene is found on the following regulatory lists

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

myrcene is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

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

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

terpinolene is found on the following regulatory lists

Europe EC Inventory	European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
alpha-pinene is found on the following regulatory lists	
Europe EC Inventory	European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)
alpha-terpinene is found on the following regulatory lists	
Europe EC Inventory	European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : Directives 98/24/EC, - 92/85/EEC, - 94/33/EC, - 2008/98/EC, - 2010/75/EU; Commission Regulation (EU) 2020/878; Regulation (EC) No 1272/2008 as updated through ATPs.

15.2. Chemical safety assessment

No Chemical Safety Assessment has been carried out for this substance/mixture by the supplier.

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (C14-20 aliphatics (<=2% aromatics); d-limonene; gamma-terpinene; beta-pinene; myrcene; terpinolene; alpha-terpinene)
China - IECSC	No (1,3,3,3-tetrafluoropropene)
Europe - EINEC / ELINCS / NLP	No (1,3,3,3-tetrafluoropropene)
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	No (1,3,3,3-tetrafluoropropene)
Philippines - PICCS	No (1,3,3,3-tetrafluoropropene)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (1,3,3,3-tetrafluoropropene; alpha-terpinene)
Vietnam - NCI	Yes
Russia - FBEPH	No (1,3,3,3-tetrafluoropropene)
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	11/08/2021
Initial Date	21/11/2017
Full text Risk and Hazard codes	

Flammable liquid and vapour.
Contains gas under pressure; may explode if heated.
Harmful if swallowed.
Harmful if swallowed, in contact with skin or if inhaled.
Causes serious eye irritation.
May cause respiratory irritation.
Suspected of damaging fertility.
Very toxic to aquatic life.
Very toxic to aquatic life with long lasting effects.

Other information

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

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered. For detailed advice on Personal Protective Equipment, refer to the following EU CEN Standards:

EN 166 Personal eye-protection

EN 340 Protective clothing

EN 374 Protective gloves against chemicals and micro-organisms

EN 13832 Footwear protecting against chemicals

EN 133 Respiratory protective devices

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average PC—STEL: Permissible Concentration-Short Term Exposure Limit IARC: International Agency for Research on Cancer

ACGIH: American Conference of Governmental Industrial Hygienists

STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit。

IDLH: Immediately Dangerous to Life or Health Concentrations

ES: Exposure Standard

OSF: Odour Safety Factor

NOAEL :No Observed Adverse Effect Level LOAEL: Lowest Observed Adverse Effect Level

TLV: Threshold Limit Value LOD: Limit Of Detection OTV: Odour Threshold Value BCF: BioConcentration Factors BEI: Biological Exposure Index

AIIC: Australian Inventory of Industrial Chemicals

DSL: Domestic Substances List NDSL: Non-Domestic Substances List

IECSC: Inventory of Existing Chemical Substance in China

EINECS: European INventory of Existing Commercial chemical Substances

ELINCS: European List of Notified Chemical Substances

NLP: No-Longer Polymers

ENCS: Existing and New Chemical Substances Inventory

KECI: Korea Existing Chemicals Inventory NZIoC: New Zealand Inventory of Chemicals

PICCS: Philippine Inventory of Chemicals and Chemical Substances

TSCA: Toxic Substances Control Act TCSI: Taiwan Chemical Substance Inventory INSQ: Inventario Nacional de Sustancias Químicas

NCI: National Chemical Inventory

FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

Reason For Change

A-2.00 - Added UFI number and safety data sheet format update