

### **MG Chemicals UK Limited**

Version No: A-1.00

Safety Data Sheet (Conforms to Regulation (EU) No 2015/830)

Issue Date:25/03/2020 Revision Date: 25/03/2020 L.REACH.GBR.EN

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

### 1.1. Product Identifier

Product name	8361-P	
Synonyms	SDS Code: 8361-Pen; 8361-P	
Other means of identification	Label & Adhesive Remover Pen	

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

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Relevant identified uses	Adhesive and label 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

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

Classification according to regulation (EC) No 1272/2008 [CLP] [1]	H226 - Flammable Liquid Category 3, H336 - Specific target organ toxicity - single exposure Category 3 (narcotic effects), H315 - Skin Corrosion/Irritation Category 2, H317 - Skin Sensitizer Category 1, H410 - Chronic Aquatic Hazard Category 1, H304 - Aspiration Hazard Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

### 2.2. Label elements

Hazard pictogram(s)









DANGER SIGNAL WORD

### Hazard statement(s)

H226	Flammable liquid and vapour.			
H336	May cause drowsiness or dizziness.			
H315	Causes skin irritation.			
H317	May cause an allergic skin reaction.			
H410	Very toxic to aquatic life with long lasting effects.			
H304	May be fatal if swallowed and enters airways.			

P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.		
P271	Use in a well-ventilated area.		
P280	Wear protective gloves/protective clothing/eye protection/face protection.		
P240	Ground and bond container and receiving equipment.		
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.		
P242	Use non-sparking tools.		
P243	P243 Take action to prevent static discharges.		
P261	P261 Avoid breathing mist/vapours/spray.		
P273	Avoid release to the environment.		
P272	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.			
P321	Specific treatment (see advice on this label).			
P331	Do NOT induce vomiting.			
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.			
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.			
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].			
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.			

### Precautionary statement(s) Storage

P403+P235	Store in a well-ventilated place. Keep cool.	
P405	Store locked up.	

### Precautionary statement(s) Disposal

P501 Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.	
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### 2.3. Other hazards

d-limonene	Listed in the Europe Regulation (EC) No 1907/2006 - Annex XVII (Restrictions may apply)
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### SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

### 3.1.Substances

See 'Composition on ingredients' in Section 3.2

### 3.2.Mixtures

O.Z.IIII.ACI O			
1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP]
1.64742-47-8 2.265-149-8 3.649-422-00-2 4.01-2119484819-18-XXXX	73	distillates, petroleum, light, hydrotreated	Aspiration Hazard Category 1; H304 [2]
1.5989-27-5 2.227-813-5 3.601-029-00-7 4.01-2120766421-57- XXXX 01-2119529223-47-XXXX	27	d-limonene	Flammable Liquid Category 3, Chronic Aquatic Hazard Category 1, Acute Aquatic Hazard Category 1, Skin Corrosion/Irritation Category 2, Skin Sensitizer Category 1; H226, H410, H400, H315, H317 [2]
Legend:	Legend: 1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 3. Classification drawn from C&L * EU IOELVs available		

### **SECTION 4 FIRST AID MEASURES**

### 4.1. Description of first aid measures

If this product comes in contact with the eyes:

▶ Wash out immediately with fresh running water.

### Eye Contact

- 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.
- ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention.
- Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

Skin Contact	If skin contact occurs:  Immediately remove all contaminated clothing, including footwear.  Flush skin and hair with running water (and soap if available).  Seek medical attention in event of irritation.
Inhalation	<ul> <li>If fumes, aerosols or combustion products are inhaled remove from contaminated area.</li> <li>Other measures are usually unnecessary.</li> </ul>
Ingestion	<ul> <li>If swallowed do NOT induce vomiting.</li> <li>If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.</li> <li>Observe the patient carefully.</li> <li>Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.</li> <li>Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.</li> <li>Seek medical advice.</li> <li>If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.</li> </ul>

#### 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

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours. 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
- 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

### **SECTION 5 FIREFIGHTING MEASURES**

### 5.1. Extinguishing media

- Foam.
- Dry chemical powder.
- ► BCF (where regulations permit).
- Carbon dioxide.
- Water spray or fog Large fires only.

### 5.2. Special hazards arising from the substrate or mixture

Alert Fire Brigade and tell them location and nature of hazard.
May be violently or explosively reactive.

- Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course.
  - ▶ If safe, switch off electrical equipment until vapour fire hazard removed.
  - ▶ Use water delivered as a fine spray to control fire and cool adjacent area.
  - Avoid spraying water onto liquid pools.
  - ▶ DO NOT approach containers suspected to be hot.
  - ▶ Cool fire exposed containers with water spray from a protected location.
  - If safe to do so, remove containers from path of fire.

Fire Fighting

- Liquid and vapour are flammable.
- ▶ Moderate fire hazard when exposed to heat or flame. ► Vapour forms an explosive mixture with air.
- Moderate explosion hazard when exposed to heat or flame.
- ▶ Vapour may travel a considerable distance to source of ignition.
- Heating may cause expansion or decomposition leading to violent rupture of containers.
- On combustion, may emit toxic fumes of carbon monoxide (CO).

#### Fire/Explosion Hazard Combustion products include:

carbon monoxide (CO)

carbon dioxide (CO2)

other pyrolysis products typical of burning organic material.

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

of potentially explosive peroxides.

### **SECTION 6 ACCIDENTAL RELEASE MEASURES**

### 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

Environmental hazard - contain spillage.

- Remove all ignition sources
- ► Clean up all spills immediately.
- Minor Spills
- Avoid breathing vapours and contact with skin and eyes.
  - ▶ Control personal contact with the substance, by using protective equipment.
  - ► Contain and absorb small quantities with vermiculite or other absorbent material.
  - Wipe up.
  - ► Collect residues in a flammable waste container.

Environmental hazard - contain spillage.

Chemical Class: aliphatic hydrocarbons

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

#### 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- pillow	2	throw	pitchfork	DGC, RT
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
expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC
polypropylene - mat	4	throw	skiploader	DGC, RT

### **Major Spills**

### Legend

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 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 er
- ▶ Prevent, by any means available, spillage from entering drains or water course.
- ► Consider evacuation (or protect in place).
- ▶ No smoking, naked lights or ignition sources.
- ► Increase ventilation.
- ▶ Stop leak if safe to do so.
- ▶ Water spray or fog may be used to disperse /absorb vapour.
- ▶ Contain spill with sand, earth or vermiculite.
- ▶ Use only spark-free shovels and explosion proof equipment.
- ▶ Collect recoverable product into labelled containers for recycling.
- ▶ Absorb remaining product with sand, earth or vermiculite.
- ► Collect solid residues and seal in labelled drums for disposal.
- Wash area and prevent runoff into drains.
- ▶ If contamination of drains or waterways occurs, advise emergency services.

### 6.4. Reference to other sections

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

### **SECTION 7 HANDLING AND STORAGE**

#### 7.1. Precautions for safe handling

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.

- ▶ Containers, even those that have been emptied, may contain explosive vapours.
- ▶ Do NOT cut, drill, grind, weld or perform similar operations on or near containers.
- ▶ Avoid all personal contact, including inhalation.
- Wear protective clothing when risk of overexposure 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 generation of static electricity.
  - ► DO NOT use plastic buckets
  - ▶ Earth all lines and equipment
  - Use spark-free tools when handling.

  - Avoid contact with incompatible materials.
  - When handling, DO NOT eat, drink or smoke.
  - Keep containers securely sealed when not in use.
  - Avoid physical damage to containers.
  - Always wash hands with soap and water after handling.
  - Work clothes should be laundered separately.
  - Use good occupational work practice.
  - ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
  - Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
  - DO NOT allow clothing wet with material to stay in contact with skin

### Fire and explosion protection

Other information

#### See section 5

#### Consider storage under inert gas.

- ▶ Store in original containers in approved flammable liquid storage area.
- Store away from incompatible materials in a cool, dry, well-ventilated area.
- DO NOT store in pits, depressions, basements or areas where vapours may be trapped.
- No smoking, naked lights, heat or ignition sources.
- Storage areas should be clearly identified, well illuminated, clear of obstruction and accessible only to trained and authorised personnel adequate security must be provided so that unauthorised personnel do not have access.
- Store according to applicable regulations for flammable materials for storage tanks, containers, piping, buildings, rooms, cabinets, allowable quantities and minimum storage distances.
- Use non-sparking ventilation systems, approved explosion proof equipment and intrinsically safe electrical systems.
- Have appropriate extinguishing capability in storage area (e.g. portable fire extinguishers dry chemical, foam or carbon dioxide) and flammable gas detectors.
- Keep adsorbents for leaks and spills readily available.
- ▶ Protect containers against physical damage and check regularly for leaks.
- ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

In addition, for tank storages (where appropriate):

- ▶ Store in grounded, properly designed and approved vessels and away from incompatible materials.
- For bulk storages, consider use of floating roof or nitrogen blanketed vessels; where venting to atmosphere is possible, equip storage tank vents with flame arrestors; inspect tank vents during winter conditions for vapour/ ice build-up.
- ► Storage tanks should be above ground and diked to hold entire contents.

### 7.2. Conditions for safe storage, including any incompatibilities

- ▶ Glass container is suitable for laboratory quantities
- Packing as supplied by manufacturer.
- ▶ Plastic containers may only be used if approved for flammable liquid.
- Check that containers are clearly labelled and free from leaks.
- For low viscosity materials (i): Drums and jerry cans must be of the non-removable head type. (ii): Where a can is to be used as an inner package, the can must have a screwed enclosure.
- For materials with a viscosity of at least 2680 cSt. (23 deg. C) Suitable containe
  - For manufactured product having a viscosity of at least 250 cSt. (23 deg. C)
  - Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C); (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used.
  - Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages
  - In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic

- ▶ 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
- ▶ 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.

## Storage incompatibility

- 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.

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
- ▶ Avoid reaction with oxidising agents

### 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
distillates, petroleum, light, hydrotreated	Oral 19 mg/kg bw/day (Systemic, Chronic) *	Not Available
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) *	5.4 µg/L (Water (Fresh)) 0.54 µg/L (Water - Intermittent release) 1.32 mg/kg sediment dw (Sediment (Fresh Water)) 0.13 mg/kg sediment dw (Sediment (Marine)) 0.262 mg/kg soil dw (Soil) 1.8 mg/L (STP) 3.33 mg/kg food (Oral)

<sup>\*</sup> Values for General Population

### OCCUPATIONAL EXPOSURE LIMITS (OEL)

### INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Not Available						

### EMERGENCY LIMITS

EWERGENCT LIMITS				
Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3

distillates, petroleum, light, hydrotreated	Mineral oil, heavy or light; (paraffin oil; Deobase, deodorized; heavy paraffinic; heavy naphthenic); distillates; includes 64741-53-3, 64741-88-4, 8042-47-5, 8012-95-1; 64742-54-7	140 mg/m3	1,500 mg/m3	8,900 mg/m3
d-limonene	Limonene, d-	15 ppm	67 ppm	170 ppm

Ingredient	Original IDLH	Revised IDLH
distillates, petroleum, light, hydrotreated	2,500 mg/m3	Not Available
d-limonene	Not Available	Not Available

#### OCCUPATIONAL EXPOSURE BANDING

Ingredient	Occupational Exposure Band Rating Occupational Exposure Band Limit			
distillates, petroleum, light, hydrotreated	Е	≤ 0.1 ppm		
d-limonene	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.			

#### MATERIAL DATA

Fragrance substance with positive human data, which is, however, not sufficient to categorise as "established contact allergen in humans" 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.

for kerosene CAS 8008-20-6

TLV TWA: 100 mg/m3 as total hydrocarbon vapour Skin A3

OEL TWA: 14 ppm, 100 mg/m3 [NIOSH, 1985]

REL TWA: 150 ppm [Shell] CEL TWA: 300 ppm, 900 mg/m3 (CEL = Chemwatch Exposure Limit)

for petroleum distillates:

CEL TWA: 500 ppm, 2000 mg/m3 (compare OSHA TWA)

(CEL = Chemwatch Exposure Limit)

NOTE H: Special requirements exist in relation to classification and labelling of this substance. This note applies to certain coal- and oil-derived substances and to certain entries for groups of 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.

For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant.

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:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity

3: Intermittent, low production.		3: High production, heavy use			
	4: Large hood or large air mass in motion	4: Small hood-local control only			

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











# Eye 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]

### Skin protection

See Hand protection below

- ▶ Wear chemical protective gloves, e.g. PVC.
- ▶ Wear safety footwear or safety gumboots, e.g. Rubber

#### NOTE:

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

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

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

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

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

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

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

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

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

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

For general applications, gloves with a thickness typically greater than  $0.35\,\mathrm{mm}$ , are recommended.

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

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

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

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

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

### **Body protection**

Hands/feet protection

### See Other protection below

- Overalls.
- ► PVC Apron.
- PVC protective suit may be required if exposure severe.
- ► Eyewash unit.
- Ensure there is ready access to a safety shower

### Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity.

- For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets).
- Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.

### 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 computergenerated selection:

8361-P Label & Adhesive Remover Pen

Material	СРІ
NITRILE	A
PVA	A
VITON	A

<sup>\*</sup> 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

### 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	Colorless		
Physical state	Liquid	Relative density (Water = 1)	0.82
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	>216
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)	>176	Molecular weight (g/mol)	Not Available
Flash point (°C)	43	Taste	Not Available
Evaporation rate	<0.3 BuAC = 1	Explosive properties	Not Available
Flammability	Flammable.	Oxidising properties	Not Available
Upper Explosive Limit (%)	7.0	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	0.6	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	0.40	Gas group	Not Available
Solubility in water	Partly miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	>4.7	VOC g/L	Not Available

### 9.2. Other information

Not Available

### **SECTION 10 STABILITY AND REACTIVITY**

ction 7.2

10.2. Chemical stability	<ul> <li>Unstable in the presence of incompatible materials.</li> <li>Product is considered stable.</li> <li>Hazardous polymerisation will not occur.</li> </ul>
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.

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

Inhalation hazard is increased at higher temperatures.

#### Inhaled

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.

Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination

## Ingestion

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).

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

## .....

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.

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

Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.

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

### Skin Contact

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.

Application of d-limonene produced moderate irritation to both intact and abraded skin. High purity d-limonene does not cause significant allergic reaction in guinea pigs; d-limonene, exposed to air for 2-months, sensitised the animals and it is surmised that allergenic compounds are formed after prolonged air contact. In human patch testing, weak or moderate reactions (erythema, swelling) have been observed. Positive eczematous responses to purified limonene were observed in 5 of 16 previously sensitised to oil of turpentine. In one study, a 39-year old male immersed one hand in a jar of solvent, ensuring that inhalation exposure was minimal. After only few minutes of exposure, the subject experienced painful itching and burning. Itching decreased after the exposure but burning continued for 10 minutes. Swelling disappeared after 100 minutes. Six hours post-exposure, a purpuric eruption was observed; this persisted for several weeks. Blood concentrations during skin exposure were low compared to those during inhalation exposure.

### Eye

Limited evidence exists, or practical experience suggests, that the material may cause eye irritation in a substantial number of individuals and/or is expected to 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.

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 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.

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 species 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. Repeated application of mildly hydrotreated oils (principally paraffinic), to mouse skin, induced skin tumours; no tumours were induced with severely hydrotreated oils.

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.

### Chronic

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.

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.

#### 8361-P Label & Adhesive Remover Pen

TOXICITY	IRRITATION
Not Available	Not Available

# distillates, petroleum, light, hydrotreated

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating)[1]
Oral (rat) LD50: >5000 mg/kg <sup>[2]</sup>	Skin: adverse effect observed (irritating) <sup>[1]</sup>

### d-limonene

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >5000 mg/kg <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
Oral (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Skin (rabbit): 500mg/24h moderate
	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>

#### Legend:

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

#### 8361-P Label & Adhesive Remover Pen

Epoxidation of double bonds is a common bioactivation pathway for alkenes. The allylic epoxides, so formed, were found to possess sensitising capacity in vivo and in vitro and to chemically reactive towards a common hexapeptide containing the most common nucleophilic amino acids. Further-more, a SAR study of potentially prohaptenic alkenes demonstrated that conjugated dienes in or in conjunction with a six-membered ring are prohaptens, whereas related alkenes containing isolated double bonds or an acyclic conjugated diene were weak or nonsensitizing compounds. This difference in sensitizing capacity of conjugated dienes as compared to alkenes with isolated double bonds was found to be due to the high reactivity and sensitizing capacity of the allylic epoxides metabolically formed from conjugated dienes.

Allergic Contact Dermatitis—Formation, Structural Requirements, and Reactivity of Skin Sensitizers.

Ann-Therese Karlberg et al: Chem. Res. Toxicol. 2008, 21, pp 53–69 http://ftp.cdc.gov/pub/Documents/OEL/06.%20Dotson/References/Karlberg\_2008.pdf

#### DISTILLATES, PETROLEUM, LIGHT, HYDROTREATED

No significant acute toxicological data identified in literature search.

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.

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.

### D-LIMONENE

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

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).

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

Sensitisation:

No data on sensitization are available on MMT(EHTG/(IOTG), but the hydrolysis products EHTG or IOTG are sensitizers. No primary irritation

data were available for TERP, but it was a sensitizer in the mouse Local Lymph Node Assay.

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

#### Repeat dose toxicity:

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

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

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

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

#### Neurotoxicity:

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

#### Immunotoxicity:

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

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

#### Genotoxicity

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

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

From the results obtained in a micronucleus test with MMT(2-EHMA), it was demonstrated that the substance was weakly genotoxic to bone marrow cells of rats and that the substance has the potential to induce damage to the mitotic spindle apparatus of the bone marrow target cells. Carcinogenicity:

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

### Toxicity to reproduction:

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

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

Tumorigenic by RTECS criteria

The following information refers to contact allergens as a group and may not be specific to this product.

Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.

Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis,

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

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

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

Axillae Bilateral axillary (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy.

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

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

Pigmentary anomalies: The term "pigmented cosmetic dermatitis" was introduced in 1973 for what had previously been known as 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 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.

### Prehaptens

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.

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

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

Renal tumours induced by limonene in male rats is 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.

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. For 'kerosenes'

Acute toxicity: Oral LD50s for three kerosenes (Jet A, CAS No. 8008-20-6 and CAS No. 64742-81-0) ranged from > 2 to >20 g/kg The dermal LD50s of the same three kerosenes were all >2.0 g//kg. Inhalation LC50 values in Sprague-Dawley rats for straight run kerosene (CAS No. 8008-20-6) and hydrodesulfurised kerosene (CAS No. 64742-81-0) were reported to be > 5 and > 5.2 mg/l, respectively. No mortalities in rats were reported in rats when exposed for eight hours to saturated vapor of deodorised kerosene (probably a desulfurised kerosene). Six hour exposures of cats to the same material produced an LC50 of >6.4 mg/l

When tested in rabbits for skin irritation, straight run kerosene (CAS No. 8008-20-6) produced "moderate" to "severe" irritation. Six additional skin irritation studies on a range of kerosenes produced "mild" to "severe" irritation.

An eye irritation in rabbits of straight run kerosene (CAS No. 8008-20-6) produced Draize scores of 0.7 and 2.0 (unwashed and washed eyes) at 1 hour. By 24 hours, the Draize scores had returned to zero. Eye irritation studies have also been reported for hydrodesulfurized kerosene and jet fuel. These materials produced more irritation in the unwashed eyes at 1 hour than had the straight run kerosene. The eye irritation persisted longer than that seen with straight run kerosene, but by day 7 had resolved.

Straight run kerosene (CAS No. 8008-20-6), Jet A, and hydrodesulfurized kerosene (CAS No. 64742-81-0) have not produced sensitisation when tested in guinea pigs

Repeat-Dose toxicity: Multiple repeat-dose toxicity studies have been reported on a variety of kerosenes or jet fuels. When applied dermally, kerosenes and jet fuels have been shown to produce dermal and systemic effects

Dose levels of 200, 1000 and 2000 mg/kg of a straight run kerosene (CAS No. 8008-20-6) were applied undiluted to the skin of male and female New Zealand white rabbits. The test material was applied 3x/week for 28 days. One male and one female in the 2000 mg/kg dose group found dead on days 10 and 24 respectively were thought to be treatment-related. Clinical signs that were considered to be treatment-related included: thinness, nasal discharge, lethargy, soiled anal area, anal discharge, wheezing. The high dose group appeared to have a treatment related mean body weight loss when compared to controls. Dose-related skin irritation was observed, ranging from "slight" to "moderate" in the low and high dose groups, respectively. Other treatment-related dermal findings included cracked, flaky and/or leathery skin, crusts and/or hair loss.

Reductions in RBC, haemoglobin and haematocrit were seen in the male dose groups. There were no treatment related effects on a variety of clinical chemistry values. Absolute and relative weights for a number of organs were normal, with the following exceptions that were judged to be treatment-related:

increased relative heart weights for the mid- and high- dose males and females,

- increased absolute and relative spleen weights in treated females, and
- differences in absolute and relative adrenal weights in both male and female treated animals (considered to be stress-related and therefore, indirectly related to treatment).

Gross necropsy findings were confined largely to the skin. Enlarged spleens were seen in the female groups. Microscopic examination of tissues taken at necropsy found proliferative inflammatory changes in the treated skin of all male and female animals in the high dose group. These changes were, in the majority of animals, accompanied by an increase in granulopoiesis of the bone marrow. Four of six high dose males had testicular changes (multifocal or diffuse tubular hypoplasia) that were considered by the study authors to be secondary to the skin and/or weight changes.

In a different study, hydrodesulfurised kerosene was tested in a thirteen-week dermal study using Sprague-Dawley rats. Test material was applied 5x/week to the skin of male and female rats at dose levels of 165, 330 and 495 mg/kg. Aside from skin irritation at the site of application, there were no treatment-related clinical signs during the study. Screening of all animals using a functional observation battery (FOB) did not find any substance-related effects. Opthalomological examination of all animals also found no treatment-related effects. There were no treatment-related effects on growth rates, hematological or clinical chemical values, or absolute or relative organ weights. Microscopic examination of tissues from animals surviving to termination found no treatment-related changes, with the exception of a minimal degree of a proliferative and inflammatory changes in the skin.

A hydrodesulfurised middle distillate (CAS no. 64742-80-9) has also been tested in a four week inhalation study. In the study, Sprague-Dawley rats were exposed to a nominal concentration of 25mg/m3 kerosene. Exposures were for approximately 6 hr/day, five days each week for four consecutive weeks. There were no treatment-related effects on clinical condition, growth rate, absolute or relative organ weights, or any of the hematological or clinical chemistry determinations. Microscopic examination found no treatment-related changes observed in any tissues.

Carcinogenicity: In addition to the repeat-dose studies discussed above, a number of dermal carcinogenicity studies have been performed on kerosenes or jet fuels. .Following the discovery that hydrodesulfurised (HDS) kerosene caused skin tumors in lifetime mouse skin painting studies, the role of dermal irritation in tumor formation was extensively studied. HDS kerosene proved to be a mouse skin tumor promoter rather than initiator, and this promotion required prolonged dermal irritation . If the equivalent dose of kerosene was applied to the skin in manner that did not cause significant skin irritation (eg, dilution with a mineral oil) no skin tumors occurred . Dermal bioavailability studies in mice confirmed that the reduced irritation seen with samples in mineral oil was not due to decreased skin penetration . The effect of chronic acanthosis on the dermal tumorigenicity of a hydrodesulfurised kerosene was studied and the author concluded that hyperplasia was essential for tumor promotion. However, the author also concluded that subacute inflammation did not appear to be a significant factor

A sample of a hydrodesulfurised kerosene has been tested in an initiation-promotion assay in male CD-1 mice. Animal survivals were not effected by exposure to the kerosene. The study's authors concluded that the kerosene was not an initiator but it did show tumor promoting activity.

In-Vitro (Genotoxicity): The potential in vitro genotoxicities of kerosene and jet fuel have been evaluated in a variety of studies. Standard Ames assays on two kerosene samples and a sample of Jet A produced negative results with/without activation. Modified Ames assays on four

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kerosenes also produced negative results (with/without activation) except for one positive assay that occurred with activation . The testing of five kerosene and jet fuel samples in mouse lymphoma assays produced a mixture of negative and positive results . Hydrodesulfurized kerosene tested in a sister chromatid exchange assay produced negative results (with/without activation)

*In-Vivo* **Genotoxicity**: Multiple *in vivo* genotoxicity studies have been done on a variety of kerosene-based materials. Four samples of kerosene were negative and a sample of Jet A was positive in *in vivo* bone marrow cytogenetic tests in Sprague-Dawley rats. One of the kerosene samples produced a positive response in male mice and negative results in females when tested in a sister chromatid exchange assay. Both deodorised kerosene and Jet A samples produced negative results in dominant lethal assays. The kerosene was administered to both mice and rats intraperitoneally, while the jet fuel was administered only to mice via inhalation.

Reproductive/Developmental Toxicity Either 0, 20, 40 or 60% (v/v) kerosene in mineral oil was applied to the skin of the rats. The dose per body weight equivalents were 0, 165, 330 and 494 mg/kg. Test material was applied daily, 7 days/week from 14 days premating through 20 days of gestation. There were no treatment-related effects on mortality and no clinical signs of toxicity were observed. There were no compound-related effects on any of the reproductive/developmental parameters. The authors concluded that the no observable effect level (NOEL) for reproductive/developmental toxicity of HDS kerosene under the treatment conditions of the study was 494 mg/kg/day.

Developmental toxicity screening studies on a kerosene and a sample of Jet A have been reported. There were no compound-related deaths in either study. While kerosene produced no clinical signs, the jet fuel produced a dose-related eye irritation (or infection). The signs of irritation lasted from 2 to 8 days with most animals showing signs for 3 days. Neither of the test materials had an effect on body weights or food consumption. Examination of offspring at delivery did not reveal any treatment-related abnormalities, soft tissue changes or skeletal abnormalities. The sex ratio of the fetuses was also unaffected by treatment with either of the compounds.

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

Legend:

X − Data either not available or does not fill the criteria for classification
✓ − Data available to make classification

### **SECTION 12 ECOLOGICAL INFORMATION**

#### 12.1. Toxicity

8361-P Label & Adhesive	ENDPOINT	TEST DURATION (HR)	\$	SPECIES	VALUE		SOURCE
Remover Pen	Not Available	ble Not Available		Not Available Not Available		Not Available	
	ENDPOINT	TEST DURATION (HR)	SPECII	ES		VALUE	SOURCE
	LC50	96	Fish			>1-mg/L	2
stillates, petroleum, light, hydrotreated	EC50	48	Crustad	cea		>1-mg/L	2
nyuroneateu	EC50	72	Algae o	Algae or other aquatic plants		>1-mg/L	2
	NOEC	3072	Fish	Fish		=1mg/L	1
d-limonene	ENDPOINT	TEST DURATION (HR)	SPECIE	S	,	VALUE	SOURCE
	LC50	96	Fish			0.199mg/L	3
	EC50	48	Crustace	ea		0.307mg/L	2
	EC50	96	Algae or	other aquatic plants	3	0.212mg/L	3
	NOEC	504	Crustace	ea	(	0.05mg/L	2
Legend:		IUCLID Toxicity Data 2. Europe ECI quatic Toxicity Data (Estimated) 4. U					

May cause long-term adverse effects in the aquatic environment.

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

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

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
- (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:

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 Ecotoxicity:

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 loss was recorded

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[isooctylmercaptoacetate (MMT(IOTG), 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. Ecotoxicity:

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).

 $MMT(EHTG) \ was \ not \ acutely \ toxic \ to \ B. \ rerio \ (LC50 > 6 \ mg/L), \ NOEC = 3.6 \ mg/L) \ and \ did \ not \ inhibit \ the \ growth \ of \ S. \ subspicatus \ (72-h \ EC50 > 1.84 \ mg/L), \ NOEC = 0.6 \ mg/L). \ The \ normalized \ heavily \ (10.10 \ mg/L) \ normalized \ normalin$ 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. 

Occupants (exhaled breath, ski oils, personal care products)

cypress, cedar and silver fir boards, sesquiterpenes

houseplants Carpets and carpet backing

Linoleum and paints/polishes containing linseed oil Latex paint

Natural rubber adhesive Photocopier toner, printed paper, styrene polymers

Environmental tobacco smoke

Soiled clothing, fabrics, bedding

Ventilation ducts and duct liners 'Urban grime

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

Overall home emissions

Soiled particle filters

neoprene Polycyclic aromatic hydrocarbons

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

Isoprene, nitric oxide, squalene, unsaturated sterols,

oxidation products

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

4-Phenylcyclohexene, 4-vinylcyclohexene, styrene, 2-ethylhexyl acrylate, unsaturated fatty acids and esters

Linoleic acid, linolenic acid

Residual monomers

Certain cleaning products, polishes, linalool, linalyl acetate and other terpenoids, longifolene Limonene, alpha-pinene, terpinolene, alpha-terpineol, and other sesquiterpenes

Isoprene, terpenes

Styrene

Styrene, acrolein, nicotine

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

Unsaturated fatty acids from plant waxes, leaf litter, and other vegetative debris; soot; diesel particles

Unsaturated fatty acids and esters, unsaturated oils,

Limonene, alpha-pinene, styrene

Major Stable Products produced following reaction with ozone.

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

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

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

Formaldehyde

Formaldehyde, acetaldehyde, glycoaldehyde, formic acid, acetic acid, hydrogen and 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

Formaldehyde, benzaldehyde

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

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-oxononanoic acid and other oxo-acids; compounds with mixed functional groups (=O, -OH, and -COOH)

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

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.

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

#### 12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
distillates, petroleum, light, hydrotreated	LOW (BCF = 159)
d-limonene	HIGH (LogKOW = 4.8275)

### 12.4. Mobility in soil

Ingredient	Mobility
d-limonene	LOW (KOC = 1324)

#### 12.5 Results of PBT and vPvB assessment

	P	В	Т
Relevant available data	Not Applicable	Not Applicable	Not Applicable
PBT Criteria fulfilled?	Not Applicable	Not Applicable	Not Applicable

### 12.6. Other adverse effects

No data available

### **SECTION 13 DISPOSAL CONSIDERATIONS**

#### 13.1. Waste treatment methods

- ► Containers may still present a chemical hazard/ danger when empty.
- ▶ Return to supplier for reuse/ recycling if possible.

### Otherwise:

- If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.
- ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product.

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
- Product / Packaging disposal

► Disposal (if all else fails)

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

- ▶ DO NOT allow wash water from cleaning or process equipment to enter drains.
- It may be necessary to collect all wash water for treatment before disposal.
- In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
- ▶ Where in doubt contact the responsible authority.
- ► Recycle wherever possible.
- Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified.
- Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material).
- ▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

Waste treatment options

Not Available

Sewage disposal options Not Available

## **SECTION 14 TRANSPORT INFORMATION**

### Labels Required



### **Excepted Quantity**

Code E1 for all modes of transport.
On air waybill, write "Dangerous Goods in Excepted Quantity"

### Land transport (ADR)

14.1. UN number	3295	
14.2. UN proper shipping name	HYDROCARBONS, LIQUID, N.O.S. (contains distillates, petroleum, light, hydrotreated)	
14.3. Transport hazard class(es)	Class 3 Subrisk Not Applicable	
14.4. Packing group	Ш	
14.5. Environmental hazard	Environmentally hazardous	

	Hazard identification (Kemler)	33
	Classification code	F1
14.6. Special precautions for	Hazard Label	3
user	Special provisions	640C; 640D
	Limited quantity	1 L
	Tunnel Restriction Code	2 (D/E)

### Air transport (ICAO-IATA / DGR)

• •			
14.1. UN number	3295		
14.2. UN proper shipping name	Hydrocarbons, liquid, n.o.s. (contains distillates, petroleum, light, hydrotreated)		
	ICAO/IATA Class 3		
14.3. Transport hazard class(es)	ICAO / IATA Subrisk Not Applicable		
ciass(cs)	ERG Code 3H		
14.4. Packing group	П		
14.5. Environmental hazard	Environmentally hazardous		
	Special provisions	A3 A324	
	Cargo Only Packing Instructions	364	
	Cargo Only Maximum Qty / Pack	60 L	
14.6. Special precautions for user	Passenger and Cargo Packing Instructions	353	
	Passenger and Cargo Maximum Qty / Pack	5 L	
	Passenger and Cargo Limited Quantity Packing Instructions	Y341	
	Passenger and Cargo Limited Maximum Qty / Pack	1L	

### Sea transport (IMDG-Code / GGVSee)

14.1. UN number	3295		
14.2. UN proper shipping name	HYDROCARBONS, LIQUID, N.O.S. (contains distillates, petroleum, light, hydrotreated)		
14.3. Transport hazard class(es)	IMDG Class 3 IMDG Subrisk Not Applicable		
14.4. Packing group	II .		
14.5. Environmental hazard	Marine Pollutant		
14.6. Special precautions for user	EMS Number F-E , S-D  Special provisions Not Applicable  Limited Quantities 1 L		

### Inland waterways transport (ADN)

14.1. UN number	3295	
14.2. UN proper shipping name	HYDROCARBONS, LIQUID, N.O.S. (contains distillates, petroleum, light, hydrotreated)	
14.3. Transport hazard class(es)	3 Not Applicable	
14.4. Packing group	II	
14.5. Environmental hazard	Environmentally hazardous	
	Classification code	F1
	Special provisions	640C 640D
14.6. Special precautions for user	Limited quantity	1L
455.	Equipment required	PP, EX, A
	Fire cones number	1

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

Not Applicable

### **SECTION 15 REGULATORY INFORMATION**

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

Chemical Footprint Project - Chemicals of High Concern List

Europe EC Inventory

Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD

Europe European Customs Inventory of Chemical Substances

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

#### D-LIMONENE IS FOUND ON THE FOLLOWING REGULATORY LISTS

EU Regulation (EC) No 1223/2009 of the European Parliament and of the Council of 30 November 2009 on cosmetic products - Annex III - List of Substances which cosmetic products must not contain except subject to the restrictions laid down

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

Europe ECHA Registered Substances - Classification and Labelling - DSD-DPD

Europe European Customs Inventory of Chemical Substances

European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1 : Carcinogenic to humans

European Trade Union Confederation (ETUC) Priority List for REACH Authorisation European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31

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

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) 2015/830; 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 - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (d-limonene; distillates, petroleum, light, hydrotreated)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - ARIPS	Yes
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

### **SECTION 16 OTHER INFORMATION**

Revision Date	25/03/2020
Initial Date	16/08/2015

### Full text Risk and Hazard codes

H400	Very toxic to aquatic life.
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### **SDS Version Summary**

Version	Issue Date	Sections Updated
1.5.1.1.1	25/03/2020	Physical Properties, Name

### 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-STEL: Permissible Concentration-Short Term Exposure Limit

IARC: International Agency for Research on Cancer

ACGIH: American Conference of Governmental Industrial Hygienists

STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit。

IDLH: Immediately Dangerous to Life or Health Concentrations

OSF: Odour Safety Factor

NOAEL :No Observed Adverse Effect Level 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

### Reason for Change

A-1.00 - Update to the emergency phone number information.