



# N3 Prep Spray

Part Number: **Not Available**  
Version No: **1.2**  
Safety Data Sheet according to OSHA HazCom Standard (2012) requirements

Issue Date: **25/09/2023**  
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L.GHS.USA.EN

## SECTION 1 Identification

### Product Identifier

Product name	N3 Prep Spray
Synonyms	Not Available
Proper shipping name	Environmentally hazardous substance, liquid, n.o.s.
Other means of identification	Not Available

### Recommended use of the chemical and restrictions on use

Relevant identified uses	Not Available
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### Name, address, and telephone number of the chemical manufacturer, importer, or other responsible party

Registered company name	N3
Address	1823 Oak Industrial Dr NE, Grand Rapids MI 49505 United States
Telephone	844-432-2266
Fax	Not Available
Website	<a href="http://www.n3nano.com">www.n3nano.com</a>
Email	contact@blacktailstudio.c

### Emergency phone number

Association / Organisation	Not Available
Emergency telephone numbers	Not Available
Other emergency telephone numbers	Not Available

## SECTION 2 Hazard(s) identification

### Classification of the substance or mixture

NFPA 704 diamond



Note: The hazard category numbers found in GHS classification in section 2 of this SDSs are NOT to be used to fill in the NFPA 704 diamond. Blue = Health Red = Fire Yellow = Reactivity White = Special (Oxidizer or water reactive substances)

Classification	Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2A, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
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### Label elements

Hazard pictogram(s)	
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Signal word **Warning**

#### Hazard statement(s)

<b>H317</b>	May cause an allergic skin reaction.
<b>H319</b>	Causes serious eye irritation.
<b>H336</b>	May cause drowsiness or dizziness.
<b>H412</b>	Harmful to aquatic life with long lasting effects.

#### Hazard(s) not otherwise classified

Not Applicable

#### Precautionary statement(s) Prevention

<b>P271</b>	Use only outdoors or in a well-ventilated area.
<b>P280</b>	Wear protective gloves, protective clothing, eye protection and face protection.
<b>P261</b>	Avoid breathing mist/vapours/spray.
<b>P273</b>	Avoid release to the environment.
<b>P264</b>	Wash all exposed external body areas thoroughly after handling.
<b>P272</b>	Contaminated work clothing must not be allowed out of the workplace.

#### Precautionary statement(s) Response

<b>P302+P352</b>	IF ON SKIN: Wash with plenty of water.
<b>P305+P351+P338</b>	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
<b>P312</b>	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
<b>P333+P313</b>	If skin irritation or rash occurs: Get medical advice/attention.
<b>P337+P313</b>	If eye irritation persists: Get medical advice/attention.
<b>P362+P364</b>	Take off contaminated clothing and wash it before reuse.
<b>P304+P340</b>	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

#### Precautionary statement(s) Storage

<b>P405</b>	Store locked up.
<b>P403+P233</b>	Store in a well-ventilated place. Keep container tightly closed.

#### Precautionary statement(s) Disposal

<b>P501</b>	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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### SECTION 3 Composition / information on ingredients

#### Substances

See section below for composition of Mixtures

#### Mixtures

CAS No	%[weight]	Name
7732-18-5	60-70	<u>water</u>
64-17-5.	10-20	<u>ethanol, denatured</u>
67-63-0	10--20	<u>isopropanol</u>
5989-27-5	<1	<u>d-limonene</u>

The specific chemical identity and/or exact percentage (concentration) of composition has been withheld as a trade secret.

### SECTION 4 First-aid measures

#### Description of first aid measures

<b>Eye Contact</b>	If this product comes in contact with eyes:
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Continued...

	<ul style="list-style-type: none"> <li>▶ Wash out immediately with water.</li> <li>▶ If irritation continues, seek medical attention.</li> <li>▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> </ul>
<b>Skin Contact</b>	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> <li>▶ Immediately remove all contaminated clothing, including footwear.</li> <li>▶ Flush skin and hair with running water (and soap if available).</li> <li>▶ Seek medical attention in event of irritation.</li> </ul>
<b>Inhalation</b>	<ul style="list-style-type: none"> <li>▶ If fumes or combustion products are inhaled remove from contaminated area.</li> <li>▶ Lay patient down. Keep warm and rested.</li> <li>▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.</li> <li>▶ 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.</li> <li>▶ Transport to hospital, or doctor, without delay.</li> </ul>
<b>Ingestion</b>	<ul style="list-style-type: none"> <li>▶ Immediately give a glass of water.</li> <li>▶ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.</li> <li>▶ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.</li> </ul>

### Most important symptoms and effects, both acute and delayed

See Section 11

### 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.

For acute or short term repeated exposures to isopropanol:

- ▶ Rapid onset respiratory depression and hypotension indicates serious ingestions that require careful cardiac and respiratory monitoring together with immediate intravenous access.
- ▶ Rapid absorption precludes the usefulness of emesis or lavage 2 hours post-ingestion. Activated charcoal and cathartics are not clinically useful. Ipecac is most useful when given 30 mins. post-ingestion.
- ▶ There are no antidotes.
- ▶ Management is supportive. Treat hypotension with fluids followed by vasopressors.
- ▶ Watch closely, within the first few hours for respiratory depression; follow arterial blood gases and tidal volumes.
- ▶ Ice water lavage and serial haemoglobin levels are indicated for those patients with evidence of gastrointestinal bleeding.

## SECTION 5 Fire-fighting 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

<b>Fire Incompatibility</b>	None known.
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### Special protective equipment and precautions for fire-fighters

<b>Fire Fighting</b>	<ul style="list-style-type: none"> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ Wear full body protective clothing with breathing apparatus.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ Use water delivered as a fine spray to control fire and cool adjacent area.</li> <li>▶ Avoid spraying water onto liquid pools.</li> <li>▶ <b>DO NOT</b> approach containers suspected to be hot.</li> <li>▶ Cool fire exposed containers with water spray from a protected location.</li> <li>▶ If safe to do so, remove containers from path of fire.</li> </ul>
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<b>Fire/Explosion Hazard</b>	<p><b>WARNING:</b> In use may form flammable/ explosive vapour-air mixtures.</p> <ul style="list-style-type: none"> <li>▶ Combustible.</li> <li>▶ Slight fire hazard when exposed to heat or flame.</li> <li>▶ Heating may cause expansion or decomposition leading to violent rupture of containers.</li> <li>▶ On combustion, may emit toxic fumes of carbon monoxide (CO).</li> <li>▶ May emit acrid smoke.</li> <li>▶ Mists containing combustible materials may be explosive.</li> </ul> <p>Combustion products include: carbon dioxide (CO<sub>2</sub>) other pyrolysis products typical of burning organic material.</p> <p><b>WARNING:</b> Long standing in contact with air and light may result in the formation of potentially explosive peroxides.</p>
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**SECTION 6 Accidental release measures**

**Personal precautions, protective equipment and emergency procedures**

See section 8

**Environmental precautions**

See section 12

**Methods and material for containment and cleaning up**

<b>Minor Spills</b>	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> <li>▶ Clean up all spills immediately.</li> <li>▶ Avoid breathing vapours and contact with skin and eyes.</li> <li>▶ Control personal contact with the substance, by using protective equipment.</li> <li>▶ Contain and absorb spill with sand, earth, inert material or vermiculite.</li> <li>▶ Wipe up.</li> <li>▶ Place in a suitable, labelled container for waste disposal.</li> </ul>																																																																											
<b>Major Spills</b>	<p>Environmental hazard - contain spillage. Chemical Class: alcohols and glycols For release onto land: recommended sorbents listed in order of priority.</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="text-align: left;">SORBENT TYPE</th> <th style="text-align: left;">RANK</th> <th style="text-align: left;">APPLICATION</th> <th style="text-align: left;">COLLECTION</th> <th style="text-align: left;">LIMITATIONS</th> </tr> </thead> <tbody> <tr> <td colspan="5"><b>LAND SPILL - SMALL</b></td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>1</td> <td>shovel</td> <td>shovel</td> <td>R, W, SS</td> </tr> <tr> <td>cross-linked polymer - pillow</td> <td>1</td> <td>throw</td> <td>pitchfork</td> <td>R, DGC, RT</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>2</td> <td>shovel</td> <td>shovel</td> <td>R, I, P</td> </tr> <tr> <td>wood fiber - pillow</td> <td>3</td> <td>throw</td> <td>pitchfork</td> <td>R, P, DGC, RT</td> </tr> <tr> <td>treated wood fiber - pillow</td> <td>3</td> <td>throw</td> <td>pitchfork</td> <td>DGC, RT</td> </tr> <tr> <td>foamed glass - pillow</td> <td>4</td> <td>throw</td> <td>pitchfork</td> <td>R, P, DGC, RT</td> </tr> <tr> <td colspan="5"><b>LAND SPILL - MEDIUM</b></td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>1</td> <td>blower</td> <td>skiploader</td> <td>R, W, SS</td> </tr> <tr> <td>polypropylene - particulate</td> <td>2</td> <td>blower</td> <td>skiploader</td> <td>W, SS, DGC</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>2</td> <td>blower</td> <td>skiploader</td> <td>R, I, W, P, DGC</td> </tr> <tr> <td>polypropylene - mat</td> <td>3</td> <td>throw</td> <td>skiploader</td> <td>DGC, RT</td> </tr> <tr> <td>expanded mineral - particulate</td> <td>3</td> <td>blower</td> <td>skiploader</td> <td>R, I, W, P, DGC</td> </tr> <tr> <td>polyurethane - mat</td> <td>4</td> <td>throw</td> <td>skiploader</td> <td>DGC, RT</td> </tr> </tbody> </table> <p>Legend DGC: Not effective where ground cover is dense R: Not reusable I: Not incinerable P: Effectiveness reduced when rainy RT: Not effective where terrain is rugged SS: Not for use within environmentally sensitive sites W: Effectiveness reduced when windy Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control; R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988</p>	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS	<b>LAND SPILL - SMALL</b>					cross-linked polymer - particulate	1	shovel	shovel	R, W, SS	cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT	sorbent clay - particulate	2	shovel	shovel	R, I, P	wood fiber - pillow	3	throw	pitchfork	R, P, DGC, RT	treated wood fiber - pillow	3	throw	pitchfork	DGC, RT	foamed glass - pillow	4	throw	pitchfork	R, P, DGC, RT	<b>LAND SPILL - MEDIUM</b>					cross-linked polymer - particulate	1	blower	skiploader	R, W, SS	polypropylene - particulate	2	blower	skiploader	W, SS, DGC	sorbent clay - particulate	2	blower	skiploader	R, I, W, P, DGC	polypropylene - mat	3	throw	skiploader	DGC, RT	expanded mineral - particulate	3	blower	skiploader	R, I, W, P, DGC	polyurethane - mat	4	throw	skiploader	DGC, RT
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	<p>Moderate hazard.</p> <ul style="list-style-type: none"> <li>▶ Clear area of personnel and move upwind.</li> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ No smoking, naked lights or ignition sources.</li> <li>▶ Increase ventilation.</li> <li>▶ Stop leak if safe to do so.</li> <li>▶ Contain spill with sand, earth or vermiculite.</li> <li>▶ Collect recoverable product into labelled containers for recycling.</li> <li>▶ Absorb remaining product with sand, earth or vermiculite.</li> <li>▶ Collect solid residues and seal in labelled drums for disposal.</li> <li>▶ Wash area and prevent runoff into drains.</li> <li>▶ If contamination of drains or waterways occurs, advise emergency services.</li> </ul>
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Personal Protective Equipment advice is contained in Section 8 of the SDS.

## SECTION 7 Handling and storage

### Precautions for safe handling

<b>Safe handling</b>	<ul style="list-style-type: none"> <li>▶ Avoid all personal contact, including inhalation.</li> <li>▶ Wear protective clothing when risk of exposure occurs.</li> <li>▶ Use in a well-ventilated area.</li> <li>▶ Prevent concentration in hollows and sumps.</li> <li>▶ <b>DO NOT enter confined spaces until atmosphere has been checked.</b></li> <li>▶ Avoid smoking, naked lights or ignition sources.</li> <li>▶ Avoid contact with incompatible materials.</li> <li>▶ When handling, <b>DO NOT eat, drink or smoke.</b></li> <li>▶ Keep containers securely sealed when not in use.</li> <li>▶ Avoid physical damage to containers.</li> <li>▶ Always wash hands with soap and water after handling.</li> <li>▶ Work clothes should be laundered separately.</li> <li>▶ Use good occupational work practice.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.</li> <li>▶ <b>DO NOT allow clothing wet with material to stay in contact with skin</b></li> </ul>
<b>Other information</b>	<p>Consider storage under inert gas.</p> <ul style="list-style-type: none"> <li>▶ Store in original containers.</li> <li>▶ Keep containers securely sealed.</li> <li>▶ Store in a cool, dry, well-ventilated area.</li> <li>▶ Store away from incompatible materials and foodstuff containers.</li> <li>▶ Protect containers against physical damage and check regularly for leaks.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> </ul>

### Conditions for safe storage, including any incompatibilities

<b>Suitable container</b>	<ul style="list-style-type: none"> <li>▶ Metal can or drum</li> <li>▶ Packaging as recommended by manufacturer.</li> <li>▶ Check all containers are clearly labelled and free from leaks.</li> </ul>
<b>Storage incompatibility</b>	<p>d-Limonene:</p> <ul style="list-style-type: none"> <li>▶ forms unstable peroxides in storage, unless inhibited; may polymerise</li> <li>▶ reacts with strong oxidisers and may explode or combust</li> <li>▶ is incompatible with strong acids, including acidic clays, peroxides, halogens, vinyl chloride and iodine pentafluoride</li> <li>▶ flow or agitation may generate electrostatic charges due to low conductivity</li> </ul> <p>Isopropanol (syn: isopropyl alcohol, IPA):</p> <ul style="list-style-type: none"> <li>▶ forms ketones and unstable peroxides on contact with air or oxygen; the presence of ketones especially methyl ethyl ketone (MEK, 2-butanone) will accelerate the rate of peroxidation</li> <li>▶ reacts violently with strong oxidisers, powdered aluminium (exothermic), crotonaldehyde, diethyl aluminium bromide (ignition), dioxygenyl tetrafluoroborate (ignition/ ambient temperature), chromium trioxide (ignition), potassium-tert-butoxide (ignition), nitroform (possible explosion), oleum (pressure increased in closed container), cobalt chloride, aluminium triisopropoxide, hydrogen plus palladium dust (ignition), oxygen gas, phosgene, phosgene plus iron salts (possible explosion), sodium dichromate plus sulfuric acid (exothermic/ incandescence), triisobutyl aluminium</li> <li>▶ reacts with phosphorus trichloride forming hydrogen chloride gas</li> <li>▶ reacts, possibly violently, with alkaline earth and alkali metals, strong acids, strong caustics, acid anhydrides, halogens, aliphatic amines, aluminium isopropoxide, isocyanates, acetaldehyde, barium perchlorate (forms highly explosive perchloric ester compound), benzoyl peroxide, chromic acid, dialkylzincs, dichlorine oxide, ethylene oxide (possible explosion), hexamethylene diisocyanate (possible explosion), hydrogen peroxide (forms explosive compound), hypochlorous</li> </ul>

acid, isopropyl chlorocarbonate, lithium aluminium hydride, lithium tetrahydroaluminate, nitric acid, nitrogen dioxide, nitrogen tetroxide (possible explosion), pentafluoroguanidine, perchloric acid (especially hot), permonosulfuric acid, phosphorus pentasulfide, tangerine oil, triethylaluminium, triisobutylaluminium, trinitromethane

- attacks some plastics, rubber and coatings
- reacts with metallic aluminium at high temperature
- may generate electrostatic charges
- Avoid oxidising agents, acids, acid chlorides, acid anhydrides, chloroformates.

#### Alcohols

- are incompatible with strong acids, acid chlorides, acid anhydrides, oxidising and reducing agents.
- reacts, possibly violently, with alkaline metals and alkaline earth metals to produce hydrogen
- react with strong acids, strong caustics, aliphatic amines, isocyanates, acetaldehyde, benzoyl peroxide, chromic acid, chromium oxide, dialkylzincs, dichlorine oxide, ethylene oxide, hypochlorous acid, isopropyl chlorocarbonate, lithium tetrahydroaluminate, nitrogen dioxide, pentafluoroguanidine, phosphorus halides, phosphorus pentasulfide, tangerine oil, triethylaluminium, triisobutylaluminium
- should not be heated above 49 deg. C. when in contact with aluminium equipment

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

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

Though autoxidation has been particularly investigated in the field of fatty oils, it also plays a most crucial part for terpenoid deterioration. Although virtually all types of organic materials can undergo air oxidation, certain types are particularly prone to autoxidation, including unsaturated compounds that have allylic or benzylic hydrogen atoms (C<sub>6</sub>H<sub>5</sub>CH<sub>2</sub>-); these materials are converted to hydroperoxides by autoxidation. Promoted by heat, catalytic quantities of redox-reactive metals, and exposure to light, autoxidation may result in the formation of explosive peroxides which may become explosive upon concentration.

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

Compounds rich in allylic hydrogen atoms (2HC=CHCH<sub>2</sub>-R), found in most terpenoids, make up the most probable targets for autoxidation.

Several terpenoids (typically oxygen containing derivatives) are saturated and do not react in a similar fashion to their unsaturated congeners.

Thermolabile terpenoids, especially mere terpenes and aldehydes, are susceptible to rearrangement processes at elevated temperatures. Terpenic conversion reactions, upon heating, have been reported both for isolated compounds as well as for essential oils. (which tend to be rich in mono-, and sesqui-terpenes.

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

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

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

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

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

- The interaction of alkenes and alkynes with nitrogen oxides and oxygen may produce explosive addition products; these may form at very low temperatures and explode on heating to higher temperatures (the addition products from 1,3-butadiene and cyclopentadiene form rapidly at -150 C and ignite or explode on warming to -35 to -15 C). These derivatives ("pseudo- nitrosites") were formerly used to characterise terpene hydrocarbons.

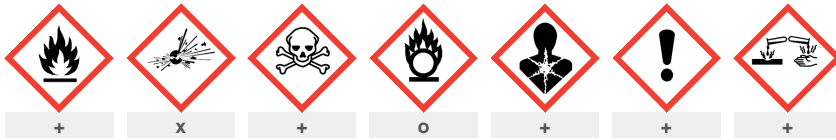
- Exposure to air must be kept to a minimum so as to limit the build-up of peroxides which will concentrate in bottoms if the product is distilled. The product must not be distilled to dryness if the peroxide concentration is substantially above 10 ppm (as active oxygen) since explosive decomposition may occur. Distillate must be immediately inhibited to prevent peroxide formation. The effectiveness of the antioxidant is limited once the peroxide levels exceed 10 ppm as active oxygen. Addition of more inhibitor at this point is generally ineffective. Prior to distillation it is recommended that the product should be washed with aqueous ferrous ammonium sulfate to destroy peroxides; the washed product should be immediately re-inhibited.

- A range of exothermic decomposition energies for double bonds is given as 40-90 kJ/mol. The relationship between energy of decomposition and processing hazards has been the subject of discussion; it is suggested that values of energy released per unit of mass, rather than on a molar basis (J/g) be used in the assessment. For example, in "open vessel processes" (with man-hole size openings, in an industrial setting), substances with exothermic decomposition energies below 500 J/g are unlikely to present a danger, whilst those in "closed vessel processes" (opening is a safety valve or bursting disk) present some danger where the decomposition energy exceeds 150 J/g.

BRETHERRICK: Handbook of Reactive Chemical Hazards, 4th Edition

- The reaction of ozone with alkenes is believed to proceed *via* the formation of a vibrationally excited Primary Ozonide (POZ) which falls apart to give a vibrationally excited Criegee Intermediate (CI). The CI can decompose to give OH radicals, or be stabilised. This may be of relevance in atmospheric chemistry.

- Violent explosions at low temperatures in ammonia synthesis gas units have been traced to the addition products of dienes and nitrogen dioxide



- X — Must not be stored together  
O — May be stored together with specific preventions  
+ — May be stored together

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

## SECTION 8 Exposure controls / personal protection

### Control parameters

#### Occupational Exposure Limits (OEL)

#### INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US OSHA Permissible Exposure Limits (PELs) Table Z-1	ethanol, denatured	Ethyl alcohol (Ethanol)	1000 ppm / 1900 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	ethanol, denatured	Ethyl alcohol	1000 ppm / 1900 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	isopropanol	Isopropyl alcohol	400 ppm / 980 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	isopropanol	Isopropyl alcohol	400 ppm / 980 mg/m <sup>3</sup>	1225 mg/m <sup>3</sup> / 500 ppm	Not Available	Not Available

#### Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
ethanol, denatured	Not Available	Not Available	15000* ppm
isopropanol	400 ppm	2000* ppm	12000** ppm
d-limonene	15 ppm	67 ppm	170 ppm

Ingredient	Original IDLH	Revised IDLH
water	Not Available	Not Available
ethanol, denatured	3,300 ppm	Not Available
isopropanol	2,000 ppm	Not Available
d-limonene	Not Available	Not Available

#### Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
d-limonene	E	≤ 0.1 ppm

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

#### MATERIAL DATA

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

For ethanol:

Odour Threshold Value: 49-716 ppm (detection), 101 ppm (recognition)

Eye and respiratory tract irritation do not appear to occur at exposure levels of less than 5000 ppm and the TLV-TWA is thought to provide an adequate margin of safety against such effects. Experiments in man show that inhalation of 1000 ppm caused slight symptoms of poisoning and 5000 ppm caused strong stupor and morbid sleepiness. Subjects exposed to 5000 ppm to 10000 ppm experienced smarting of the eyes and nose and coughing. Symptoms disappeared within minutes. Inhalation also causes local irritating effects to the eyes and upper respiratory tract, headaches, sensation of heat intraocular tension, stupor, fatigue and a need to sleep. At 15000 ppm there was continuous lachrymation and coughing.

Exposed individuals are **NOT** reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

ClassOSF Description

- A 550 Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities
- B 26-550As "A" for 50-90% of persons being distracted
- C 1-26 As "A" for less than 50% of persons being distracted
- D 0.18-1 10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached
- E <0.18 As "D" for less than 10% of persons aware of being tested

for d-Limonene:

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

(CEL = Chemwatch Exposure Limit)

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


Odour Threshold Value: 3.3 ppm (detection), 7.6 ppm (recognition)

Exposure at or below the recommended isopropanol TLV-TWA and STEL is thought to minimise the potential for inducing narcotic effects or significant irritation of the eyes or upper respiratory tract. It is believed, in the absence of hard evidence, that this limit also provides protection against the development of chronic health effects. The limit is intermediate to that set for ethanol, which is less toxic, and n-propyl alcohol, which is more toxic, than isopropanol

**Exposure controls**

<b>Appropriate engineering controls</b>	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>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.</p> <p>An approved self contained breathing apparatus (SCBA) may be required in some situations.</p> <p>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.</p>											
	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.)										
	<p>Within each range the appropriate value depends on:</p> <table border="1" style="width: 100%;"> <tr> <td style="width: 50%;">Lower end of the range</td> <td style="width: 50%;">Upper end of the range</td> </tr> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </table>		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
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<p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the</p>												



	<p>extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>
<b>Individual protection measures, such as personal protective equipment</b>	
<b>Eye and face protection</b>	<ul style="list-style-type: none"> <li>▶ Safety glasses with side shields.</li> <li>▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]</li> <li>▶ 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].</li> </ul>
<b>Skin protection</b>	See Hand protection below
<b>Hands/feet protection</b>	<ul style="list-style-type: none"> <li>▶ Wear chemical protective gloves, e.g. PVC.</li> <li>▶ Wear safety footwear or safety gumboots, e.g. Rubber</li> </ul> <p>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.</p> <p>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.</p> <p>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.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> <li>· frequency and duration of contact,</li> <li>· chemical resistance of glove material,</li> <li>· glove thickness and</li> <li>· dexterity</li> </ul> <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> <li>· 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.</li> <li>· 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.</li> <li>· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.</li> <li>· Contaminated gloves should be replaced.</li> </ul> <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> <li>· Excellent when breakthrough time &gt; 480 min</li> <li>· Good when breakthrough time &gt; 20 min</li> <li>· Fair when breakthrough time &lt; 20 min</li> <li>· Poor when glove material degrades</li> </ul> <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>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.</p> <p>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.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> <li>· 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.</li> <li>· 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</li> </ul> <p>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.</p>
<b>Body protection</b>	See Other protection below
<b>Other protection</b>	<ul style="list-style-type: none"> <li>▶ Overalls.</li> <li>▶ P.V.C apron.</li> <li>▶ Barrier cream.</li> <li>▶ Skin cleansing cream.</li> <li>▶ Eye wash unit.</li> </ul>

**Respiratory protection**

Type A 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 10 x ES	Air-line*	A-2	A-PAPR-2 ^
up to 20 x ES	-	A-3	-
20+ x ES	-	Air-line**	-

\* - Continuous-flow; \*\* - Continuous-flow or positive pressure demand

^ - Full-face

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(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

## SECTION 9 Physical and chemical properties

### Information on basic physical and chemical properties

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

## SECTION 10 Stability and reactivity

<b>Reactivity</b>	See section 7
<b>Chemical stability</b>	<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>

<b>Possibility of hazardous reactions</b>	See section 7
<b>Conditions to avoid</b>	See section 7
<b>Incompatible materials</b>	See section 7
<b>Hazardous decomposition products</b>	See section 5

## SECTION 11 Toxicological information

### Information on toxicological effects

<b>Inhaled</b>	<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> <p>The most common signs of inhalation overexposure to ethanol, in animals, include ataxia, incoordination and drowsiness for those surviving narcosis. The narcotic dose for rats, after 2 hours of exposure, is 19260 ppm.</p> <p>Not normally a hazard due to non-volatile nature of product</p> <p>Exposure to aliphatic alcohols with more than 3 carbons may produce central nervous system effects such as headache, dizziness, drowsiness, muscle weakness, delirium, CNS depression, coma, seizure, and neurobehavioural changes. Symptoms are more acute with higher alcohols. Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, respiratory depression secondary to CNS depression, pulmonary oedema, chemical pneumonitis and bronchitis. Cardiovascular involvement may result in arrhythmias and hypotension. Gastrointestinal effects may include nausea and vomiting. Kidney and liver damage may result following massive exposures. The alcohols are potential irritants being, generally, stronger irritants than similar organic structures that lack functional groups (e.g. alkanes) but are much less irritating than the corresponding amines, aldehydes or ketones. Alcohols and glycols (diols) rarely represent serious hazards in the workplace, because their vapour concentrations are usually less than the levels which produce significant irritation which, in turn, produce significant central nervous system effects as well.</p> <p>The material has <b>NOT</b> been classified by EC Directives or other classification systems as "harmful by inhalation". This is because of the lack of corroborating animal or human evidence. In the absence of such evidence, care should be taken nevertheless to ensure exposure is kept to a minimum and that suitable control measures be used, in an occupational setting to control vapours, fumes and aerosols.</p>				
<b>Ingestion</b>	<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>Effects on the nervous system characterise over-exposure to higher aliphatic alcohols. These include headache, muscle weakness, giddiness, ataxia, (loss of muscle coordination), confusion, delirium and coma. Gastrointestinal effects may include nausea, vomiting and diarrhoea. In the absence of effective treatment, respiratory arrest is the most common cause of death in animals acutely poisoned by the higher alcohols. Aspiration of liquid alcohols produces an especially toxic response as they are able to penetrate deeply in the lung where they are absorbed and may produce pulmonary injury. Those possessing lower viscosity elicit a greater response. The result is a high blood level and prompt death at doses otherwise tolerated by ingestion without aspiration. In general the secondary alcohols are less toxic than the corresponding primary isomers. As a general observation, alcohols are more powerful central nervous system depressants than their aliphatic analogues. In sequence of decreasing depressant potential, tertiary alcohols with multiple substituent OH groups are more potent than secondary alcohols, which, in turn, are more potent than primary alcohols. The potential for overall systemic toxicity increases with molecular weight (up to C7), principally because the water solubility is diminished and lipophilicity is increased.</p> <p>Within the homologous series of aliphatic alcohols, narcotic potency may increase even faster than lethality</p> <p>Only scanty toxicity information is available about higher homologues of the aliphatic alcohol series (greater than C7) but animal data establish that lethality does not continue to increase with increasing chain length. Aliphatic alcohols with 8 carbons are less toxic than those immediately preceding them in the series. 10 -Carbon n-decyl alcohol has low toxicity as do the solid fatty alcohols (e.g. lauryl, myristyl, cetyl and stearyl). However the rat aspiration test suggests that decyl and melted dodecyl (lauryl) alcohols are dangerous if they enter the trachea. In the rat even a small quantity (0.2 ml) of these behaves like a hydrocarbon solvent in causing death from pulmonary oedema.</p> <p>Primary alcohols are metabolised to corresponding aldehydes and acids; a significant metabolic acidosis may occur. Secondary alcohols are converted to ketones, which are also central nervous system depressants and which, in the case of the higher homologues persist in the blood for many hours. Tertiary alcohols are metabolised slowly and incompletely so their toxic effects are generally persistent.</p> <p>Ingestion of ethanol (ethyl alcohol, "alcohol") may produce nausea, vomiting, bleeding from the digestive tract, abdominal pain, and diarrhoea. Effects on the body:</p> <table border="1" style="margin-left: auto; margin-right: auto;"> <thead> <tr> <th>Blood concentration</th> <th>Effects</th> </tr> </thead> <tbody> <tr> <td>&lt;1.5 g/L</td> <td>Mild: impaired vision, co-ordination and reaction time; emotional instability</td> </tr> </tbody> </table>	Blood concentration	Effects	<1.5 g/L	Mild: impaired vision, co-ordination and reaction time; emotional instability
Blood concentration	Effects				
<1.5 g/L	Mild: impaired vision, co-ordination and reaction time; emotional instability				

	<p>1.5-3.0 g/L</p>	<p>Moderate: Slurred speech, confusion, inco-ordination, emotional instability, disturbances in perception and senses, possible blackouts, and impaired objective performance in standardized tests. Possible double vision, flushing, fast heart rate, sweating and incontinence. Slow breathing may occur rarely and fast breathing may develop in cases of metabolic acidosis, low blood sugar and low blood potassium. Central nervous system depression may progress to coma.</p>
	<p>3-5 g/L</p>	<p>Severe: cold clammy skin, low body temperature and low blood pressure. Atrial fibrillation and heart block have been reported. Depression of breathing may occur, respiratory failure may follow serious poisoning, choking on vomit may result in lung inflammation and swelling. Convulsions due to severe low blood sugar may also occur. Acute liver inflammation may develop.</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> <p>Swallowing 10 millilitres of isopropanol may cause serious injury; 100 millilitres may be fatal if not properly treated. The adult single lethal dose is approximately 250 millilitres. Isopropanol is twice as poisonous as ethanol, and the effects caused are similar, except that isopropanol does not cause an initial feeling of well-being. Swallowing may cause nausea, vomiting and diarrhea; vomiting and stomach inflammation is more prominent with isopropanol than with ethanol. Animals given near-lethal doses also showed inco-ordination, lethargy, inactivity and loss of consciousness.</p> <p>There is evidence that a slight tolerance to isopropanol may be acquired.</p>		
<p><b>Skin Contact</b></p>	<p>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.</p> <p>Limited evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man.</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> <p>511ipa</p>	
<p><b>Eye</b></p>	<p>Although the liquid is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).</p> <p>Direct contact of the eye with ethanol may cause immediate stinging and burning with reflex closure of the lid and tearing, transient injury of the corneal epithelium and hyperaemia of the conjunctiva. Foreign-body type discomfort may persist for up to 2 days but healing is usually spontaneous and complete.</p> <p>Isopropanol vapour may cause mild eye irritation at 400 ppm. Splashes may cause severe eye irritation, possible corneal burns and eye damage. Eye contact may cause tearing or blurring of vision.</p>	
<p><b>Chronic</b></p>	<p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p>	

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

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

Long-term exposure to ethanol may result in progressive liver damage with fibrosis or may exacerbate liver injury caused by other agents.

Repeated ingestion of ethanol by pregnant women may adversely affect the central nervous system of the developing foetus, producing effects collectively described as foetal alcohol syndrome. These include mental and physical retardation, learning disturbances, motor and language deficiency, behavioural disorders and reduced head size.

Consumption of ethanol (in alcoholic beverages) may be linked to the development of Type I hypersensitivities in a small number of individuals. Symptoms, which may appear immediately after consumption, include conjunctivitis, angioedema, dyspnoea, and urticarial rashes. The causative agent may be acetic acid, a metabolite (1).

(1) Boehncke W.H., & H.Gall, Clinical & Experimental Allergy, 26, 1089-1091, 1996

A number of common flavor and fragrance chemicals can form peroxides surprisingly fast in air. Antioxidants can in most cases minimize the oxidation.

Fragrance terpenes are easily oxidized in air. Non-oxidised forms are very weak sensitizers; however, after oxidation, the hydroperoxides are strong sensitizers which may cause allergic reactions. Autooxidation of fragrance terpenes contributes greatly to fragrance allergy. There is the need to test for compounds the patients are actually exposed to, not only the ingredients originally applied in commercial formulations.

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

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

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

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

Long term, or repeated exposure of isopropanol may cause inco-ordination and tiredness.

Repeated inhalation exposure to isopropanol may produce sleepiness, inco-ordination and liver degeneration. Animal data show developmental effects only at exposure levels that produce toxic effects in adult animals. Isopropanol does not cause genetic damage.

There are inconclusive reports of human sensitisation from skin contacts with isopropanol. Chronic alcoholics are more tolerant of the whole-body effects of isopropanol.

Animal testing showed the chronic exposure did not produce reproductive effects.

NOTE: Commercial isopropanol does not contain "isopropyl oil", which caused an excess incidence of sinus and throat cancers in isopropanol production workers in the past. "Isopropyl oil" is no longer formed during production of isopropanol.

Ingestion may result in intoxication, drunkenness. In chronic form this may result in alcoholism, liver damage.

<b>N3 Prep Spray</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
<b>water</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral (Rat) LD50: >90000 mg/kg <sup>[2]</sup>	Not Available

<b>ethanol, denatured</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 17100 mg/kg <sup>[1]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
	Inhalation(Rat) LC50: 64000 ppm4h <sup>[2]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (Rat) LD50: 7060 mg/kg <sup>[2]</sup>	
<b>isopropanol</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 12800 mg/kg <sup>[2]</sup>	Eye (rabbit): 10 mg - moderate
	Inhalation(Mouse) LC50; 53 mg/L4h <sup>[2]</sup>	Eye (rabbit): 100 mg - SEVERE
	Oral (Mouse) LD50; 3600 mg/kg <sup>[2]</sup>	Eye (rabbit): 100mg/24hr-moderate
		Skin (rabbit): 500 mg - mild
<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>Legend:</b>	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	

<b>WATER</b>	No significant acute toxicological data identified in literature search.
<b>ETHANOL, DENATURED</b>	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>
<b>ISOPROPANOL</b>	<p>For isopropanol (IPA):</p> <p><b>Acute toxicity:</b> Isopropanol has a low order of acute toxicity. It is irritating to the eyes, but not to the skin. Very high vapor concentrations are irritating to the eyes, nose, and throat, and prolonged exposure may produce central nervous system depression and narcosis. Human volunteers reported that exposure to 400 ppm isopropanol vapors for 3 to 5 min. caused mild irritation of the eyes, nose and throat.</p> <p>Although isopropanol produced little irritation when tested on the skin of human volunteers, there have been reports of isolated cases of dermal irritation and/or sensitization. The use of isopropanol as a sponge treatment for the control of fever has resulted in cases of intoxication, probably the result of both dermal absorption and inhalation. There have been a number of cases of poisoning reported due to the intentional ingestion of isopropanol, particularly among alcoholics or suicide victims. These ingestions typically result in a comatose condition. Pulmonary difficulty, nausea, vomiting, and headache accompanied by various degrees of central nervous system depression are typical. In the absence of shock, recovery usually occurred.</p> <p><b>Repeat dose studies:</b> The systemic (non-cancer) toxicity of repeated exposure to isopropanol has been evaluated in rats and mice by the inhalation and oral routes. The only adverse effects-in addition to clinical signs identified from these studies were to the kidney.</p> <p><b>Reproductive toxicity:</b> A recent two-generation reproductive study characterised the reproductive hazard for isopropanol associated with oral gavage exposure. This study found that the only reproductive parameter apparently affected by isopropanol exposure was a statistically significant decrease in male mating index of the F1 males. It is possible that the change in this reproductive parameter was treatment related and significant, although the mechanism of this effect could not be discerned from the results of the study. However, the lack of a significant effect of the female mating index in either generation, the absence of any adverse effect on litter size, and the lack of histopathological findings of the testes of the high-dose males suggest that the observed reduction in male mating index may not be biologically meaningful.</p> <p><b>Developmental toxicity:</b> The developmental toxicity of isopropanol has been characterized in rat and rabbit developmental toxicity studies. These studies indicate that isopropanol is not a selective developmental hazard. Isopropanol produced developmental toxicity in rats, but not in rabbits. In the rat, the developmental toxicity occurred only at maternally toxic doses and consisted of decreased foetal body weights, but no teratogenicity</p> <p><b>Genotoxicity:</b> All genotoxicity assays reported for isopropanol have been negative</p> <p><b>Carcinogenicity:</b> rodent inhalation studies were conducted to evaluate isopropanol for cancer potential. The only tumor rate increase seen was for interstitial (Leydig) cell tumors in the male rats. Interstitial cell tumors of the testis is typically the most frequently observed spontaneous tumor in aged male Fischer 344 rats. These studies demonstrate that isopropanol does not exhibit carcinogenic potential relevant to humans. Furthermore, there was no evidence from this study to indicate the development of carcinomas of the testes in the male rat, nor has isopropanol been found to be genotoxic. Thus, the testicular tumors seen in the isopropanol exposed male rats are considered of no significance in terms of human cancer risk assessment. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>
<b>D-LIMONENE</b>	<p>Tumorigenic by RTECS criteria</p> <p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The</p>

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 fewer 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.

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

Acute toxicity:

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

Oral:

Acute oral LD<sub>50</sub> values for MMTC, MMT(EHTG), MMT(IOTG), and TERP indicated low to moderate toxicity; the most reliable data place the LD<sub>50</sub>s in the range of 1000 mg/kg.

The acute oral LD<sub>50</sub> 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 LD<sub>50</sub> values were =1000 mg/kg bw, and inhalation LC<sub>50</sub> was >200 mg/L. MMTC was corrosive to skin and assumed corrosive to eyes.

The acute dermal LD<sub>50</sub> 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 LC<sub>50</sub> of MMT(2-EHMA) was 240 mg/L.

The study reported an acute inhalation LC<sub>50</sub> 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 [ $<1-3.6$  mg/kg bw/d]) and additional effects including increased relative thymus weights in females and urinalysis results in both sexes at higher doses.

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

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

#### Neurotoxicity:

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

#### Immunotoxicity:

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

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

#### Genotoxicity:

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

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

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

#### Carcinogenicity:

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

#### Toxicity to reproduction:

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

#### SIDS 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)

### **N3 Prep Spray & ISOPROPANOL**

Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.

### **N3 Prep Spray & D-LIMONENE**

Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and connubial contact dermatitis occur.

Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to



ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes.

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

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

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

Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a 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.

#### **Prehaptens**

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

Autoxidation is a free radical chain reaction in which hydrogen atom abstraction in combination with addition of oxygen forms 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.

#### **Prohaptens**

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

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

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

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

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

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

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

#### **ISOPROPANOL & D-LIMONENE**

The substance is classified by IARC as Group 3:  
**NOT** classifiable as to its carcinogenicity to humans.  
Evidence of carcinogenicity may be inadequate or limited in animal testing.

**Acute Toxicity**

**×**

**Carcinogenicity**

**×**

**Continued...**

**N3 Prep Spray**

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

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

**SECTION 12 Ecological information**

**Toxicity**

	Endpoint	Test Duration (hr)	Species	Value	Source
<b>N3 Prep Spray</b>	Not Available	Not Available	Not Available	Not Available	Not Available
<b>water</b>	Not Available	Not Available	Not Available	Not Available	Not Available
<b>ethanol, denatured</b>	EC50	72h	Algae or other aquatic plants	275mg/l	2
	EC50	48h	Crustacea	2mg/l	4
	EC50	96h	Algae or other aquatic plants	<0.001mg/L	4
	LC50	96h	Fish	42mg/l	4
	EC50(ECx)	96h	Algae or other aquatic plants	<0.001mg/L	4
<b>isopropanol</b>	EC50	72h	Algae or other aquatic plants	>1000mg/l	1
	EC50	48h	Crustacea	7550mg/l	4
	EC50	96h	Algae or other aquatic plants	>1000mg/l	1
	LC50	96h	Fish	>1400mg/l	4
	EC50(ECx)	24h	Algae or other aquatic plants	0.011mg/L	4
<b>d-limonene</b>	EC50	72h	Algae or other aquatic plants	0.214mg/l	2
	EC50	48h	Crustacea	0.307mg/l	2
	LC50	96h	Fish	0.46mg/l	2
	NOEC(ECx)	0h	Algae or other aquatic plants	<0.05-1.5mg/l	4
<b>Legend:</b>	<i>Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data</i>				

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 Ethanol:

log Kow: -0.31 to -0.32;

Koc 1: Estimated BCF= 3;

Half-life (hr) air: 144;

Half-life (hr) H2O surface water: 144;

Henry's atm m3 /mol: 6.29E-06;

BOD 5 if unstated: 0.93-1.67,63%

COD: 1.99-2.11,97%;

ThOD : 2.1.

Environmental Fate: Terrestrial - Ethanol quickly biodegrades in soil but may leach into ground water; most is lost by evaporation. Ethanol is expected to have very high mobility in soil. Volatilization of ethanol from moist soil surfaces is expected to be an important fate process. The potential for volatilization of ethanol from dry soil surfaces may exist. Biodegradation is expected to be an important fate process for ethanol based on half-lives on the order of a few days for ethanol in sandy soil/groundwater microcosms.

**Atmospheric Fate:** Ethanol is expected to exist solely as a vapour in the ambient atmosphere. Vapour-phase ethanol is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 5 days. Ethanol readily degraded by reaction with photochemically produced hydroxy radicals; release into air will result in photodegradation and wet deposition.

**Aquatic Fate:** When released into water ethanol readily evaporates and is biodegradable. Ethanol is not expected to adsorb to suspended solids and sediment. Volatilization from water surfaces is expected and volatilization half-lives for a model river and model lake are 3 and 39 days, respectively. Bioconcentration in aquatic organisms is considered to be low. Hydrolysis and photolysis in sunlit surface waters is not expected to be an important environmental fate process for ethanol and is unlikely to be persistent in aquatic environments.

For isopropanol (IPA):

log Kow : -0.16- 0.28

Half-life (hr) air : 33-84

Half-life (hr) H2O surface water : 130

Henry's atm m3 /mol: 8.07E-06

BOD 5: 1.19,60%

COD : 1.61-2.30,97%

ThOD : 2.4

BOD 20: >70% \* [Akzo Nobel]

#### **Environmental Fate**

Based on calculated results from a level 1 fugacity model, IPA is expected to partition primarily to the aquatic compartment (77.7%) with the remainder to the air (22.3%). IPA has been shown to biodegrade rapidly in aerobic, aqueous biodegradation tests and therefore, would not be expected to persist in aquatic habitats. IPA is also not expected to persist in surface soils due to rapid evaporation to the air. In the air, physical degradation will occur rapidly due to hydroxy radical (OH) attack. Overall, IPA presents a low potential hazard to aquatic or terrestrial biota.

IPA is expected to volatilise slowly from water based on a calculated Henry's Law constant of  $7.52 \times 10^{-6}$  atm.m<sup>3</sup>/mole. The calculated half-life for the volatilisation from surface water (1 meter depth) is predicted to range from 4 days (from a river) to 31 days (from a lake). Hydrolysis is not considered a significant degradation process for IPA. However, aerobic biodegradation of IPA has been shown to occur rapidly under non-acclimated conditions, based on a result of 49% biodegradation from a 5 day BOD test. Additional biodegradation data developed using standardized test methods show that IPA is readily biodegradable in both freshwater and saltwater media (72 to 78% biodegradation in 20 days).

IPA will evaporate quickly from soil due to its high vapor pressure (43 hPa at 20°C), and is not expected to partition to the soil based on a calculated soil adsorption coefficient (log Koc) of 0.03.

IPA has the potential to leach through the soil due to its low soil adsorption

In the air, isopropanol is subject to oxidation predominantly by hydroxy radical attack. The room temperature rate constants determined by several investigators are in good agreement for the reaction of IPA with hydroxy radicals. The atmospheric half-life is expected to be 10 to 25 hours, based on measured degradation rates ranging from  $5.1$  to  $7.1 \times 10^{-12}$  cm<sup>3</sup>/molecule-sec, and an OH concentration of  $1.5 \times 10^6$  molecule/cm<sup>3</sup>, which is a commonly used default value for calculating atmospheric half-lives. Using OH concentrations representative of polluted ( $3 \times 10^6$ ) and pristine ( $3 \times 10^5$ ) air, the atmospheric half-life of IPA would range from 9 to 126 hours, respectively. Direct photolysis is not expected to be an important transformation process for the degradation of IPA.

#### **Ecotoxicity:**

IPA has been shown to have a low order of acute aquatic toxicity. Results from 24- to 96-hour LC50 studies range from 1,400 to more than 10,000 mg/L for freshwater and saltwater fish and invertebrates. In addition, 16-hour to 8-day toxicity threshold levels (equivalent to 3% inhibition in cell growth) ranging from 104 to 4,930 mg/L have been demonstrated for various microorganisms.

Chronic aquatic toxicity has also been shown to be of low concern, based on 16- to 21-day NOEC values of 141 to 30 mg/L, respectively, for a freshwater invertebrate. Bioconcentration of IPA in aquatic organisms is not expected to occur based on a measured log octanol/water partition coefficient (log Kow) of 0.05, a calculated bioconcentration factor of 1 for a freshwater fish, and the unlikelihood of constant, long-term exposures.

#### **Toxicity to Plants**

Toxicity to plants is expected to be low, based on a 7-day toxicity threshold value of 1,800 mg/L for a freshwater algae, and an EC50 value of 2,100 mg/L from a lettuce seed germination test.

For Terpenes such as Limonene and Isoprene:

**Atmospheric Fate:** Contribute to aerosol and photochemical smog formation. When terpenes are introduced to the atmosphere, may either decrease ozone concentrations when oxides of nitrogen are low or, if emissions take place in polluted air (i.e. containing high concentrations of nitrogen oxides), leads to an increase in ozone concentrations. Lower terpenoids can react with unstable reactive gases and may act as precursors of photochemical smog therefore indirectly influencing community and ecosystem properties. The reactions of ozone with larger unsaturated compounds, such as the terpenes can give rise to oxygenated species with low vapour pressures that subsequently condense to form secondary organic aerosol.

**Aquatic Fate:** Complex chlorinated terpenes such as toxaphene (a persistent, mobile and toxic insecticide) and its degradation products were produced by photoinitiated reactions in an aqueous system, initially containing limonene and other monoterpenes, simulating pulp bleach conditions.

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, 6MHO, 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-ol, 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

For Limonenes:

**Atmospheric Fate:** Due to the high volatility of limonene, the atmosphere is expected to be the major environmental sink for this chemical. The oxidation of limonene may contribute to aerosol and photochemical smog formation. The daytime atmospheric lifetime of d-limonene is estimated to range from 12 to 48 minutes depending upon local hydroxyl rate and ozone concentrations. 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. 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. It is expected that limonene will rapidly volatilize 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 is 3.4 h. Some limonene is expected to absorb to sediment and suspended organic matter. 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.

**Ecotoxicity:** Biotic degradation of limonene has been shown with some species of microorganisms such as *Penicillium digitatum*, *Corynespora cassiicola*, *Diplodia gossypina* and a soil strain of *Pseudomonas* sp (SL strain). Limonene is readily biodegradable under aerobic conditions. Biodegradation has been assessed under anaerobic conditions; there was no indication of any metabolisms, possibly because of the toxicity to micro-organisms. Limonene may bioaccumulate in fish and other aquatic species. Technical limonene is practically nontoxic to birds and is slightly toxic to freshwater fish and invertebrates on an acute basis. Limonene has low subacute toxicity to bobwhite quail.

**DO NOT discharge into sewer or waterways.**

## Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
water	LOW	LOW
ethanol, denatured	LOW (Half-life = 2.17 days)	LOW (Half-life = 5.08 days)
isopropanol	LOW (Half-life = 14 days)	LOW (Half-life = 3 days)
d-limonene	HIGH	HIGH

## Bioaccumulative potential

Ingredient	Bioaccumulation
ethanol, denatured	LOW (LogKOW = -0.31)
isopropanol	LOW (LogKOW = 0.05)
d-limonene	HIGH (LogKOW = 4.8275)

## Mobility in soil

Ingredient	Mobility
ethanol, denatured	HIGH (KOC = 1)
isopropanol	HIGH (KOC = 1.06)
d-limonene	LOW (KOC = 1324)

## SECTION 13 Disposal considerations

### Waste treatment methods


Product / Packaging disposal	Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws

Continued...

	<p>operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> <li>▸ Reduction</li> <li>▸ Reuse</li> <li>▸ Recycling</li> <li>▸ Disposal (if all else fails)</li> </ul> <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> <li>▸ <b>DO NOT allow wash water from cleaning or process equipment to enter drains.</b></li> <li>▸ It may be necessary to collect all wash water for treatment before disposal.</li> <li>▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.</li> <li>▸ Where in doubt contact the responsible authority.</li> <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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## SECTION 14 Transport information

### Labels Required

	
<b>Marine Pollutant</b>	NO

Shipping container and transport vehicle placarding and labeling may vary from the below information. Products that are regulated for transport will be packaged and marked as Dangerous Goods in Limited Quantities according to US DOT, IATA and IMDG regulations. In case of reshipment, it is the responsibility of the shipper to determine the appropriate labels and markings in accordance with applicable transport regulations.

### Land transport (DOT)

14.1. <b>UN number or ID number</b>	3082	
14.2. <b>UN proper shipping name</b>	Environmentally hazardous substance, liquid, n.o.s.	
14.3. <b>Transport hazard class(es)</b>	Class	9
	Subsidiary risk	Not Applicable
14.4. <b>Packing group</b>	III	
14.5. <b>Environmental hazard</b>	Not Applicable	
14.6. <b>Special precautions for user</b>	Hazard Label	9
	Special provisions	8, 146, 173, 335, 441, IB3, T4, TP1, TP29

For Individual Packages of Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082 that contain LESS THAN the reportable quantity (5 kg or 5 L) - Not Regulated

For Individual Packages of Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082 that contain MORE THAN the reportable quantity (5 kg or 5 L) - Regulated and classified as below:

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

14.1. <b>UN number</b>	3082	
14.2. <b>UN proper shipping name</b>	Environmentally hazardous substance, liquid, n.o.s.	
14.3. <b>Transport hazard class(es)</b>	ICAO/IATA Class	9
	ICAO / IATA Subsidiary Hazard	Not Applicable
	ERG Code	9L
14.4. <b>Packing group</b>	III	

<b>14.5. Environmental hazard</b>	Not Applicable	
<b>14.6. Special precautions for user</b>	Special provisions	A97 A158 A197 A215
	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)**

<b>14.1. UN number</b>	3082	
<b>14.2. UN proper shipping name</b>	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S.	
<b>14.3. Transport hazard class(es)</b>	IMDG Class	9
	IMDG Subrisk	Not Applicable
<b>14.4. Packing group</b>	III	
<b>14.5. Environmental hazard</b>	Not Applicable	
<b>14.6. Special precautions for user</b>	EMS Number	F-A, S-F
	Special provisions	274 335 969
	Limited Quantities	5 L

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

Not Applicable

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

Product name	Group
water	Not Available
ethanol, denatured	Not Available
isopropanol	Not Available
d-limonene	Not Available

**14.7.3. Transport in bulk in accordance with the IGC Code**

Product name	Ship Type
water	Not Available
ethanol, denatured	Not Available
isopropanol	Not Available
d-limonene	Not Available

**SECTION 15 Regulatory information**

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

**water is found on the following regulatory lists**

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

**ethanol, denatured is found on the following regulatory lists**

US - Massachusetts - Right To Know Listed Chemicals

US DOE Temporary Emergency Exposure Limits (TEELs)

US NIOSH Recommended Exposure Limits (RELs)

US OSHA Permissible Exposure Limits (PELs) Table Z-1

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

**isopropanol is found on the following regulatory lists**

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic  
 US - Massachusetts - Right To Know Listed Chemicals  
 US DOE Temporary Emergency Exposure Limits (TEELs)  
 US EPCRA Section 313 Chemical List

US NIOSH Recommended Exposure Limits (RELs)  
 US OSHA Permissible Exposure Limits (PELs) Table Z-1  
 US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory  
 US TSCA Section 4/12 (b) - Sunset Dates/Status

#### **d-limonene is found on the following regulatory lists**

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic  
 US AIHA Workplace Environmental Exposure Levels (WEELs)  
 US DOE Temporary Emergency Exposure Limits (TEELs)

US EPA Integrated Risk Information System (IRIS)  
 US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory  
 US Toxicology Excellence for Risk Assessment (TERA) Workplace Environmental Exposure Levels (WEEL)

## **Federal Regulations**

### **Superfund Amendments and Reauthorization Act of 1986 (SARA)**

#### **Section 311/312 hazard categories**

Flammable (Gases, Aerosols, Liquids, or Solids)	No
Gas under pressure	No
Explosive	No
Self-heating	No
Pyrophoric (Liquid or Solid)	No
Pyrophoric Gas	No
Corrosive to metal	No
Oxidizer (Liquid, Solid or Gas)	No
Organic Peroxide	No
Self-reactive	No
In contact with water emits flammable gas	No
Combustible Dust	No
Carcinogenicity	No
Acute toxicity (any route of exposure)	No
Reproductive toxicity	No
Skin Corrosion or Irritation	No
Respiratory or Skin Sensitization	Yes
Serious eye damage or eye irritation	Yes
Specific target organ toxicity (single or repeated exposure)	Yes
Aspiration Hazard	No
Germ cell mutagenicity	No
Simple Asphyxiant	No
Hazards Not Otherwise Classified	No

#### **US. EPA CERCLA Hazardous Substances and Reportable Quantities (40 CFR 302.4)**

None Reported

## **State Regulations**

#### **US. California Proposition 65**

None Reported

## **National Inventory Status**

<b>National Inventory</b>	<b>Status</b>
Australia - AIIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (water; ethanol, denatured; isopropanol; d-limonene)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes



National Inventory	Status
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 - FBEPH	Yes
<b>Legend:</b>	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

**SECTION 16 Other information**

<b>Revision Date</b>	25/09/2023
<b>Initial Date</b>	26/09/2023

**Other information****Ingredients with multiple cas numbers**

Name	CAS No
d-limonene	5989-27-5, 138-86-3

Classification of the preparation and its individual components has drawn on official and authoritative sources using available literature references.

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

**Definitions and abbreviations**

PC - TWA: Permissible Concentration-Time Weighted Average

PC - STEL: Permissible Concentration-Short Term Exposure Limit

IARC: International Agency for Research on Cancer

ACGIH: American Conference of Governmental Industrial Hygienists

STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit,

IDLH: Immediately Dangerous to Life or Health Concentrations

ES: Exposure Standard

OSF: Odour Safety Factor

NOAEL :No Observed Adverse Effect Level

LOAEL: Lowest Observed Adverse Effect Level

TLV: Threshold Limit Value

LOD: Limit Of Detection

OTV: Odour Threshold Value

BCF: BioConcentration Factors

BEI: Biological Exposure Index

AIIC: Australian Inventory of Industrial Chemicals

DSL: Domestic Substances List

NDSL: Non-Domestic Substances List

IECSC: Inventory of Existing Chemical Substance in China

EINECS: European INventory of Existing Commercial chemical Substances

ELINCS: European List of Notified Chemical Substances

NLP: No-Longer Polymers

ENCS: Existing and New Chemical Substances Inventory

KECI: Korea Existing Chemicals Inventory

NZIoC: New Zealand Inventory of Chemicals

PICCS: Philippine Inventory of Chemicals and Chemical Substances

TSCA: Toxic Substances Control Act

TCSI: Taiwan Chemical Substance Inventory

INSQ: Inventario Nacional de Sustancias Químicas

NCI: National Chemical Inventory

FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances