

# Safety Data Sheet



## WESTOX D-LAM 20

Date of Issue 27/08/2019  
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### 1 - IDENTIFICATION

Product Name	WESTOX D-LAM 20
Synonyms	graffiti remover; paint remover; contaminant stripper
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains d-limonene)
Recommended Use	Used for graffiti and paint removal and contaminant stripping.
Company Details	Westlegate Pty Ltd
Address	16 Frost Road Campbelltown NSW 2560 Australia
Phone	61 2 4628 5010
Email	info@westox.com
Website	www.westox.com
Emergency Contact Point	Australian Poisons Information Centre 24 Hour Service 13 11 26 Police, Fire Brigade or Ambulance 000  New Zealand Poisons Information Centre 24 Hour Service 0800 764 766 NZ Emergency Services 111

### 2 - HAZARD(S) IDENTIFICATION

Poisons Schedule	Not Applicable
Hazard Classification	Skin Corrosion/Irritation Category 2 Eye Irritation Category 2A Skin Sensitizer Category 1 Reproductive Toxicity Category 1B Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation) Chronic Aquatic Hazard Category 2

**Legend:** 1. Classification drawn from HCIS; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

#### Pictograms



#### Signal Word

**DANGER**

#### Hazard statement(s)

**H315** Causes skin irritation.  
**H319** Causes serious eye irritation.  
**H317** May cause an allergic skin reaction.  
**H360D** May damage the unborn child.  
**H335** May cause respiratory irritation.  
**H411** Toxic to aquatic life with long lasting effects

#### Precautionary statement(s) Prevention

**P201** Obtain special instructions before use.  
**P271** Use only outdoors or in a well-ventilated area.  
**P280** Wear protective gloves/protective clothing/eye protection/face protection.  
**P261** Avoid breathing mist/vapours/spray.  
**P273** Avoid release to the environment.  
**P272** Contaminated work clothing should not be allowed out of the workplace.

#### Precautionary statement(s) Response

**P308+P313** IF exposed or concerned: Get medical advice/attention.  
**P321** Specific treatment (see advice on this label).  
**P362** Take off contaminated clothing and wash before reuse.  
**P302+P352** IF ON SKIN: Wash with plenty of soap and water.  
**P305+P351+P338** IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.

**P312** Call a POISON CENTER or doctor/physician if you feel unwell.  
**P333+P313** If skin irritation or rash occurs: Get medical advice/attention.  
**P337+P313** If eye irritation persists: Get medical advice/attention.  
**P391** Collect spillage.  
**P304+P340** IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.

Precautionary statement(s) Storage **P405** Store locked up.  
**P403+P233** Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal **P501** Dispose of contents/container in accordance with local regulations.

### 3 - COMPOSITION AND INFORMATION ON INGREDIENTS

Name	CAS Number	Content %
dibasic acid ester	Not Available	10-15
N-methyl-2-pyrrolidone	872-50-4	1-9
d-limonene	5989-27-5	1-9
Ingredients determined not to be hazardous	Not Available	10-30
water	7732-18-5	NotSpec

### 4 - FIRST AID MEASURES

**Eye Contact** If this product comes in contact with the eyes:

- ▶ Wash out immediately with fresh running water.
- ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.
- ▶ 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**

- ▶ If fumes or combustion products are inhaled remove from contaminated area.
- ▶ Lay patient down. Keep warm and rested.
- ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.
- ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.
- ▶ Transport to hospital, or doctor.

**Ingestion**

- ▶ Immediately give a glass of water.
- ▶ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.
- ▶ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

#### 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.  
Treat symptomatically

### 5 - FIREFIGHTING MEASURES

#### Extinguishing Media

The product contains a substantial proportion of water, therefore there are no restrictions on the type of extinguishing media which may be used. Choice of extinguishing media should take into account surrounding areas.

Though the material is non-combustible, evaporation of water from the mixture, caused by the heat of nearby fire, may produce floating layers of combustible substances.

In such an event consider:

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

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

**Fire Incompatibility** None known.

#### Fire Fighting

- ▶ Alert Fire Brigade and tell them location and nature of hazard.
- ▶ Wear breathing apparatus plus protective gloves in the event of a fire.
- ▶ Prevent, by any means available, spillage from entering drains or water courses.
- ▶ Use firefighting procedures suitable for surrounding area.
- ▶ **DO NOT** approach containers suspected to be hot.
- ▶ Cool fire exposed containers with water spray from a protected location.
- ▶ If safe to do so, remove containers from path of fire.
- ▶ Equipment should be thoroughly decontaminated after use.

#### Fire/Explosion Hazard

- ▶ The material is not readily combustible under normal conditions.
- ▶ However, it will break down under fire conditions and the organic component may burn.

- ▶ Not considered to be a significant fire risk.
- ▶ Heat may cause expansion or decomposition with violent rupture of containers.
- ▶ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO).
- ▶ May emit acrid smoke.

Decomposes on heating and produces toxic fumes of:  
carbon dioxide (CO<sub>2</sub>)  
aldehydes  
nitrogen oxides (NO<sub>x</sub>)  
other pyrolysis products typical of burning organic material.

Hazchem

\*3Z

## 6 - ACCIDENTAL RELEASE MEASURES

### Personal precautions, protective equipment and emergency procedures

See section 8

### Environmental precautions

See section 12

### Methods and material for containment and cleaning up

#### Minor Spills

Environmental hazard - contain spillage.

- ▶ Clean up all spills immediately.
- ▶ Avoid breathing vapours and contact with skin and eyes.
- ▶ Control personal contact with the substance, by using protective equipment.
- ▶ Contain and absorb spill with sand, earth, inert material or vermiculite.
- ▶ Wipe up.
- ▶ Place in a suitable, labelled container for waste disposal.

#### Major Spills

- ▶ Clear area of personnel and move upwind.
- ▶ Alert Fire Brigade and tell them location and nature of hazard.
- ▶ Wear breathing apparatus plus protective gloves.
- ▶ Prevent, by any means available, spillage from entering drains or water course.
- ▶ Stop leak if safe to do so.
- ▶ Contain spill with sand, earth or vermiculite.
- ▶ Collect recoverable product into labelled containers for recycling.
- ▶ Neutralise/decontaminate residue (see Section 13 for specific agent).
- ▶ Collect solid residues and seal in labelled drums for disposal.
- ▶ Wash area and prevent runoff into drains.
- ▶ After clean-up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.
- ▶ If contamination of drains or waterways occurs, advise emergency services.

Environmental hazard - contain spillage.

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

## 7 - HANDLING AND STORAGE

### Precautions for safe handling

#### Safe Handling

- ▶ **DO NOT** allow clothing wet with material to stay in contact with skin.
- ▶ Avoid all personal contact, including inhalation.
- ▶ Wear protective clothing when risk of exposure occurs.
- ▶ Use in a well-ventilated area.
- ▶ Avoid contact with moisture.
- ▶ Avoid contact with incompatible materials.
- ▶ **When handling, DO NOT eat, drink or smoke.**
- ▶ Keep containers securely sealed when not in use.
- ▶ Avoid physical damage to containers.
- ▶ Always wash hands with soap and water after handling.
- ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use.
- ▶ Use good occupational work practice.
- ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
- ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.

#### Other information

- ▶ Store in original containers.
- ▶ Keep containers securely sealed.
- ▶ Store in a cool, dry, well-ventilated area.
- ▶ Store away from incompatible materials and foodstuff containers.
- ▶ Protect containers against physical damage and check regularly for leaks.
- ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

### Conditions for safe storage, including any incompatibilities

#### Suitable container

- ▶ Polyethylene or polypropylene container.
- ▶ Packing as recommended by manufacturer.
- ▶ Check all containers are clearly labelled and free from leaks.

#### Storage Incompatibility

None Known

## 8 - EXPOSURE CONTROLS AND PERSONAL PROTECTION

### Control parameters

### Occupational Exposure Limits (OEL)

#### Ingredient Data

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	N-methyl-2-pyrrolidinone	1-Methyl-2-pyrrolidone	25ppm / 103 mg/m <sup>3</sup>	309 mg/m <sup>3</sup> / 75ppm	Not Available	Not Available

#### Emergency Limits

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
N-methyl-2-pyrrolidinone	Methyl 2-pyrrolidine, 1-; (N-Methylpyrrolidone)	30 ppm	32 ppm	190 ppm
d-limonene	Limonene, d-	15 ppm	67 ppm	170 ppm

Ingredient	Original IDLH	Revised IDLH
dibasic acid ester	Not Available	Not Available
N-methyl-2-pyrrolidone	Not Available	Not Available
d-limonene	Not Available	Not Available
water	Not Available	Not Available

### Material Data

### Exposure Controls

#### Engineering Controls

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

The basic types of engineering controls are:

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

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

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

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.

An approved self-contained breathing apparatus (SCBA) may be required in some situations.

Provide adequate ventilation in warehouse or closed storage area. 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.

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.)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 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.

## Personal Protective Equipment



### 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/hands/ feet protection

- ▶ 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 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/other protection

- ▶ Overalls.
- ▶ PVC Apron.
- ▶ Barrier cream.
- ▶ Skin cleansing cream.
- ▶ Eye wash unit.

### Glove selection index

Glove selection is based on a modified presentation of the:

**"Forsberg Clothing Performance Index".**

The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:

Material	Rating
BUTYL	POOR
BUTYL/NEOPRENE	POOR
NAT+NEOPR+NITRILE	POOR
NATURAL RUBBER	POOR
NATURAL+NEOPRENE	POOR
NEOPRENE	POOR
NEOPRENE/NATURAL	POOR
NITRILE	POOR
PE	POOR
PE/EVAL/PE	POOR
PVA	POOR
PVC	POOR
PVDC/PE/PVDC	POOR
SARANEX-23	POOR
SARANEX-23 2-PLY	POOR
TEFLON	POOR
VITON	POOR
VITON/NEOPRENE	POOR

<b>Good</b>	Best Selection
<b>Satisfactory</b>	May degrade after 4 hours continuous immersion
<b>Poor</b>	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 AK Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-face Respirator	Powered Air-Respirator
Up to 5 x ES	AK-AUS / Class 1	-	AK-PAPR-AUS / Class 1
Up to 25 x ES	Air-line*	AK-2	AK-PAPR-2
Up to 50 x ES	-	AK-3	-
50+ x ES	-	Air-line**	-

^ - Full-face

A (All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide (HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO<sub>2</sub>), G = Agricultural chemicals, K = Ammonia(NH<sub>3</sub>), 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.

## 9 - PHYSICAL AND CHEMICAL PROPERTIES

### General Information

**Appearance** Buff to cream coloured, viscous, fibrous and thixotropic liquid; mixes with water. NOTE: The product does not sustain combustion. Fire point >100 degC. Flash point: 62 degC (PMCC)

<b>Physical state</b>	Liquid	<b>Relative density (Water = 1)</b>	1.02-1.04
<b>Odour</b>	Not Available	<b>Partition coefficient n-octanol/water</b>	Not Available
<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	Not Applicable

pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

## 10 - STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> <li>▶ Unstable in the presence of incompatible materials</li> <li>▶ Product is considered stable</li> <li>▶ Hazardous polymerisation will not occur</li> </ul>
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

## 11 - TOXICOLOGICAL INFORMATION

Inhaled	<p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p>
Ingestion	<p>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.</p> <p>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).</p> <p>The material has <b>NOT</b> been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.</p>
Skin Contact	<p>The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either</p> <ul style="list-style-type: none"> <li>▶ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or</li> <li>▶ produces significant, but moderate, 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.</li> </ul> <p>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.</p> <p>At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material.</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
Eye	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>
Chronic	<p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>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. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>The teratogenic potential, subchronic and long-term inhalation toxicity of N-methyl-2-pyrrolidone (NMP) has been studied in rats. No evidence of nephrotoxicity was seen.</p> <p>No carcinogenic effects were observed. Very high doses are embryotoxic to rats and mice. Reproductive effects have been reported in animals. 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.</p>

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 resignification 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 litre. This requirement is based on the published literature mentioning sensitising properties when containing peroxides. Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS] May cause harm to the unborn child.

<b>WESTOX D-LAM 20</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
<b>dibasic acid ester</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Eye (rabbit): Irritant Skin (human): SEVERE
<b>N-methyl-2-pyrrolidone</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rat) LD50: 2500-5000 mg/kg <sup>[2]</sup>	Eye (rabbit): 100 mg - moderate
	Inhalation (rat) LC50: 8290.5297 mg/l/4H <sup>[2]</sup>	
	Oral (rat) LD50: 3914 mg/kg <sup>[2]</sup>	
<b>d-limonene</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	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>
<b>water</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral (rat) LD50: >90000 mg/kg <sup>[2]</sup>	Not Available

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

#### DIBASIC ACID ESTER

The family of dibasic (methyl) esters (DBEs) comprise dimethyl succinate (DMS, CAS No. 106-65-0), dimethyl glutarate (DMG, CAS No. 1119-40-0), and dimethyl adipate (DMA, CAS No. 627-93-0), and their mixture DBE (CAS No. 95481-62-2). A crude dibasic ester mixture is distilled to produce DMS, DMG, and DMA and three other fractions that are mixtures of these esters generally composed of 10-25, 55-65, and 15-25% DMA, DMG, and DMS, respectively. The three discrete compounds are all short four-to six-carbon straight-chain dicarboxylic acid dimethyl esters differing



incrementally by one carbon atom. The four members of the category produce similar levels of acute and repeated-dose toxicity in experimental animals. DBEs have very low acute oral toxicities with LD50s in rats generally > 5,000 mg/kg (with two exceptions reported as >500 and <5,000 mg/kg b.wt. for DBE (the mixture) and DMS).

By skin absorption, DBEs have a low order of acute toxicity to rabbits with dermal LD50s of 3,000 mg/kg. Based upon the most recent GLP studies, DBEs are not considered to produce primary dermal irritation as defined in EPA Guidelines. Earlier studies did show moderate irritation in one of six rabbits, but these results were not repeated in later studies. All four DBE materials are considered to produce eye irritation as defined by EPA Guidelines. Mild to moderate irritation involving the cornea was observed in rabbits with recovery by 7 days. DBEs are not skin sensitizers, and are not harmful via skin or inhalation exposures. DBE is slightly toxic by inhalation with 1- and 4-hour LC50s in rats of > 10.7 and > 11 mg/L, respectively. In subchronic inhalation studies with all four DBEs, degeneration of the olfactory epithelium of the nose was observed. This change in the nasal tissues is related to enzymatic hydrolysis of DBE within the nasal cavity. However, risk to human nasal tissue due to DBE toxicity is likely to be reduced when compared to rats since DBEs are hydrolysed more slowly in humans. No information is available on the carcinogenic potential of DBEs. A range of studies with DMS, DMG, DMA and DBE did not produce genetic damage in animals or bacterial cell cultures. DBE was positive in one study with cultured mammalian cells, but the positive findings were not apparent when the assay was repeated. Testing in rats indicates DBEs are not developmental or reproductive toxicants.

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

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

## **N-METHYL-2-PYRROLIDONE**

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

for N-methyl-2-pyrrolidone (NMP):

**Acute toxicity:** In rats, NMP is absorbed rapidly after inhalation, oral, and dermal administration, distributed throughout the organism, and eliminated mainly by hydroxylation to polar compounds, which are excreted via urine. About 80% of the administered dose is excreted as NMP and NMP metabolites within 24 h. A probably dose-dependent yellow coloration of the urine in rodents is observed. The major metabolite is 5-hydroxy-N-methyl-2-pyrrolidone. Studies in humans show comparable results. Dermal penetration through human skin has been shown to be very rapid. NMP is rapidly biotransformed by hydroxylation to 5-hydroxy-N-methyl-2-pyrrolidone, which is further oxidized to N-methylsuccinimide; this intermediate is further hydroxylated to 2-hydroxy-N-methylsuccinimide. These metabolites are all colourless. The excreted amounts of NMP metabolites in the urine after inhalation or oral intake represented about 100% and 65% of the administered doses, respectively.

NMP has a low potential for skin irritation and a moderate potential for eye irritation in rabbits. Repeated daily doses of 450 mg/kg body weight administered to the skin caused painful and severe haemorrhage and eschar formation in rabbits. These adverse effects have not been seen in workers occupationally exposed to pure NMP, but they have been observed after dermal exposure to NMP used in cleaning processes. No sensitisation potential has been observed.

In acute toxicity studies in rodents, NMP showed low toxicity. Uptake of oral, dermal, or inhaled acutely toxic doses causes functional disturbances and depressions in the central nervous system. Local irritation effects were observed in the respiratory tract when NMP was inhaled and in the pyloric and gastrointestinal tracts after oral administration. In humans, there was no irritative effect in the respiratory system after an 8-h exposure to 50 mg/m<sup>3</sup>.

**Repeat dose toxicity:** There is no clear toxicity profile of NMP after multiple administration. In a 28-day dietary study in rats, a compound-related decrease in body weight gain was observed in males at 1234 mg/kg body weight and in females at 2268 mg/kg body weight. Testicular degeneration and atrophy in males and thymic atrophy in females were observed at these dose levels. The no-observed-adverse-effect level (NOAEL) was 429 mg/kg body weight in males and 1548 mg/kg body weight in females. In a 28-day intubation study in rats, a dose-dependent increase in relative liver and kidney weights and a decrease in lymphocyte count in both sexes were observed at 1028 mg/kg body weight. The NOAEL in this study was 514 mg/kg body weight. In another rat study, daily dietary intake for 90 days caused decreased body weights at doses of 433 and 565 mg/kg body weight in males and females, respectively. There were also neurobehavioural effects at these dose levels. The NOAELs in males and females were 169 and 217 mg/kg body weight, respectively.

The toxicity profile after exposure to airborne NMP depends strongly on the ratio of vapour to aerosol and on the area of exposure (i.e., head-only or whole-body exposure). Because of higher skin absorption for the aerosol, uptake is higher in animals exposed to aerosol than in those exposed to vapour at similar concentrations. Studies in female rats exposed head only to 1000 mg/m<sup>3</sup> showed only minor nasal irritation, but massive mortality and severe effects on major organs were observed when the females were whole-body exposed to the same concentration of coarse droplets at high relative humidity. Several studies in rats following repeated exposure to NMP at concentrations between 100 and 1000 mg/m<sup>3</sup> have shown systemic toxicity effects at the lower dose levels. In most of the studies, the effects were not observed after a 4-week observation period. In rats, exposure to 3000 mg NMP/m<sup>3</sup> (head only) for 6 h/day, 5 days/week, for 13 weeks caused a decrease in body weight gain, an increase in erythrocytes, haemoglobin, haematocrit, and mean corpuscular volume, decreased absolute testis weight, and cell loss in the germinal epithelium of the testes. The NOAEL was 500 mg/m<sup>3</sup>.

There are no data in humans after repeated-dose exposure.

**Carcinogenicity:** NMP did not show any clear evidence for carcinogenicity in rats exposed to concentrations up to 400 mg/m<sup>3</sup> in a long-term inhalation study.

**Genotoxicity:** The mutagenic potential of NMP is weak. Only a slight increase in the number of revertants was observed when tested in a *Salmonella* assay with base-pair substitution strains. NMP has been shown to induce aneuploidy in yeast *Saccharomyces cerevisiae* cells. No investigations regarding mutagenicity in humans were available.

**Reproductive toxicity:** In a two-generation reproduction study in rats, whole-body exposure of both males and females to 478 mg/m<sup>3</sup> of NMP vapour for 6 h/day, 7 days/week, for a minimum of 100 days (pre-mating, mating, gestation, and lactation periods) resulted in a 7% decrease in fetal weight in the F1 offspring. A 4-11% transient, non-dose-dependent decrease was observed in the average pup weight at all exposure levels tested (41, 206, and 478 mg/m<sup>3</sup>).

**Developmental toxicity:** When NMP was administered dermally, developmental toxicity was registered in rats at 750 mg/kg body weight. The observed effects were increased preimplantation losses, decreased fetal weights, and delayed ossification. The NOAEL for both developmental effects and maternal toxicity (decreased body weight gain) was 237 mg/kg body weight.

Inhalation studies in rats (whole-body exposure) demonstrated developmental toxicity as increased preimplantation loss without significant effect on implantation rate or number of live fetuses at 680 mg/m<sup>3</sup> and behavioural developmental toxicity at 622 mg/m<sup>3</sup>. In an inhalation study (whole-body exposure), the NOAEL for maternal effects was 100 mg/m<sup>3</sup>, and the NOAEL for developmental effects was 360 mg/m<sup>3</sup>.

A tolerable inhalation concentration, 0.3 mg/m<sup>3</sup>, based on mortality and organ damage, is expected to be protective against any possible reproductive toxicity. Similarly, an oral tolerable intake of 0.6 mg/kg body weight per day, based on a 90-day study, is expected to provide adequate

protection against possible reproductive effects. Because of non-existent data on the exposure of the general population and very limited information on occupational exposure, no meaningful risk characterisation can be performed

## D-LIMONENE

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

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

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

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

Renal tumours induced by limonene in male rats is thought to be sex and species specific and are not considered relevant to humans. Repeated exposure affects the amount and activity of liver enzymes, liver weight, blood cholesterol levels and bile flow in animals. Increase in liver weight is considered a physiological 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.

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 conubial contact dermatitis occur. Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma).

Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction.

This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes.

Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits.

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

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

Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a sufficient degree of fragrance contact allergens.

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

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

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

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

**Irritant reactions (including contact urticaria):** Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this. Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures.

Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported. The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested, but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen.

**Pigmentary anomalies:** The term "pigmented cosmetic dermatitis" was introduced in 1973 for what had previously been known as 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 prehapten, it is possible to prevent activation outside the body to a certain extent by different measures, e.g. prevention of air exposure during handling and storage of the ingredients and the final product, and by the addition of suitable antioxidants. When antioxidants are used, care should be taken that they will not be activated themselves and thereby form new sensitisers.

#### Prehapten

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

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

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

#### Prohapten

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

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

The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species.

Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin. These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohapten can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity.

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

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 HCl] under physiological conditions. For the MMT(EHTG) >90% conversion to MMTC occurred within 0.5 hours. For TERP, 68% of the monomethyltin portion of the compound was converted to MMTC within 1 hour. Thus, MMTC is the appropriate surrogate for mammalian toxicology studies via the oral route.

TERP is a reaction product of MMTC and dimethyltin dichloride (DMTC), Na<sub>2</sub>S, and tall oil fatty acid [a mixture of carboxylic acids, predominantly C-18].

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

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

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

#### Acute toxicity:

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

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

#### Oral:

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

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

#### Dermal:

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

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

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

#### Inhalation:

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

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

#### Irritation:

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

#### Sensitisation:

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

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

#### 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 [ $<1\text{-}3.6\text{ mg/kg bw/d}$ ]) and additional effects including increased relative thymus weights in females and urinalysis results in both sexes at higher doses.

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

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

#### Neurotoxicity:

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

The NOAEL was the next lower dose of 150 ppm (equivalent to 9.8 mg/kg bw/day in males and 10.2 mg/kg bw/day in females)

#### Immunotoxicity:

Immune function was assessed in male Sprague-Dawley rats exposed to the mixture of organotin compounds 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 organotin compounds present in drinking water delivered via PVC pipes, as the concentrations used were several orders of magnitude higher than those expected to leach from PVC pipes.

#### Genotoxicity:

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

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

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

Carcinogenicity:

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

Toxicity to reproduction:

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

SIDS Initial Assessment Profile (SIAM 23 2006)

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

Tumorigenic by RTECS criteria.

## WATER

No significant acute toxicological data identified in literature search.

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

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

✓ – Data available to make classification

## 12 - ECOLOGICAL INFORMATION

### Toxicity

Westox D-LAM 20	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available

dibasic acid ester	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available

N-methyl-2-pyrrolidone	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	464 mg/L	1
	EC50	48	Crustacea	ca.4897 mg/L	1
	EC50	72	Algae or other aquatic plants	>500 mg/L	2
	EC0	24	Crustacea	>1 mg/L	2
	NOEC	504	Crustacea	12.5 mg/L	2

d-limonene	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.199 mg/L	3
	EC50	48	Crustacea	0.307 mg/L	2
	EC50	96	Algae or other aquatic plants	0.212 mg/L	3
	NOEC	504	Crustacea	0.05 mg/L	2

water	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	897.520mg/L	3
	EC50	96	Algae or other aquatic plants	8768.874mg/L	3

Legend: Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

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

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

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

for N-methyl-2-pyrrolidinone (NMP):

log Kow : -0.44-0.1

### Environmental Fate

NMP may enter the environment as emissions to the atmosphere, as the substance is volatile and widely used as a solvent, or it may be released to water as a component of municipal and industrial wastewaters. The substance is mobile in soil, and leaching from landfills is thus a possible route of contamination of groundwater.

In air, NMP is expected to be removed by wet deposition or by photochemical reactions with hydroxyl radicals. As the substance is completely miscible in water, it is not expected to adsorb to soil, sediments, or suspended organic matter or to bioconcentrate. NMP is not degraded by chemical hydrolysis. Data from screening tests on the biodegradability of NMP show that the substance is rapidly biodegraded. This material is not expected to persist in the environment. It is water soluble and is expected to have low volatility. Hydrolysis is not expected to be an important factor in the environmental fate process for this material.

**Persistence and Degradability**  
 Biodegradation: BOD (Modified MITI Method) = 73% (28 days). BOD (Modified MITI Method) = 92% (14 days). This material is expected to be readily biodegradable.  
 Bioaccumulation: BCF = 0.16. This material is not expected to bioaccumulate.

**Ecotoxicity**  
 This material is expected to be non-hazardous to aquatic species.  
 Fish LC50 (96 h): bluegill. 832 mg/l, fathead minnow 1072 mg/l; rainbow trout 3048 mg/l  
 Daphnia magna EC50 (24 h): > 1000 mg/l  
 Algae EC50 (72 h): *Scenedesmus subspicatus* > 500 mg/l

Comparison of available data for ecotoxicity shows dibasic methyl esters (DBEs) are "slightly" to "non-toxic" to fish and aquatic invertebrates. LC50 or EC50 values for fish, invertebrates, and algae range from: 50-100 mg/L to 25.7 mg/L, 497 mg/L to 3,3 17 mg/L, and 4.4 mg/L to 11.9 mg/L, respectively. These values appear to be correlated with a gradient in solubility for the three, dimethyl esters and inversely correlated with the molecular weights.

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), 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 organotin [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  $3.83 \times 10^{-7}$  atm-m<sup>3</sup>/mol predicts MMTC will volatilize from surface water (t<sub>1/2</sub> = 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 (t<sub>1/2</sub> > 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  $3.18 \times 10^{-4}$  atm-m<sup>3</sup>/mol predicts MMT(EHTG) will volatilize from surface water (t<sub>1/2</sub> = 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 21-d EC50 for reproduction in a chronic *Daphnia magna* study was > 0.134 mg/L (NOEC = 0.134 mg/L).  
 Substances containing unsaturated carbons are ubiquitous in indoor environments. They result from many sources (see below). Most are reactive with environmental ozone and many produce stable products which are thought to adversely affect human health. The potential for surfaces in an enclosed space to facilitate reactions should be considered.

Source of unsaturated substances	Unsaturated substances (Reactive Emissions)	Major Stable Products produced following reaction with ozone.
Occupants (exhaled breath, ski oils, personal care products)	Isoprene, nitric oxide, squalene, unsaturated sterols, oleic acid and other unsaturated fatty acids, unsaturated oxidation products	Methacrolein, methyl vinyl ketone, nitrogen dioxide, acetone, 6MHQ, geranyl acetone, 4OPA, formaldehyde, nonanol, decanal, 9-oxo-nonanoic acid, azelaic acid, nonanoic acid.
Soft woods, wood flooring, including cypress, cedar and silver fir boards, houseplants	Isoprene, limonene, alpha-pinene, other terpenes and sesquiterpenes	Formaldehyde, 4-AMC, pinoaldehyde, pinic acid, pinonic acid, formic acid, methacrolein, methyl vinyl ketone, SOAs including ultrafine particles
Carpets and carpet backing	4-Phenylcyclohexene, 4-vinylcyclohexene, styrene, 2-ethylhexyl acrylate, unsaturated fatty acids and esters	Formaldehyde, acetaldehyde, benzaldehyde, hexanal, nonanal, 2-nonenal

Linoleum and paints/polishes containing linseed oil	Linoleic acid, linolenic acid	Propanal, hexanal, nonanal, 2-heptenal, 2-nonenal, 2-decenal, 1-pentene-3-one, propionic acid, n-butyric acid
Latex paint	Residual monomers	Formaldehyde
Certain cleaning products, polishes, waxes, air fresheners	Limonene, alpha-pinene, terpinolene, alpha-terpineol, linalool, linalyl acetate and other terpenoids, longifolene and other sesquiterpenes	Formaldehyde, acetaldehyde, glycoaldehyde, formic acid, acetic acid, hydrogen and organic peroxides, acetone, benzaldehyde, 4-hydroxy-4-methyl-5-hexen-1-al, 5-ethenyl-dihydro-5-methyl-2(3H)-furanone, 4-AMC, SOAs including ultrafine particles
Natural rubber adhesive	Isoprene, terpenes	Formaldehyde, methacrolein, methyl vinyl ketone
Photocopier toner, printed paper, styrene polymers	Styrene	Formaldehyde, benzaldehyde
Environmental tobacco smoke	Styrene, acrolein, nicotine	Formaldehyde, benzaldehyde, hexanal, glyoxal, N-methylformamide, nicotinaldehyde, cotinine
Soiled clothing, fabrics, bedding	Squalene, unsaturated sterols, oleic acid and other saturated fatty acids	Acetone, geranyl acetone, 6MHQ, 4OPA, formaldehyde, nonanal, decanal, 9-oxo-nonanoic acid, azelaic acid, nonanoic acid
Soiled particle filters	Unsaturated fatty acids from plant waxes, leaf litter, and other vegetative debris; soot; diesel particles	Formaldehyde, nonanal, and other aldehydes; azelaic acid; nonanoic acid; 9-oxo-nonanoic acid and other oxo-acids; compounds with mixed functional groups (=O, -OH, and -COOH)
Ventilation ducts and duct liners	Unsaturated fatty acids and esters, unsaturated oils, neoprene	C5 to C10 aldehydes
"Urban grime"	Polycyclic aromatic hydrocarbons	Oxidized polycyclic aromatic hydrocarbons
Perfumes, colognes, essential oils (e.g. lavender, eucalyptus, tea tree)	Limonene, alpha-pinene, linalool, linalyl acetate, terpinene-4-ol, gamma-terpinene	Formaldehyde, 4-AMC, acetone, 4-hydroxy-4-methyl-5-hexen-1-al, 5-ethenyl-dihydro-5-methyl-2(3H) furanone, SOAs including ultrafine particles
Overall home emissions	Limonene, alpha-pinene, styrene	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 Health Perspectives, Vol 114, October 2006

The alkali metal cyanides (and other metal cyanides) are very soluble in water. As a result, they readily dissociate into their respective anions and cations when released into water. Depending on the pH of the water, the resulting cyanide ion may then form hydrogen cyanide or react with various metals in natural water. The proportion of hydrogen cyanide formed from soluble cyanides increases as the water pH decreases. At pH <7, >99% of the cyanide ions in water are converted to hydrogen cyanide. As the pH increases, cyanide ions in the water may form complex metalocyanides in the presence of excess cyanides; however, if metals are prevalent, simple metal cyanides are formed. Volatilization is the dominant mechanism for the removal of free cyanide. At pH >9.2, most of the free cyanide should exist as HCN, a volatile form of cyanide. Wide variations in the rate of volatilization are expected since this process is affected by a number of parameters such as temperature, pH, wind speed, and cyanide concentration. Volatilization of free cyanide from concentrated solutions is most effective under conditions of high temperatures, high dissolved oxygen levels, and at increased concentrations of atmospheric carbon dioxide.

Unlike water-soluble alkali metal cyanides, insoluble metal cyanides are not expected to degrade to hydrogen cyanide. Cyanide occurs most commonly as hydrogen cyanide in water, although it can also occur as the cyanide ion, alkali and alkaline earth metal cyanides (potassium cyanide, sodium cyanide, calcium cyanide), relatively stable metalocyanide complexes (ferricyanide complex  $[\text{Fe}(\text{CN})_6]^{3-}$ ), moderately stable metalocyanide complexes (complex nickel and copper cyanide), or easily decomposable metalocyanide complexes (zinc cyanide  $[\text{Zn}(\text{CN})_2]$ , cadmium cyanide  $[\text{Cd}(\text{CN})_2]$ ). Oxidation, hydrolysis, and photolysis (photodegradation) are the three predominant chemical processes that may cause loss of simple cyanides in aquatic media.

Certain cyanides are oxidised to isocyanates by strong oxidising agents; the isocyanates may be further hydrolysed to ammonia and carbon dioxide. However, it has not yet been determined whether such oxidation and subsequent hydrolysis of isocyanate is a significant fate process in natural waters known to contain peroxy radicals. In water, hydrogen cyanide and cyanide ion exist in equilibrium with their relative concentrations primarily dependent on pH and temperature. At pH <8, >93% of the free cyanide in water will exist as undissociated hydrogen cyanide. Hydrogen cyanide can be hydrolysed to formamide, which is subsequently hydrolysed to ammonium and formate ions. However, the relatively slow rates of hydrolysis reported for hydrogen cyanide in acidic solution and of cyanides under alkaline conditions indicate that hydrolysis is not competitive with volatilisation and biodegradation for removal of free cyanide from ambient waters. At pH <9.2, most of the free cyanide in solution should exist as hydrogen cyanide, a volatile cyanide form. On the basis of Henry's law constant and the volatility characteristics associated with various ranges of Henry's law constant, volatilization is a significant and probably dominant fate process for hydrogen cyanide in surface water. The most common alkali metal cyanides (e.g., sodium and potassium cyanide) may also be lost from surface water primarily through volatilization; whereas, the sparingly soluble metal cyanides such as copper (I) cyanide are removed from water predominantly by sedimentation and biodegradation. Because volatilisation is not an important fate process for cyanide in groundwater, cyanide would be expected to persist for considerably longer periods of time in underground aquifers than in surface water.

The significance of photolysis in the fate of cyanides in water has not been fully investigated. Hydrogen cyanide and cyanide ions in aqueous solution have been found to be very resistant to photolysis by natural sunlight, except under heterogeneous photocatalytic conditions. Photocatalytic oxidation may not be significant in natural waters, however, because of significant light reduction at increasingly greater depths. In clear water or at water surfaces, some metalocyanides, such as ferrocyanides and ferricyanides, may decompose to the cyanide ion by photodissociation and subsequently form hydrogen cyanide.

Biodegradation is an important transformation process for cyanide in natural surface waters, and is dependent on such factors as cyanide concentrations, pH, temperature, availability of nutrients, and acclimation of microbes. Although the cyanide ion is toxic to microorganisms at concentrations as low as 5-10 mg/L, acclimation increases tolerance to this compound. Mixed microorganisms in sewage sludge or activated sludge acclimated to cyanide also significantly biodegrade concentrations  $\leq 100$  mg/L of most simple and complex cyanides. It is known that there is a natural attenuation of the cyanide ion and thiocyanide concentrations in waste waters, for example those obtained gold mill tails, that is due the acclimation of indigenous microflora in the tailings. A number of microorganisms have been identified that are capable of uptake, conversion, sorption, and/or precipitation of the cyanide ion, cyanate, and thiocyanate, including species of the genera, *Actinomyces*, *Alcaligenes*, *Arthrobacter*, *Bacillus*, *Micrococcus*, *Neisseria*, *Paracoccus*, *Pseudomonas*, and *Thiobacillus*. Some of these species, for example *Pseudomonas*, are capable of using the cyanide ion and thiocyanate as the sole source of carbon and nitrogen and therefore, are particularly effective at cyanide degradation. In fact, *Pseudomonas* is the basis of commercial applications for degrading the cyanide ion to ammonia and carbonate in waste waters generated in mining operations that use the cyanide ion to leach gold and other precious metals for low-grade ores. Sulfur transferases such as rhodanese are involved in substitution reactions that result in the conversion of the cyanide ion to the less toxic thiocyanate, whereas pyridoxal phosphate enzymes are involved in substitution/addition reactions that result in production of nitrile derivatives of  $\alpha$ -amino acids. These organic nitriles may then be ultimately degraded via enzyme catalysed hydrolysis to either the corresponding amino acid and ammonia or the carboxylic acid and ammonia. The cyanide hydratase and cyanidase enzymes catalyse the hydrolysis of the cyanide ion to formamide or formic acid and ammonia, respectively. In soil, cyanide present at low concentrations would biodegrade under aerobic conditions with the initial formation of ammonia, which would be converted to nitrite and nitrate in the presence of nitrifying bacteria. Under anaerobic conditions, the cyanides ion will denitrify to gaseous nitrogen. Upper limits of 200 and 2 ppm (mg/kg CN<sup>-</sup>), respectively, have been reported for uninhibited aerobic and anaerobic biodegradation of



cyanide in soil; however, these limits have not been confirmed in other studies. Cyanide ions in soil are not involved in oxidation-reduction reactions but may undergo complexation reactions with metal ions in soil.

Cyanides are sorbed by various natural media, including clays, biological solids and sediments. Hydrogen cyanide and the alkali metal cyanides are not likely to be strongly sorbed onto sediments and suspended solids because of their high water solubilities. Soluble metal cyanides may show somewhat stronger sorption than hydrogen cyanide, with the extent of sorption increasing with decreasing pH and increasing iron oxide, clay, and organic material contents of sediment and suspended solids. However, sorption is probably insignificant even for metal cyanides when compared to volatilisation and biodegradation. Cyanides are fairly mobile in soil. Mobility is lowest in soils with low pH and high concentrations of free iron oxides, positively charged particles, and clays (e.g., chlorite, kaolin, gibbsite), and highest in soils with high pH, high concentrations of free CaCO<sub>3</sub> and negatively charged particles, and low clay content. Although cyanide has a low soil sorption capability, it is usually not detected in groundwater, probably because of fixation by trace metals through complexation or transformation by soil microorganisms. In soils where cyanide levels are high enough to be toxic to microorganisms (i.e., landfills, spills), this compound may leach into groundwater. Leaching of cyanide into a shallow aquifer has been demonstrated.

Volatilisation of hydrogen cyanide would be a significant loss mechanism for cyanides from soil surfaces at a pH <9.2.

Most cyanide in the atmosphere exists almost entirely as hydrogen cyanide gas, although small amounts of metal cyanides may be present as particulate matter in the air. Hydrogen cyanide is very resistant to photolysis at wavelengths of normal sunlight. The most important reaction of hydrogen cyanide in air is the reaction with photochemically-generated hydroxyl radicals and subsequent rapid oxidation to carbon monoxide (CO) and nitric oxide (NO); photolysis and reaction with ozone are not important transformation processes, and reaction with singlet oxygen is not a significant transformation process except at stratospheric altitudes where singlet oxygen is present in significant concentrations. The rate of hydroxyl radical reaction with hydrogen cyanide in the atmosphere depends on the altitude, and the rate of the reaction is at least an order of magnitude faster at lower tropospheric altitudes (0–8 km) than at upper tropospheric altitudes (10–12 km). Based on a reaction rate constant of 3x10<sup>-14</sup> cm<sup>3</sup>/(molecule-sec) at 25 °C and assuming an average hydroxyl radical concentration of 5x10<sup>5</sup> molecules/cm<sup>3</sup>, the residence time for the reaction of hydrogen cyanide vapor with hydroxyl radicals in the atmosphere is approximately 2 years.

There is some evidence that certain metal cyanide complexes bioaccumulate in aquatic organisms. Fish from water with soluble silver and copper cyanide complexes were found to have metal cyanides in their tissues at concentrations ranging up to 168 and 304 µg/g, respectively (wet or dry weight not specified). It is difficult to evaluate the toxicologic significance of bioaccumulation of metal cyanide complexes because these compounds are much less toxic than soluble hydrogen cyanide, sodium cyanide, or potassium cyanide. There is no evidence of biomagnification of cyanides in the food chain. Accumulation of cyanide in food webs is not expected, considering the rapid detoxification of cyanide by most species and the lethal effects of large doses of cyanide.

For limonenes

**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 from 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 hydroxyl radical reaction with limonene are 4-acetyl-1-methylcyclohexene, a keto-aldehyde, formaldehyde, 3-oxobutanal, glyoxal and a C<sub>10</sub> dicarbonyl. The same carbonyls, along with formic acid and C<sub>8</sub> and C<sub>9</sub> 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(hydroxymethyl)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 (K<sub>oc</sub>) 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 cassicola*, *Diplodia gossypina* and a soil strain of *Pseudomonas* 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 K<sub>ow</sub>) is 246-262, suggesting that limonene may bioaccumulate in fish and other aquatic species.

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

for d-limonene:

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

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

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

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

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

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

LC50 *Pimephales promelas* (Fathead minnow) 966 ppm/96 hr (95% confidence limit: 740-1652 ppm); static /4.0% AI 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.**

## Persistence and degradability

**Ingredient**

**Persistence: Water/Soil**

**Persistence: Air**

Westox Dlam 20

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N-methyl-2-pyrrolidone	LOW	LOW
d-limonene	HIGH	HIGH
water	LOW	LOW

#### Bioaccumulative potential

Ingredient	Bioaccumulation
N-methyl-2-pyrrolidone	LOW (BCF = 0.16)
d-limonene	HIGH (LogKOW = 4.8275)
water	LOW (LogKOW = -1.38)

#### Mobility in soil

Ingredient	Mobility
N-methyl-2-pyrrolidone	LOW (KOC = 20.94)
d-limonene	LOW (KOC = 1324)
water	LOW (KOC = 14.3)



### 13 - DISPOSAL CONSIDERATIONS

#### Waste treatment methods

<b>Product / Packaging disposal</b>	<ul style="list-style-type: none"> <li>▶ Recycle wherever possible or consult manufacturer for recycling options.</li> <li>▶ Consult State Land Waste Authority for disposal.</li> <li>▶ Bury or incinerate residue at an approved site.</li> <li>▶ Recycle containers if possible, or dispose of in an authorised landfill.</li> </ul>
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### 14 - TRANSPORT INFORMATION

#### Labels Required

	
<b>Marine Pollutant</b>	
<b>HAZCHEM</b>	*3Z

#### Land transport (ADG)

<b>UN number</b>	3082				
<b>UN proper shipping name</b>	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains d-limonene)				
<b>Transport hazard class(es)</b>	<table> <tr> <td>Class</td><td>9</td></tr> <tr> <td>Subrisk</td><td>Not Applicable</td></tr> </table>	Class	9	Subrisk	Not Applicable
Class	9				
Subrisk	Not Applicable				
<b>Packing group</b>	III				
<b>Environmental hazard</b>	Environmentally hazardous				
<b>Special precautions for user</b>	<table> <tr> <td>Special provisions</td><td>274 331 335 375 AU01</td></tr> <tr> <td>Limited quantity</td><td>5 L</td></tr> </table>	Special provisions	274 331 335 375 AU01	Limited quantity	5 L
Special provisions	274 331 335 375 AU01				
Limited quantity	5 L				

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082 are not subject to this Code when transported by road or rail in;

(a) packaging's;

(b) IBCs; or

(c) any other receptacle not exceeding 500 kg(L).

- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

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

<b>UN number</b>	3082						
<b>UN proper shipping name</b>	Environmentally hazardous substance, liquid, n.o.s. * (contains d-limonene)						
<b>Transport hazard class(es)</b>	<table> <tr> <td>ICAO / IATA Class</td><td>9</td></tr> <tr> <td>ICAO / IATA Subrisk</td><td>Not applicable</td></tr> <tr> <td>ERG Code</td><td>9 L</td></tr> </table>	ICAO / IATA Class	9	ICAO / IATA Subrisk	Not applicable	ERG Code	9 L
ICAO / IATA Class	9						
ICAO / IATA Subrisk	Not applicable						
ERG Code	9 L						
<b>Packing group</b>	III						

**Environmental hazard** Environmentally hazardous

<b>Special precautions for user</b>	Special provisions	A97 A158 A197
	Cargo only packing instructions	964
	Cargo only maximum qty/pack	450 L
	Passenger and cargo packing instructions	964
	Passenger and cargo maximum qty/pack	450 L
	Passenger and cargo limited quantity packing instructions	Y964
	Passenger and cargo limited maximum Qty/Pack	30 kg G

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

**UN number** 3082

**UN proper shipping name** ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains d-limonene)

<b>Transport hazard class(es)</b>	IMDG	Class 9
	IMDG Subrisk	Not Applicable

**Packing group** III

**Environmental hazard** Marine Pollutant

<b>Special precautions for user</b>	EMS Number	F-A, S-F
	Special provisions	274 335 969
	Limited quantities	5 L

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

Not Applicable

**15 - REGULATORY INFORMATION**

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

**DIBASIC ACID ESTER IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Not Applicable

**N-METHYL-2-PYRROLIDONE IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards  
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals  
Australia Inventory of Chemical Substances (AICS)  
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) – Schedule 5  
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) – Schedule 6  
GESAMP/EHS Composite List - GESAMP Hazard Profiles  
IMO IBC Code Chapter 17: Summary of minimum requirements  
IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk

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

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List  
Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes  
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals  
Australia Inventory of Chemical Substances (AICS)  
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) – Appendix B (Part 3)  
GESAMP/EHS Composite List - GESAMP Hazard Profiles  
IMO IBC Code Chapter 17: Summary of minimum requirements  
IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk  
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs  
International Air Transport Association (IATA) Dangerous Goods Regulations  
International Maritime Dangerous Goods Requirements (IMDG Code)  
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

**WATER IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Inventory of Chemical Substances (AICS)  
IMO IBC Code Chapter 18: List of products to which the Code does not apply

National Inventory	Status
Australia – AICS	Yes
Canada – DSL	Yes

Canada – NDSL	No (water; d-limonene; N-methyl-2-pyrrolidone)
China – IECSC	Yes
Europe – EINEK / 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
<i>Legend</i>	<i>Yes = All CAS declared ingredients are on the inventory</i> <i>No = One or more of the CAS listed ingredients are not on the inventory and are not on the inventory and are not exempt from listing (see specific items in brackets)</i>

## 16 - OTHER RELEVANT INFORMATION

**Revision Date** 08/10/2024  
**Initial Date** 21/11/2002

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

### Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average  
 PC—STEL: Permissible Concentration-Short Term Exposure Limit  
 IARC: International Agency for Research on Cancer  
 ACGIH: American Conference of Governmental Industrial Hygienists  
 STEL: Short Term Exposure Limit  
 TEEL: Temporary Emergency Exposure Limit.  
 IDLH: Immediately Dangerous to Life or Health Concentrations  
 OSF: Odour Safety Factor  
 NOAEL: No Observed Adverse Effect Level  
 LOAEL: Lowest Observed Adverse Effect Level  
 TLV: Threshold Limit Value  
 LOD: Limit Of Detection  
 OTV: Odour Threshold Value  
 BCF: BioConcentration Factors  
 BEI: Biological Exposure Index

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