

Flyaway Insecticidal Spray for Horses (Flyaway Insecticidal Spray for Horses)

Virbac (Australia) Pty Limited

Chemwatch Hazard Alert Code: 3

Chemwatch: 23-0246

Version No: 5.1

Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: 12/10/2021

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

Product Identifier

Product name	Flyaway Insecticidal Spray for Horses (Flyaway Insecticidal Spray for Horses)
Chemical Name	Not Applicable
Synonyms	APVMA No: 46791
Proper shipping name	ETHANOL (ETHYL ALCOHOL) or ETHANOL SOLUTION (ETHYL ALCOHOL SOLUTION)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Topical spray for horses for the treatment of nuisance insects such as bush flies, horse and stable flies, midges or sand flies and aiding the prevention of Queensland itch.
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Details of the manufacturer or supplier of the safety data sheet

Registered company name	Virbac (Australia) Pty Limited
Address	361 Horsley Road Milperra NSW 2214 Australia
Telephone	1800 242 100
Fax	+61 2 9772 9773
Website	au.virbac.com
Email	customer@virbac.com.au

Emergency telephone number

Association / Organisation	Poisons Information Centre
Emergency telephone numbers	13 11 26
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Chemwatch Hazard Ratings

	Min	Max
Flammability	3	3
Toxicity	1	1
Body Contact	1	1
Reactivity	1	1
Chronic	3	3

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

Poisons Schedule	Not Applicable
Classification [1]	Flammable Liquids Category 2, Sensitisation (Skin) Category 1, Reproductive Toxicity Category 1B, Hazardous to the Aquatic Environment Long-Term Hazard Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
Signal word	Danger

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Hazard statement(s)

H225	Highly flammable liquid and vapour.
H317	May cause an allergic skin reaction.
H360Df	May damage the unborn child. Suspected of damaging fertility.
H410	Very toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P233	Keep container tightly closed.
P280	Wear protective gloves and protective clothing.
P240	Ground and bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use non-sparking tools.
P243	Take action to prevent static discharges.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.
P302+P352	IF ON SKIN: Wash with plenty of water.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

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

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
64-17-5	>60	<u>ethanol</u>
134-62-3	1-5	<u>N,N-diethyl-m-toluamide</u>
51-03-6	1-5	<u>piperonyl butoxide</u>
52645-53-1	1-5	<u>permethrin</u>
113-48-4	1-5	<u>2-ethylhexyl bicycloheptene dicarboximide</u>
84-74-2	1-5	<u>dibutyl phthalate</u>
8000-28-0	1-5	<u>lavender oil</u>
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available).

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	<ul style="list-style-type: none"> ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor.
Ingestion	<ul style="list-style-type: none"> ▶ For advice, contact a Poisons Information Centre or a doctor at once. ▶ Urgent hospital treatment is likely to be needed. ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Transport to hospital or doctor without delay.

Indication of any immediate medical attention and special treatment needed

For chronic or short term repeated exposures to pyrethrum and synthetic pyrethroids:

- ▶ Mammalian toxicity of pyrethrum and synthetic pyrethroids is low, in part because of poor bioavailability and a large first pass extraction by the liver.
- ▶ The most common adverse reaction results from the potent sensitising effects of pyrethrins.
- ▶ Clinical manifestations of exposure include contact dermatitis (erythema, vesiculation, bullae); anaphylactoid reactions (pallor, tachycardia, diaphoresis) and asthma. [Ellenhor Barceloux]
- ▶ In cases of skin contact, it has been reported that topical application of Vitamin E Acetate (alpha-tocopherol acetate) has been found to have high therapeutic value, eliminating almost all skin pain associated with exposure to synthetic pyrethroids. [Incitec]

For acute or short term repeated exposures to ethanol:

- ▶ Acute ingestion in non-tolerant patients usually responds to supportive care with special attention to prevention of aspiration, replacement of fluid and correction of nutritional deficiencies (magnesium, thiamine pyridoxine, Vitamins C and K).
- ▶ Give 50% dextrose (50-100 ml) IV to obtunded patients following blood draw for glucose determination.
- ▶ Comatose patients should be treated with initial attention to airway, breathing, circulation and drugs of immediate importance (glucose, thiamine).
- ▶ Decontamination is probably unnecessary more than 1 hour after a single observed ingestion. Cathartics and charcoal may be given but are probably not effective in single ingestions.
- ▶ Fructose administration is contra-indicated due to side effects.

SECTION 5 Firefighting measures

Extinguishing media

- ▶ Alcohol stable foam.
- ▶ Dry chemical powder.
- ▶ BCF (where regulations permit).
- ▶ Carbon dioxide.
- ▶ Water spray or fog - Large fires only.

Special hazards arising from the substrate or mixture

Fire Incompatibility	▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves in the event of a fire. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Consider evacuation (or protect in place). ▶ Fight fire from a safe distance, with adequate cover. ▶ If safe, switch off electrical equipment until vapour fire hazard removed. ▶ Use water delivered as a fine spray to control the fire and cool adjacent area. ▶ Avoid spraying water onto liquid pools. ▶ Do not approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Liquid and vapour are highly flammable. ▶ Severe fire hazard when exposed to heat, flame and/or oxidisers. ▶ Vapour may travel a considerable distance to source of ignition. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). <p>Combustion products include: carbon dioxide (CO₂) other pyrolysis products typical of burning organic material.</p>
HAZCHEM	•2YE

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> ▶ Remove all ignition sources.
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	<ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb small quantities with vermiculite or other absorbent material. ▶ Wipe up. ▶ Collect residues in a flammable waste container.
Major Spills	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Consider evacuation (or protect in place). ▶ No smoking, naked lights or ignition sources. ▶ Increase ventilation. ▶ Stop leak if safe to do so. ▶ Water spray or fog may be used to disperse /absorb vapour. ▶ Contain spill with sand, earth or vermiculite. ▶ Use only spark-free shovels and explosion proof equipment. ▶ Collect recoverable product into labelled containers for recycling. ▶ Absorb remaining product with sand, earth or vermiculite. ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ Avoid smoking, naked lights, heat or ignition sources. ▶ When handling, DO NOT eat, drink or smoke. ▶ Vapour may ignite on pumping or pouring due to static electricity. ▶ DO NOT use plastic buckets. ▶ Earth and secure metal containers when dispensing or pouring product. ▶ Use spark-free tools when handling. ▶ Avoid contact with incompatible materials. ▶ Keep containers securely sealed. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	<ul style="list-style-type: none"> · Store in original containers in approved flame-proof area. · No smoking, naked lights, heat or ignition sources. · DO NOT store in pits, depression, basement or areas where vapours may be trapped. · Keep containers securely sealed. · Store away from incompatible materials in a cool, dry well ventilated area. · Protect containers against physical damage and check regularly for leaks. · Observe manufacturer's storage and handling recommendations contained within this MSDS. · Tank storage: Tanks must be specifically designed for use with this product. Bulk storage tanks should be diked (bundled). Locate tanks away from heat and other sources of ignition. Cleaning, inspection and maintenance of storage tanks is a specialist operation, which requires the implementation of strict procedures and precautions. Keep in a cool place. Electrostatic charges will be generated during pumping. Electrostatic discharge may cause fire. Ensure electrical continuity by bonding and grounding (earthing) all equipment to reduce the risk. The vapours in the head space of the storage vessel may lie in the flammable/explosive range and hence may be flammable. · For containers, or container linings use mild steel, stainless steel., Examples of suitable materials are: high density polyethylene (HDPE), polypropylene (PP), and Viton (FMK), which have been specifically tested for compatibility with this product., For container linings, use amine-adduct cured epoxy paint., For seals and gaskets use: graphite, PTFE, Viton A, Viton B. · Unsuitable material: Some synthetic materials may be unsuitable for containers or container linings depending on the material specification and intended use. Examples of materials to avoid are: natural rubber (NR), nitrile rubber (NBR), ethylene propylene rubber (EPDM), polymethyl methacrylate (PMMA), polystyrene, polyvinyl chloride (PVC), polyisobutylene., However, some may be suitable for glove materials o not cut, drill, grind, weld or perform similar operations on or near containers. Containers, even those that have been emptied, can contain explosive vapours

Conditions for safe storage, including any incompatibilities

Suitable container	▶ Packing as supplied by manufacturer.
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	<ul style="list-style-type: none"> ▶ Plastic containers may only be used if approved for flammable liquid. ▶ Check that containers are clearly labelled and free from leaks. ▶ For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : Where a can is to be used as an inner package, the can must have a screwed enclosure. ▶ For materials with a viscosity of at least 2680 cSt. (23 deg. C) ▶ For manufactured product having a viscosity of at least 250 cSt. (23 deg. C) ▶ Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used. ▶ Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages ▶ In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Avoid reaction with oxidising agents ▶ Avoid strong acids, bases.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	ethanol	Ethyl alcohol	1000 ppm / 1880 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	dibutyl phthalate	Dibutyl phthalate	5 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
ethanol	Not Available	Not Available	15000* ppm
piperonyl butoxide	6.5 mg/m3	72 mg/m3	1,200 mg/m3
dibutyl phthalate	15 mg/m3	1,600 mg/m3	9300* mg/m3

Ingredient	Original IDLH	Revised IDLH
ethanol	3,300 ppm	Not Available
N,N-diethyl-m-toluamide	Not Available	Not Available
piperonyl butoxide	Not Available	Not Available
permethrin	Not Available	Not Available
2-ethylhexyl bicycloheptene dicarboximide	Not Available	Not Available
dibutyl phthalate	4,000 mg/m3	Not Available
lavender oil	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
N,N-diethyl-m-toluamide	E	≤ 0.1 ppm
permethrin	D	> 0.01 to ≤ 0.1 mg/m ³
2-ethylhexyl bicycloheptene dicarboximide	E	≤ 0.1 ppm
lavender oil	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

Exposure controls

Appropriate engineering controls	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant.</p> <p>Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p>	
	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, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)	

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	<p>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</p> <p>1-2.5 m/s (200-500 f/min.)</p> <p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="384 309 1118 472"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <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> </tbody> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p> <ul style="list-style-type: none"> - Adequate ventilation is typically taken to be that which limits the average concentration to no more than 25% of the LEL within the building, room or enclosure containing the dangerous substance. - Ventilation for plant and machinery is normally considered adequate if it limits the average concentration of any dangerous substance that might potentially be present to no more than 25% of the LEL. However, an increase up to a maximum 50% LEL can be acceptable where additional safeguards are provided to prevent the formation of a hazardous explosive atmosphere. For example, gas detectors linked to emergency shutdown of the process might be used together with maintaining or increasing the exhaust ventilation on solvent evaporating ovens and gas turbine enclosures. - Temporary exhaust ventilation systems may be provided for non-routine higher-risk activities, such as cleaning, repair or maintenance in tanks or other confined spaces or in an emergency after a release. The work procedures for such activities should be carefully considered. The atmosphere should be continuously monitored to ensure that ventilation is adequate and the area remains safe. Where workers will enter the space, the ventilation should ensure that the concentration of the dangerous substance does not exceed 10% of the LEL (irrespective of the provision of suitable breathing apparatus) 	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>Personal protection</p>												
<p>Eye and face protection</p>	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] 											
<p>Skin protection</p>	<p>See Hand protection below</p>											
<p>Hands/feet protection</p>	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber <p>NOTE:</p> <ul style="list-style-type: none"> ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. <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"> - frequency and duration of contact, - chemical resistance of glove material, - glove thickness and - dexterity <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"> - When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. - When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. - Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. - Contaminated gloves should be replaced. <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> - Excellent when breakthrough time > 480 min - Good when breakthrough time > 20 min - Fair when breakthrough time < 20 min - Poor when glove material degrades <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"> - Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. - Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or 											

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	puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ PVC Apron. ▶ PVC protective suit may be required if exposure severe. ▶ Eyewash unit. ▶ Ensure there is ready access to a safety shower. ▶ Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity. ▶ For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets). ▶ Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.

Recommended material(s)**GLOVE SELECTION INDEX**

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

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

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Material	CPI
BUTYL	A
NEOPRENE	A
NITRILE	A
PE/EVAL/PE	A
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE+PVC	C
PVA	C
PVC	C
VITON	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Respiratory protection

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

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

^ - Full-face

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO₂), G = Agricultural chemicals, K = Ammonia(NH₃), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

SECTION 9 Physical and chemical properties**Information on basic physical and chemical properties**

Appearance	Clear flammable liquid; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	14	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	HIGHLY FLAMMABLE.	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available

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Solubility in water	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	<p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</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>Inhalation of pyrethrins may produce nausea, vomiting, sneezing, serious nasal discharge, nasal stuffiness and asthma. High concentrations may produce hyperexcitability, incoordination, tremors, muscular paralysis and death (due to respiratory failure).</p> <p>There have been some reports of transient facial tingling (paraesthesia) which lasts a few hours after exposure.</p> <p>In common with natural pyrethrins, the material may produce central nervous system stimulation with nausea, vomiting, gastroenteritis, diarrhoea, hypersensitivity, incoordination, tremors, muscle paralysis, convulsion, coma, and respiratory failure. Synthetic pyrethroids are neuropoisons acting on the axons in the peripheral and central nervous systems by interacting with sodium channels in mammals and/or insects. A single dose produces toxic signs in mammals, such as tremors, hyperexcitability, salivation, choreoathetosis (a condition characterised by aimless muscle movements and involuntary motions), and paralysis. The signs disappear fairly rapidly, and the animals recover, generally within a week. At near-lethal dose levels, synthetic pyrethroids cause transient changes in the nervous system, such as axonal swelling and/or breaks and myelin degeneration in the sciatic nerves. They are not considered to cause delayed neurotoxicity of the kind induced by some organophosphorus compounds. Pyrethroids have at least two distinct actions- a short-term pharmacological effect at near-lethal dose levels and a more long-term neurotoxic effect that results in sparse axonal nerve damage.</p> <p>This synthetic pyrethroid produces Type I poisoning syndrome (or "T" syndrome which is characteristic of those esters lacking an alpha-cyano substituent) and, in rats, causes such signs as sparring and aggressive behaviour, enhanced startle response, whole body tremor and prostration. Evidence indicates that Type I syndrome involves peripheral nerves in the mammal. Although this insecticide cannot be considered highly toxic in mammals its use indoors, in enclosed and poorly ventilated spaces may result in toxic effects in humans. Although natural pyrethroid produces allergic responses (rather than direct neurotoxicity) there is little evidence of allergic-type responses amongst humans exposed to the synthetic pyrethroid esters.</p> <p>[Cassaret & Doull's Toxicology: The Basic Science of Poisoning, 4th Ed.]</p> <p>Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination</p>
Ingestion	<p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>Ingestion of pyrethrins may produce nausea, vomiting, headache and other central nervous system disturbances. Excitation, muscular tremors and a period of shock may be followed by death. Dogs fed 5000 ppm of pyrethrum, for 90 days, developed dyspnae, tremors, ataxia and excessive salivation. An estimated fatal human dose is thought to be 100 gms. for a typical 70 kg man (1430 mg/kg).</p> <p>Phthalates (aromatic dicarboxylic acid esters), in general, exhibit low toxicity, partly because of poor absorption but mainly as a result of rapid metabolism in which the esters are saponified to phthalic acid (which is rapidly excreted) and the parent alcohol (which is subsequently metabolised). The pathology of these compounds seems to be related to the released alcohol and its biological effects. The rate of absorption of ingested phthalate esters is influenced by the content of dietary fat. Ingested phthalate esters may to a lesser degree be absorbed as the monoester derivatives or in the case of di(2-ethylhexyl)phthalate, as the diester. Cumulative toxicity of the phthalates has been observed on repeated administration. Both di-n-octyl phthalate and di(2-ethylhexyl)phthalate were found to have 22-28 times greater toxicity (based on LD50s) following repeated administration to animals. The liver has been shown to be the target organ affected by the phthalates. In general phthalates have induced liver enlargement; this increase in liver weight has been attributed to rapid cell division (hyperplasia) along with the detachment of cells (hypertrophy). The increase in liver weight caused by phthalates has been found to reverse to normal or even below normal levels on prolonged exposure.</p> <p>Exposure to phthalates, in general, has been found to be associated with a reduction in circulating cholesterol and serum triglyceride levels which accounted for a reduction in liver steroidogenesis. The phthalates also effect carbohydrate metabolism in the liver producing depleted glycogen electron transport inhibitors following interaction with mitochondria. Testicular atrophy produced in rats during feeding studies depends on the length and structure of the alcohol; in general the lower molecular weight esters produce the more severe effects. The toxicity of phthalic acid isomers decreases in the order o-phthalic acid, isophthalic acid and terephthalic acid. Phthalic acid is not metabolised but is excreted, unchanged, in the urine and faeces. Terephthalic acid appears to potentiate the biological effects of substances such as antibiotics, thiamine and sulfonamides.</p>
Skin Contact	<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>The material may produce moderate skin irritation; limited evidence or practical experience suggests, that the material either:</p> <ul style="list-style-type: none"> ▶ produces moderate inflammation of the skin in a substantial number of individuals following direct contact and/or ▶ produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. <p>Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and</p>

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	<p>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>Skin contact with natural pyrethrins may result in severe dermatitis and may also be associated with allergic rhinitis and asthma. Absorption through the skin may result in a toxic syndrome similar to that produced by inhalation. Systemic effects, following skin absorption, may include liver and kidney damage. Prolonged or repeated exposure may cause central nervous system effects and allergic skin reaction.</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 style="text-align: center;">Eye</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 style="text-align: center;">Chronic</p>	<p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.</p> <p>Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers</p> <p>Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.</p> <p>Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.</p> <p>There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of:</p> <ul style="list-style-type: none"> - clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. <p>On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>There is some evidence to provide a presumption that human exposure to the material may result in impaired fertility on the basis of: some evidence in animal studies 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 is not a secondary non-specific consequence of other toxic effects.</p> <p>Long-term exposure to ethanol may result in progressive liver damage with fibrosis or may exacerbate liver injury caused by other agents.</p> <p>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.</p> <p>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).</p> <p>(1) Boehncke W.H., & H.Gall, Clinical & Experimental Allergy, 26, 1089-1091, 1996</p> <p>Chronic poisoning by natural pyrethrins may result in convulsion, tetanic paralysis, rapid and uneven heart beat, liver and kidney damage, or death.</p> <p>The natural pyrethrins may produce hypersensitivity, especially following previous sensitising exposure. In general, repeated exposures over 2 or 3 years are required to elicit a response and involve exposure to pyrethrum rather than its individual components (including pyrethrins). The sesquiterpene lactone (pyrethrosin) and the pyrethrum glycoproteins account for the immediate and delayed hypersensitivity seen in guinea pigs following a single injection of ground chrysanthemum in Freud's adjuvant. Mild erythematous vesicular dermatitis (with papules), pruritus, localized oedema (particularly of the face, lips and eyelids), rhinitis, tachycardia, pallor and sweating are the most common syndromes. An initial skin sensitisation can progress to marked dermal oedema and skin cracking. Pyrethrum dermatitis appears to increase in hot weather or under conditions where heavy perspiration is produced. The active ingredients of pyrethrum (except pyrethrin II) are inactive in patch tests. Those patients allergic to ragweed pollen are particularly sensitive to pyrethrin.</p> <p>Rats fed on a diet of pyrethrins for 5000 ppm for 2 years showed some signs of tissue damage including liver lesions, bile duct proliferation and focal necrosis of the liver cells. A no-effect level of 1000 ppm found in animal experiments correspond to a daily dose of 3600 mg/man.</p> <p>Principal routes of exposure are usually by inhalation of mists or vapours from heated material and skin contact/absorption.</p> <p>A 5 year old girl sprayed with Deet nightly for 3 months, developed headaches and slurred speech, progressing to athetosis (ceaseless slow, writhing motions especially of the hands), shaking, screaming and convulsion. She died 24 days after hospitalisation; autopsy revealed generalised oedema of the brain with intense congestion of the meninges. The effect was thought to represent sensitisation to the substance</p> <p>Repeated application to human skin resulted in slight irritation and dryness of the face, desquamation around the nose and a slight tingling sensation. Incidences of sporadic allergy (anaphylaxis) and scarring dermatitis have been reported. Some individuals repeatedly exposed to the substance have shown encephalopathy and neurological symptoms (muscle cramp, urinary hesitation, insomnia, abnormal sweating, irritability, depression, paranoia, episodes of confusion, and aggressive behaviour).</p> <p>An increased incidence sperm head abnormalities and period nausea, vomiting and nasal exudate were observed in animals following chronic exposure</p> <p>Oral or intraperitoneal administration of dibutyl phthalate, at high doses relative to the TLV, produced a number of resorptions, neural tube defects, skeletal abnormalities and increased foetal deaths.</p> <p>Certain substances, commonly found in perfumes or perfumed products, produce hypersensitivity. Sensitising constituents have been classified as Class A (common sensitisers) or Class B (rare sensitisers) in a Japanese study (Nakayama 1998). Contact allergy to perfumes occurs with a relatively high incidence, such incidence only surpassed by nickel allergy in the community. In a Danish study, it was found that about 1.1% of the population was allergic to Peru balsam or "fragrance mix".</p> <p>There is no cure for perfume allergy. Once sensitised, exposure to even minute amounts of the perfume, gives rise to eruptions and eczema. These symptoms may be treated with steroid creams, although frequent recourse to this treatment produces unwanted side-effects.</p> <p>A number of common flavor and fragrance chemicals can form peroxides surprisingly fast in air. Antioxidants can in most cases minimize the oxidation.</p> <p>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.</p> <p>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</p>

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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.

The primary effect of long-term exposure to methylenedioxyphenol insect synergists such as the piperonyls (such as piperonyl butoxide - PBO) in animals is an increase in liver and thyroid weight, liver and kidney damage, and a decrease in body weight. These symptoms were observed in a diet of 52.8 mg/kg or more a day in a chronic study with dogs.

PBO is a possible human carcinogen. Currently there is no data from accidental exposure available regarding its carcinogenicity in humans; the only information is from animal studies. Several studies have shown that PBO treatment in rats causes an increase in liver cancer at high doses. The incidence of hepatocellular carcinoma, in male and female rats given 2.4% piperonyl butoxide was 80.0% and 57.7% respectively. Preneoplastic hepatic lesions such as nodular hyperplasia, cholangiofibrosis, and nodular hyperplasia were also seen.

Some studies have shown that PBO treatment in rats corresponds with a very slight increase in thyroid cancer.

Rats fed diets containing from 0.6 to 2.4% piperonyl butoxide for approximately two years showed dose-related decreases in body weight. Roughened hair, lethargy, epistaxis, abdominal swelling, and decreased food consumption were observed at 2.4%. All dose rates induced skin tumours after about 1 year. Cumulative mortality varied from around 15 to 50%. Caecal haemorrhage was the cause of death. Dead rats with hepatic tumours were seen from week 74, but caecal haemorrhage or possible leukaemia was the cause of death. At necropsy in rats surviving to the end of the study, hepatocellular adenomas and carcinomas occurred in both sexes in a dose-related manner. A dose-related increase in thrombocytopenia was seen in male rats. The authors * of this study concluded that the primary feature of chronic piperonyl-butoxide toxicity is hepatocarcinogenicity.

It is generally accepted that PBO does not demonstrate any significant potential for mutagenicity (genetic damage) but debate still exists.

PBO weakens the immune system by inhibiting lymphocyte response. Lymphocytes are a class of white blood cells that consume potentially dangerous pathogens and release antibodies. Inhibiting lymphocyte response weakens the body's ability to defend against foreign invaders. Preventing the breakdown of toxic chemicals, may exacerbate potentially toxic effects.

PBO has been shown to adversely affect a variety of reproductive functions. Two-generational laboratory studies on rats show that litter weight and size are less for mothers exposed to high concentrations of PBO, and there is an increase in birth defects and fetal death. In one study the difference in the average weight of PBO-exposed offspring immediately after birth is negligible, but 7-14 days post-natal is significantly greater for those mothers that are exposed to PBO than for those that are not. The U.S. EPA maintains that results for teratogenicity (the ability to produce birth defects) in animals have been mixed, and while some studies suggest some teratogenicity, most do not. PBO may also interfere with sexual development because the enzymes it inhibits are responsible not only for the breakdown of toxic chemicals but also for the metabolism of other compounds such as steroids, which include the sex hormones. Rats exposed to PBO over the course of two years experience an atrophy of the testes a decrease in weight of the seminal vesicles (sperm producing structures), and an increase in ovarian weights. There is no evidence that PBO affects fertility.

Data has shown that PBO alone interferes with enzymes that maintain homeostasis of sodium and calcium in the brain and nervous system, possibly affecting neural response. Additionally, it increases the neurotoxicity of other compounds. Despite this data, EPA believes that these neurotoxic effects are slight and maintains that PBO poses no neurological risk.

Behavioral changes have been noted with PBO as well. In a laboratory experiment, exposed rats experience more trouble navigating a maze than unexposed rats. The exposed rats travel longer distances and turned more frequently in the maze. PBO also induces changes in olfactory behavior of the offspring of exposed mothers. Offspring of exposed mothers are less likely to enter a compartment that smells like home than unexposed mothers. Exploratory behavior in mice increases as the dose of PBO they were treated with increased. This data shows that PBO has the ability to affect behaviors in mammals.

Research on rats has found that PBO can cause intestinal ulcers and bleeding. Liver damage is common in studies, and kidney damage has been found as well. Long-term ingestion of PBO causes anemia, a decrease in the amount of hemoglobin (oxygen-transporting molecules) in blood, and increases the blood cholesterol level in rats. PBO can also damage the larynx, and there have been reports that it can cause labored breathing, an accumulation of fluid in the lungs, nasal bleeding, abdominal swelling, and loss of the ability to coordinate muscle movement. There has been a fair amount of investigation into the effects of dermal contact with PBO since it is used as a topical agent for lice, but there has been no evidence of it causing any local or systemic toxicity, and the amount of PBO absorbed from skin contact is characterized by some researchers as low.

ChemicalWatch Fact Sheet

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TOXICITY

Not Available

IRRITATION

Not Available

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ethanol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 17100 mg/kg ^[1]	Eye (rabbit): 500 mg SEVERE
	Inhalation(Rat) LC50: 64000 ppm4h ^[2]	Eye (rabbit):100mg/24hr-moderate
	Oral (Rat) LD50; 7060 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit):20 mg/24hr-moderate
		Skin (rabbit):400 mg (open)-mild
		Skin: no adverse effect observed (not irritating) ^[1]
N,N-diethyl-m-toluamide	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 3180 mg/kg ^[2]	Eye (rabbit) : 10 mg - moderate
	Oral (Rat) LD50; 1950 mg/kg ^[2]	Eye (rabbit): 100 mg
		Skin (rabbit): 500 mg - moderate
piperonyl butoxide	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Not Available
	Inhalation(Rat) LC50: >5.2 mg/4h ^[1]	
	Oral (Rat) LD50; >2000 mg/kg ^[1]	
permethrin	TOXICITY	IRRITATION
	dermal (rat) LD50: 1750 mg/kg ^[2]	Skin (rabbit): 500 mg/24h - mild
	Oral (Rat) LD50; 383 mg/kg ^[2]	
2-ethylhexyl bicycloheptene dicarboximide	TOXICITY	IRRITATION
	dermal (rat) LD50: 470 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation(Rat) LC50: 1.94 mg/4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Mouse) LD50; 1000 mg/kg ^[2]	
dibutyl phthalate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation(Rat) LC50: >=15.68 mg/4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50; 8000 mg/kg ^[2]	
lavender oil	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]
	Oral (Rat) LD50; 4250 mg/kg ^[2]	Skin (rabbit): 500 mg/24h mild
		Skin: adverse effect observed (irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
Legend:	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

N,N-DIETHYL-M-TOLUAMIDE	<p>Reproductive effector in rats For N,N-diethyl-m-toluamide (Deet)</p> <p>Acute toxicity: Different preparations of Deet with different proportions of the m-isomer produced different oral LD50s. Rats killed by dosages in the LD50 range showed lacrimation, chromodacryorrhea, depression, prostration, tremors, and asphyxial convulsions. Respiratory failure usually preceded cardiac failure.</p> <p>In rabbits, an intravenous dosage of 75 mg/kg was rapidly fatal, but 50 mg/kg was not. Five doses at the rate of 25 mg/kg/day produced no cumulative effect, except for injury of the intima of some veins used for injection. Single dermal applications to rabbits at rates of 2 or 4 ml/kg produced no systemic effect, but did produce mild to moderate erythema. Repeated dermal application of 50% solutions for 13 weeks at the rate of 2 ml/kg/day produced no evidence of systemic toxicity but did produce desquamation, coriaceousness, dryness, and fissuring in the same species. Except for some scarring, these lesions cleared within 3 weeks. Instillation of Deet into the eyes of rabbits produced mild to moderate edema of the nictitating membrane, lacrimation, conjunctivitis, and some corneal injury, as revealed by fluorescein staining. After 5 days, all eyes appeared normal. No sensitisation was seen in guinea pigs .</p> <p>Animals topically exposed to Deet have developed dermal and ocular reactions. Dermal effects including erythema, desquamation and scarring in rabbits and profuse sweating, irritation and exfoliation in horses have been reported following repeated applications of Deet at concentrations of 50 percent or greater. Direct ocular application of either diluted (30 or 40 percent Deet) or undiluted Deet in rabbits has produced edema, tearing, conjunctivitis, pus and clouding in the eyes.</p> <p>Repeated dermal application to horses produced hypersteatosis, an overactivity of the sebaceous glands, when the solution of Deet was 15% or higher.</p> <p>Dermal application in humans of insect repellents containing Deet can produce a variety of skin reactions in humans. Cases of localized skin irritation, large painful blisters and permanent scarring of skin at the crease of the elbow have been reported in soldiers who applied solutions of 50 or 75 percent Deet. Results from questionnaire surveys conducted by the National Institute for Occupational Safety and Health (NIOSH) among Everglades National Park Employees indicated a variety of dermal reactions including rashes, irritation of skin and mucous membranes, and numb or burning sensations of the lips among park workers who were highly exposed to Deet-containing repellents. Urticaria or dermatitis, resulting from topical Deet exposure has been noted in both children and adults. In one instance involving only limited Deet exposure, the urticaria was accompanied by an anaphylactic reaction .</p> <p>Controlled human exposure studies using 50 or 75 percent Deet have reproduced many of the dermal effects noted in field studies. The U.S. Army conducted an investigation in volunteers using 75 percent Deet applied to the upper arm and elbow's crease. Of the 77 volunteers, 37</p>
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	<p>(48%) had severe dermal reactions at the crease of the elbow. No dermal reactions were observed on the upper arm or in the control group of men tested with ethanol solvent alone.</p> <p>Several cases of toxic encephalopathy associated with the use of Deet in children have been reported in the medical literature. The first reported case involved a 3.5 year old girl whose body, bedclothes and bedding were sprayed each night for two weeks with an insect repellent containing 15 percent Deet. Since then, five additional cases of toxic encephalopathy have been temporally associated with the use of Deet products in children, all of whom were females. The toxic encephalopathy was characterised by agitation, weakness, disorientation, ataxia, seizures, coma and in three cases resulted in death. Autopsies conducted on two fatalities indicated oedema of the brain, with one case presenting necrotic lesions in the cerebellum and spinal cord and an enlarged liver accompanied by microscopic changes. One child was reported to be heterozygous for ornithine carbamoyl transferase deficiency (a sex linked enzyme deficiency which may produce effects similar to those reported above) and it has been hypothesised that children with this enzyme disorder may be at greater risk of adverse reactions to Deet. This enzyme deficiency which usually causes infant death in males is of variable severity in females. Accidental and deliberate ingestion of Deet-containing products has produced neurotoxic effects similar to those described following dermal exposure.</p> <p>Generalised seizures have also been temporally associated with the use of Deet-containing insect repellent on skin. These cases differ from those described above in that they involved males (four boys aged 3-7 years and one 29-year-old adult), had few associated neurotoxic effects and resolved rapidly. Lower exposure to Deet in these males (four of five males had either one or two dermal applications) may have accounted for the effects being less severe than in females. That the majority of identified neurotoxic cases involved children raises concerns that this subpopulation is at greater risk of adverse reaction following exposure to Deet than are adults.</p> <p>Signs and symptoms of more subtle neurotoxicity have also been associated with extensive dermal application of Deet in adults. Questionnaire results indicate that Everglades National Park employees having extensive Deet exposure were more likely to have insomnia, mood disturbances and impaired cognitive function than were lesser exposed co-workers. A young male who repeatedly applied Deet to his skin prior to spending prolonged periods in a sauna was reported to develop acute manic psychosis characterized by aggressive behavior, delusions and hyperactivity. Either o-DET or p-DET, or both occur as impurities in commercial m-DET (Deet). A thorough study of the o- and p-isomers showed that the o-isomer is slightly more toxic than the others (oral LD50 1,210 mg/kg in rats). However, no alarming difference was found, and it was concluded that the presence of 5% of o-DET or p-DET as impurities in the</p> <p>Chronic toxicity: When rats were fed Deet at a dietary level of 10,000 ppm for about 200 days, their growth rate was decreased without a decrease in food intake. There was a significant increase in the relative weight of the testes and liver in males, of the liver and spleen in females, and the kidneys of both males and females. Some of these changes were seen in lesser degree at a dietary level of 1,000 ppm. No gross or significant histological changes were seen at any dietary level and no changes of any kind were noted at 100 ppm or 500 ppm (about 25 mg/kg/day).</p> <p>Essentially identical results were found in other subacute dermal and feeding studies each carried out on rats, rabbits, and dogs. In these oral studies, 2,000 ppm proved to be a no-effect-level. Oral administration of Deet to dogs at rates of 100 and 300 mg/kg/day caused tremor and hyperactivity and occasional vomiting, but no other effects. Blood studies (hemoglobin, haematocrit, sedimentation rate, platelet counts, total and differential white cell counts) on dogs receiving 300 mg/kg orally or dermally or on rabbits receiving 300 mg/kg dermally revealed no effect on the haematopoietic system. Gross and microscopic examination of the organs of all three species revealed only slight kidney damage in rabbits typical of that associated with burns of the skin. Thirteen other organs, including liver, spleen, and bone marrow, were normal in the three species.</p> <p>No systemic toxicity was observed in rats exposed 8 hours/day, 5 days/week for 7 weeks to air saturated with Deet. No toxic effects were observed in rats exposed for 6 hours to an aerosol of Deet. No gross or significant histological changes were seen.</p> <p>Organ Toxicity: Hypertrophy of the kidneys and liver and effects of mild central nervous system stimulation including tremors and hyperactivity were noted in animals following repeated exposure. Significant testicular hypertrophy was observed in male rats repeatedly fed a diet containing from 48 to 531 mg/kg/day of Deet</p> <p>Reproductive Effects: When Deet was applied to the skin of rats at the rate of 1,000 mg/kg/day throughout pregnancy, implantation was reduced significantly. Prenatal mortality was 34.1%, compared with 20.9% in the control. Mortality between birth and weaning was 44.0%, compared to 15.7% in the control. Injury was less (but probably significant) at a dosage of 100 mg/kg/day throughout pregnancy.</p> <p>Teratogenic Effects: A dermal teratology study was conducted on rabbits. Groups of 20 pregnant rabbits received daily dermal applications of 0, 50, 100, 500, 1000, or 5000 mg Deet/kg/day in ethanol on shaved backs from day 0 through day 29 of gestation. There were no significant differences between control and treated animals with respect to the fertility index, number of implantations per animal, or number of fetuses per animal. In addition, treatment did not change fetal weight, fetal length or placental weights and no increases in the incidence of skeletal or soft tissue anomalies were observed in treated groups when compared with untreated controls. This study demonstrated that Deet has no teratogenic or embryotoxic effects in rabbits exposed dermally to technical Deet.</p> <p>An additional supplementary teratology study was conducted on rats. Groups of 20 pregnant rats were daily administered 10 ml of peanut oil containing 0, 8, 20 or 80 mg/kg/Deet by gavage from day 5 through day 15 of gestation. No significant differences were reported between control and treated mothers with respect to fertility, fetuses per litter, foetal weight or foetal survival. However, the study did show decreases in number of implantation sites per dam and number of fetuses per animal. In addition, a related increase was observed in the number of resorptions per dam</p> <p>Carcinogenicity: Researchers fed Deet to male and female rats in the diet for two years at doses of 10, 30, or 100 mg/kg/day, and 30, 100, or 400 mg/kg/day, respectively. Researchers fed mice 250, 500, or 1,000 mg/kg/day for 18 months, and dogs 30, 100, or 400 mg/kg/day. No specific target organ toxicity or oncogenicity was observed in any of the animals. Researchers often use studies designed to test for mutagenicity to screen chemicals for carcinogenicity. Sufficient evidence indicates that DEET does not have significant potential for mutagenicity</p> <p>Fate in Humans and Animals: Deet is absorbed promptly from the skin and distributed to all organs including the brain and the foetus. The compound is excreted in the milk but primarily in the urine</p> <p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
PIPERONYL BUTOXIDE	Dermal (rabbit) LD50: >1880 mg/kg [Handbook of Toxicology] *Published value - probably not peer-reviewed ADI: 0.03 mg/kg
PERMETHRIN	<p>Oral (rat) LD50: 430-4000 mg/kg * Oral (mouse) LD50: 540-2960 mg/kg * cis/trans ratio: 40:60 cis/trans ratio: 20:80 ADI: 0.05 mg/kg for nominal cis-trans 40:60 and 25:75 isomers only</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 epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>[* The Pesticides Manual, Incorporating The Agrochemicals Handbook, 10th Edition, Editor Clive Tomlin, 1994, British Crop Protection Council]</p>
2-ETHYLHEXYL BICYCLOHEPTENE DICARBOXIMIDE	<p>For 2-ethylhexyl (or N-octyl) bicycloheptene dicarboximide (MGK-264)</p> <p>Dermal Absorption: A study with human volunteers indicated that the dermal absorption factor for MGK-264 is approximately 10% based on the combination of radiolabelled material in the urine (about 1%) and unaccounted for radioactivity (about 9%, assumed to be retained in the body).</p> <p>Subchronic Inhalation Toxicity: A 90-day rat inhalation toxicity study demonstrated that at the lowest dose tested, there were indications of metaplasia/hyperplasia and changes in the larynx. At higher doses, histopathology of the larynx revealed additional changes and more intense changes in the epithelium and throat. Thus, inhalation exposure is capable of causing alterations in the respiratory tract.</p> <p>Immunotoxicity and Neurotoxicity: There were no indications of immunotoxicity or specific neurotoxicity</p> <p>Subchronic and Chronic Oral Toxicity: The liver is the target organ of MGK-264. Liver effects were noted in the adults in the rat chronic/oncogenicity study, the mouse chronic/oncogenicity study, the rat multi-generation reproduction study and subchronic and chronic dog studies. The dog appeared to be the most sensitive species for liver alterations but these alterations were limited to slight to moderate brown pigment and circulating enzyme changes. The dog study did not include histopathology of the liver to verify the presence of degenerative conditions. In the mouse, liver changes include bile duct histological changes including liver tumors, as well as kidney weight effects and brown pigment.</p> <p>Carcinogenicity: MGK-264 has been identified as a possible human carcinogen based on statistically significant increases mainly in benign liver adenomas in both sexes of mice at doses approaching the limit dose and on increases in benign thyroid follicular tumors in male rats at doses considered to be adequate to assess carcinogenic potential.</p> <p>Developmental Toxicity: The rat and rabbit developmental toxicity studies did not demonstrate developmental toxicity for MGK-264. Maternal</p>

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toxicity consisted of body weight and food consumption decreases. However, at higher doses, abortions, resorptions, and deaths were noted.

Reproductive Toxicity: There were no effects on the reproductive performance of either males or females in the multi-generation reproduction study. Systemic effects were related to body weight decrease as well as histopathological changes in the liver similar to those seen in the rat chronic feeding study. However, offspring for all generations indicated decreased body weight during lactation at a lower dose than parental systemic effects. The effect was reversible after weaning as pups regained weight and their weights were comparable to control animal weights after weaning.

Mutagenicity: Mutagenicity and genotoxicity were not evident in the Ames test for bacterial mutations, in the unscheduled DNA synthesis, or in a chromosome aberration studies. Although MGK-264 was considered weakly positive in the mouse lymphoma assay, there was a low concern for mutagenic or genetic toxicity.

Metabolism: The metabolism and pharmacokinetics data for MGK-264 in rats demonstrated that MGK-264 is absorbed and excreted with little retention of metabolites

For dibutyl phthalate (DBP):

In studies on rats, DBP is absorbed through the skin, although in *in vitro* studies human skin has been found to be less permeable than rat skin to this compound. Studies in laboratory animals indicate that DBP is rapidly absorbed from the gastrointestinal tract, distributed primarily to the liver and kidneys of rats and excreted in urine as metabolites following oral or intravenous administration. Following inhalation, it was consistently detected at low concentrations in the brain. Available data indicate that in rats, following ingestion, DBP is metabolised by nonspecific esterases mainly in the small intestine to yield mono- *n*-butyl phthalate (MBP) with limited subsequent biochemical oxidation of the alkyl side chain of MBP. MBP is stable and resistant to hydrolysis of the second ester group. Accumulation has not been observed in any organ. The profile of effects following exposure to DBP is similar to that of other phthalate esters, which, in susceptible species, can induce hepatomegaly, increased numbers of hepatic peroxisomes, foetotoxicity, teratogenicity and testicular damage.

Acute toxicity: The acute toxicity of DBP in rats and mice is low. Signs of acute toxicity in laboratory animals include depression of activity, laboured breathing and lack of coordination. In a case of accidental poisoning of a worker who ingested approximately 10 grams of DBP, recovery was gradual within two weeks and complete after 1 month.

On the basis of limited available data in animal species, DBP appears to have little potential to irritate skin or eyes or to induce sensitization. In humans, a few cases of sensitization after exposure to DBP have been reported, although this was not confirmed in controlled studies of larger numbers of individuals reported only in secondary accounts

Repeat dose toxicity: In short-term repeated-dose toxicity studies, effects at lowest levels in rats after oral administration for 5 to 21 days included peroxisome proliferation and hepatomegaly at doses of 420 mg/kg body weight per day or more. In longer-term studies, the effects in rats observed following ingestion of DBP for periods up to 7 months included reduced rate of weight gain at doses of 250 mg/kg body weight per day or more. Increase in relative liver weight has been observed at doses of 120 mg/kg body weight or more. Peroxisomal proliferation with increased peroxisomal enzyme activity has been observed at doses of 279 mg/kg body weight per day or more. Necrotic hepatic changes in Wistar rats have been reported at doses of 250 mg/kg body weight per day or more but not in F-344 or Sprague-Dawley rats exposed to up to 2500 mg/kg body weight per day. Alteration in testicular enzymes and degeneration of testicular germinal cells of rats have been observed at doses of 250 and 571 mg/kg body weight per day. There are considerable species differences in effects on the testes following exposure to DBP, minimal effects being observed in mice and hamsters at doses as high as 2000 mg/kg body weight per day. In mice, effects on body and organ weights and histological alterations in the liver indicative of metabolic stress have been reported in a recent subchronic bioassay, for which the no-observed-effect-level (NOEL) was 353 mg/kg body weight per day.

Developmental toxicity: . In a continuous breeding protocol, which included cross-over mating and offspring assessment phases, rats were exposed to 0, 1000, 5000 or 10 000 mg DBP/kg in the diet (equivalent to 0, 66, 320 and 651 mg/kg body weight per day). In the first generation the reduction in pup weight in the mid-dose group, in the absence of any adverse effect on maternal weight, could be regarded as a developmental toxicity effect. There was also a significant reduction of live litter numbers at all three dose levels. The effects in the second generation were more severe, with reduced pup weight in all groups including the low-dose group, structural defects (such as prepuccial/ penile malformations, seminiferous tubule degeneration, and absence or underdevelopment of the epididymides) in the mid- and high-dose groups, and severe effects on spermatogenesis in the high-dose group that were not seen in the parent animals. These results suggest that the adverse effects of DBP are more marked in animals exposed during development and maturation than in animals exposed as adults only. No clear NOEL was established in this study. The lowest-observed- adverse-effect-level (LOAEL) was considered to be 66 mg/kg body weight per day. The available studies show that DBP generally induces foetotoxic effects in the absence of maternal toxicity. Available data also indicate that DBP is teratogenic at high doses and that susceptibility to teratogenesis varies with developmental stage and period of administration. In mice, DBP caused dose-dependent increases in the number of resorptions and dead fetuses at oral doses of 400 mg/kg body weight per day or more. Dose-dependent decreases in fetal weights and number of viable litters were also observed in mice at these doses. Adequate carcinogenesis bioassays for DBP have not been conducted. The weight of the available evidence indicates that DBP is not genotoxic.

DIBUTYL PHTHALATE

The material may produce peroxisome proliferation. Peroxisomes are single, membrane limited, cytoplasmic organelles that are found in the cells of animals, plants, fungi and protozoa. Peroxisome proliferators include certain hypolipidaemic drugs, phthalate ester plasticisers, industrial solvents, herbicides, food flavours, leukotriene D4 antagonists and hormones. Numerous studies in rats and mice have demonstrated the hepatocarcinogenic effects of peroxisome proliferators, and these compounds have been unequivocally established as carcinogens. However it is generally conceded that compounds inducing proliferation in rats and mice have little, if any, effect on human liver except at very high doses or extreme conditions of exposure.

Transitional Phthalate Esters: produced from alcohols with straight-chain carbon backbones of C4 to C6. This subcategory also includes a phthalate produced from benzyl alcohol as one ester group with the second ester composed of an alkyl group with a C5 carbon backbone and butyrate group. Phthalate esters containing >10% C4 to C6 molecules were conservatively included in this subcategory. Branched C7 and C8 isomers (di-iso-heptyl, di-iso-octyl and diethylhexyl phthalates) in contrast to linear dihexyl and dioctyl phthalate are members of this family. Transitional phthalates have varied uses, but are largely used as plasticisers for PVC. Physicochemical properties also vary in that the lower molecular weight transitional phthalates are more water-soluble than higher molecular weight transitional phthalates, but none would be characterised as highly water soluble. Transitional phthalates have lower water solubility than the low molecular weight phthalates and except for butylbenzyl phthalate (BBP), existing data suggest they do not exhibit acute or chronic aquatic toxicity. What distinguishes some of the transitional phthalates from others is their greater mammalian toxicity potential, particularly with regard to reproductive and developmental effects, compared to either the low or high molecular weight phthalate subcategories

Acute Toxicity. The available data on phthalates spanning the carbon range from C4 to C6 indicate that phthalate esters in the transitional subcategory are minimally toxic by acute oral and dermal administration. The oral LD50 value for BBP exceeds 2 g/ kg, and for materials with higher molecular weights, the LD50 values exceed the maximum amounts which can be administered to the animals in a manner consistent with the principles of responsible animal use.

One member of this subcategory, diethylhexyl phthalate (DEHP), has been tested for acute inhalation toxicity. It did not cause an effect at the highest concentration tested. Further, considering the low volatility of these substances, inhalation exposure at toxicologically significant levels is not anticipated.

Repeated Dose Toxicity. Several substances in the C4 to C6 range, including BBP, have been tested for repeated dose toxicity in studies ranging from 3 weeks to 2 years . The principal effects found in these studies were those associated with peroxisome proliferation including liver enlargement and induction of peroxisomal enzymes. As shown in a comparative study of liver effects, the strongest inducers of peroxisome proliferation are diisononyl phthalate (DINP) and di-iso-decyl phthalate (DIDP) with substances of shorter chain length (e.g., BBP) showing much less pronounced effects. Thus it is reasonable to conclude that other members of this subcategory would show effects similar to BBP and less pronounced than DINP or DIDP. It should also be noted that the relevance of these findings to human health is, at best, questionable. It has been shown that these effects are mediated through the peroxisome proliferation-activated receptor alpha (PPARα) and that levels of PPARα are much higher in rodents than they are in humans. Thus one would expect humans to be substantially less responsive than rodents to peroxisome proliferating agents. Empirical evidence that this is true is provided by studies in primates in which repeated administration of DINP had no effects on liver, kidney or testicular parameters.

Several of the substances in the transitional phthalate esters subcategory, however, have been shown to produce testicular atrophy when given to juvenile rats at high levels. Testicular atrophy has been associated with BBP and other substances with C4 to C6 linear carbon chains.

However, molecules with fewer than 4 or more than 6 carbons did not produce testicular atrophy in these studies. Although the relevance of these data are uncertain, as the testes is not a target organ for diethylhexyl phthalate (DEHP) in primates, these data do provide one of the

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distinguishing toxicological characteristics of this subcategory and are one of the underlying reasons supporting the differentiation of phthalate esters on the basis of length of the linear region of the carbon chain.

Genetic Toxicity (Salmonella). A number of the substances in this subcategory including the reference substance BBP has been assessed in the Salmonella and mouse lymphoma assays. All of these substances were inactive in these assays.

Chromosomal Aberrations. BBP and dihexyl phthalate (DHP) were inactive in micronucleus assays in mice. DEHP was inactive in a cytogenetics assay in rat bone marrow. Diisooheptyl phthalate was inactive in CHO cells, in vitro..

Reproductive toxicity: A series of studies assessed the structure-activity relationship of the effects of phthalate esters on fertility using a continuous breeding protocol. The test substances included in these studies were diethyl-, dipropyl-, dibutyl-, dipentyl-, d-n-hexyl-, di-(2-ethylhexyl)-, and di-n-octyl phthalates. The most profound effects were on fertility (i.e., number of females delivering/ number mated) and number of live births. The substance showing the greatest activity was DEHP which produced effects at dietary levels of 0.1 % with a no effect level of 0.01 %. The next most active compounds were di-n-hexyl- and di-n-pentyl phthalate which showed effects in the range of 0.3 to 0.5 %; no effect levels were not experimentally defined. Dipropyl phthalate had an effect on live birth index at 2.5 % but produced no effects at 1.25 %. Diethyl phthalate and di-n-octyl phthalate were inactive at the highest levels tested, 2.5 % and 5.0 %, respectively. These data demonstrated that molecules with linear alkyl chains of 4 to 6 carbons profoundly affect fertility in rodents, with DEHP being the most active. Molecules with longer or shorter side chains are essentially inactive in these assays. These data were also a basis for the separation of phthalates into three categories based on length of side chain.

In addition to these data there are reproductive toxicity studies on BBP and DEHP.

A 2-generation reproductive study was conducted in rats in which BBP was administered via the diet. Parental effects were limited to changes in body weight, weight gain, and increased absolute and relative liver weights. In the F1 parents, treatment with BBP affected mating and fertility indices and sperm number and motility. The F1 male offspring exhibited shortened anogenital distance, delayed acquisition of puberty and retention of nipples and areolae as well as reproductive effects. The NOAEL of the study was reported to be 3750 mg/ kg for reproductive effects. However, for male F1 and F2 offspring, the NOEL for reproductive effects was reported to be 50 mg/ kg based on reductions in anogenital distance. These studies along with previous data provide a good basis to assess the reproductive effects of C4 to C6 phthalate esters. Although several substances (diheptyl, heptyl nonyl, heptyl undecyl) have ester side chain constituents that predominately fall in the high molecular weight subcategory, these substances are conservatively assumed to exhibit reproductive effects similar to other transitional phthalates.

Developmental toxicity: There have been extensive studies of the developmental toxicity of BBP and DEHP. These substances produce structural malformations and also affect male reproductive development. No effect levels are in the range of 50 to 300 mg/ kg bw/ day. There is also an unpublished developmental toxicity study of di-isooheptyl phthalate (DIHP). The results of these studies are broadly consistent with the structure-activity relationships previously described, i.e., that phthalate esters with linear carbon chains of C4 to C6 carbons produce much more profound effects than either shorter or longer molecules.

Phthalate esters with >10% C4 to C6 isomers were conservatively placed in the transitional subcategory. This conclusion is supported by developmental test data on "711P" (which showed structural malformations in rats at 1000 mg/ kg/ day with a NOAEL of 200 mg/ kg/ day. "711P" is an equal composition mixture of six phthalate esters consisting of linear and methyl-branched C7, C9, and C11 ester side chains. This test substance is considered by EPA under the following CAS Numbers.: 68515-44-6 (di C7), 68515-45-7 (di C9), 3648-20-2 (di C1), 111381-89-6 (C7, C9), 111381-90-9 (C7, C11), and 111381-91-0 (C9, C11). The overall content of C4 to C6 isomers in "711P" is approximately 10%, based on the contribution from methyl-branched C7 isomers e.g., di C7 (30% C4-C6); C7, C9 (15% C4-C6); and C7, C11 (15% C4-C6). Test data on 711P were used selectively as read-across data to the C7-containing substances in the mixture, based on the C4 to C6 content of each substance in the mixture.

LAVENDER OIL

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.

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

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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.

For monoterpenes:

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

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

Members of this chemical category are of very low acute toxicity

Studies of terpene hydrocarbons indicate that they are rapidly absorbed, distributed, metabolised and excreted. The principal metabolic pathway involves side chain oxidation to yield monocyclic terpene alcohols and carboxylic acids. These metabolites are mainly conjugated with glucuronic acid and excreted in the urine, or to a lesser extent in the feces. A secondary pathway involves epoxidation of either the exocyclic or endocyclic double bond yielding an epoxide that is subsequently detoxicated *via* formation of the corresponding diol or conjugation with glutathione.

Although some species- and sex-specific differences exist, studies for *d*-limonene and *beta*-myrcene indicate that the monoterpene hydrocarbons in this chemical category will participate in common pathways of absorption, distribution, metabolism and excretion.

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

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

The kidney changes seen in male rats administered limonene have been well characterized, and are known to be specific to the male rat and of no significance in human risk assessment.

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

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

No significant acute toxicological data identified in literature search.

Cross-reactivity is also expected between ester derivatives and their parent alcohols, as the esters will be hydrolysed by esterases in the skin.

Esters of important contact allergens that can be activated by hydrolysis in the skin are isoeugenol acetate, eugenyl acetate and geranyl acetate all of which are known to be used as fragrance ingredients.

For linalool:

Linalool gradually breaks down when in contact with oxygen, forming an oxidized by-product that may cause allergic reactions such as eczema in susceptible individuals. Between 5 and 7% of patients undergoing patch testing in Sweden were found to be allergic to the oxidized form of linalool. [

Linalool has an acute oral mammalian LD50 close to 3,000 mg/kg bw; the acute dermal toxicity is ~ 2,000 mg/kg bw. After inhalation exposure of mice and man, slight sedative effects were observed; however a dose response characteristic could not be determined. Linalool is irritating to the skin, based on animal studies, and is a mild irritant from human experience. It may be moderately irritant to the eyes at the same concentration where it produces nasal pungency. Linalool is considered not to be a sensitiser. The incidence of dermal reaction to linalool is below 1% in naive probands (not knowingly pre-sensitised) while in subjects pre-sensitised to fragrances it is up to 10%.

In a 28-day oral rat study (72.9% linalool) findings were increased liver and kidney weight, thickened liver lobes and pale areas on the kidneys and in females only hepatocellular cytoplasmic vacuolisation. Other findings were related to local irritation of the gastro-intestinal tract. Based on the effects on liver and kidney a NOAEL of 160 mg/kg bw/d (equivalent to 117 mg/kg bw/d linalool) was derived. In this study no effects on male and female gonads were found.

Linalool was not mutagenic in seven out of eight bacterial tests nor in two (one *in vitro* and one *in vivo*) mammalian tests; the one positive bacterial result is estimated to be a chance event.

Linalool (72.9%) was tested in a reproduction screening test (non-OECD). The NOAEL for maternal toxicity based on clinical signs and effects on body weight and food consumption was 500 mg/kg bw/d (equivalent to 365 mg/kg bw/d linalool). The NOAEL on reproduction toxicity and developmental toxicity is 500 mg/kg bw/d (equivalent to 365 mg/kg bw linalool), based on the decreased litter size at birth and pup morbidity/mortality thereafter.

Linalool seems not to be an immunotoxicant according to one animal study.

For terpenoid tertiary alcohols and their related esters:

Substances assigned to this category, as part of the HPV Challenge Program, possess close structural relationships, similar physicochemical properties and participate in the same pathways of metabolic detoxification and have similar toxicologic potential.

Acute Toxicity: Oral and dermal LD50 values for members of this chemical category indicate a low order of both oral and dermal toxicity. All rabbit dermal, and mouse and rat oral LD50 values exceed 2000 mg/kg with the majority of values greater than 5000 mg/kg

Repeat dose toxicity: In a safety evaluation study, a 50/50 mixture of linalool and citronellol was fed to male and female rats (number and strain not specified) in the diet. The daily intake was calculated to be 50 mg/kg bw of each. Measurements of haematology, clinical chemistry, and urinalysis at weeks 6 and 12 showed no statistically significant differences between test and control groups. Histopathology revealed no

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dose-related lesions. A slight retardation of growth was observed in males only, but was concluded by the authors to be biologically insignificant

Reproductive toxicity: Four groups of 10 virgin Crl CD rats were administered 0,250,500, or 1000 mg/kg bw of an essential oil (coriander oil) known to contain 73% linalool by mass. The test material was given by gavage once daily, 7 days prior to cohabitation, through cohabitation (maximum of 7 days), gestation, delivery, and a 4-day post-parturition period. The duration of the study was 39 days. Maternal effects reported included increased body weight and increased food consumption at 250 mg/kg/d, a non-statistically significant decrease in body weight and food consumption and decreased gestation index and decreased length of gestation at 500 mg/kg/d, and a statistically significant decrease in body weight and food consumption, statistically significant decrease in gestation index, length of gestation, and litter size at 1000 mg/kg/d. The only effect on pups was a decrease in viability of pups at the highest dose level. The authors concluded that there were no effects observed in the dams at the low dose of 250 mg/kg bw/d or in the offspring at the 250 and 500 mg/kg bw/d levels. The authors concluded that the maternal NOAEL was 250 mg/kg/d and the developmental NOAEL was 500 mg/kg/d.

Four groups of 10 virgin Crl CD rats were administered 0,375,750, or 1500 mg/kg bw of an essential oil (cardamom oil) known to contain greater than 65 % tertiary terpenoid alcohols with 5 % alpha-terpineol acetate by mass. Maternal observations included a non-statistically significant decrease in body weight gain and food consumption at 375 mg/kg/d.

Mortality, clinical signs, a statistically significant decrease in body weight gain and food consumption, and gross lesions at necropsy were seen at 750 and 1500 mg/kg/d. The only effects on pups were a reduced body weight gain in pups at 750 and 1500 mg/kg/d and increased mortality at 1500 mg/kg/d. The authors concluded that there were no significant adverse effects in the dams or offspring at the 375 mg/kg/d dose. A maternal NOEL was reported to be less than 375 mg/kg/d based on reduced body weight gain and food consumption at 375 mg/kg/d and a developmental NOAEL was reported to be 375 mg/kg/d

Developmental toxicity: A range finding study and follow-up teratology study was performed with pine oil. Pregnant Crl:CD(SD) BR rats were given 0, 50, 100, 500,750, or 1000 mg/kg/d by gavage in corn oil on days 6 to 20 of gestation. Laparotomies were performed, corpora lutea were counted, and the uterus of each rat was removed, weighed and then examined for number, placement and viability of implantations. Live foetuses were weighed, sexed and gross external alternations were identified. There were no deaths or abortions during the course of this study. Necropsy revealed no gross lesions. Maternal effects included local alopecia, decreased body weight gain and food consumption for the 3 highest dose levels. At 750 and 1000 mg/kg, average gravid uterine weight was reduced. In foetuses, decreased body weight was observed at dose levels of 100 mg/kg and above, and at dose levels of 500 and above there was a slight increase in average number of resorptions/litter.

In the follow-up teratology study, pregnant Crl:CD(SD) BR rats were given 0, 50, 600, or 1200 mg/kg/d by gavage in corn oil on days 6 to 20 of gestation. Six of the 25 rats in 1200 mg/kg dose group died and necropsies revealed that adrenal weights were significantly increased in these rats. At 1200 mg/kg/d, foetuses exhibited increased incidences of delayed ossification, delayed brain development, decreased weights, increased embryo -foetal mortality, and sunken eye bulge with associated soft and hard tissue findings, a dose that also resulted in maternal death and a low incidence of embryo-foetal death (resorption). The maternal and developmental NOEL for pine oil was greater than 50 mg/kg/d but less than 600 mg/kg/d

Genotoxicity: Mutagenicity/genotoxicity testing has been performed on six members of this chemical category, including a complete battery of in vitro genotoxicity tests using linalool. In nineteen separate in vitro tests on the mutagenicity and genotoxicity of terpenoid tertiary alcohols and related esters, all but two were negative. One of the positive results for linalool was observed in a rec assay using differences in growth rates in two strains of *Bacillus subtilis* as a measure of DNA changes. In contrast, no evidence of mutagenicity was observed in the same test at a higher concentration nor was DNA damage observed in a rat hepatocyte UDS assay. The authors of the mouse lymphoma assay which gave a weak positive result for linalool, emphasized that positive results in this assay are commonly observed for polar substances in the absence of S-9 and may be associated with changes in physiologic culture conditions (pH and osmolality).

Based on a weight of evidence evaluation of the available in vitro and in vivo mutagenicity and genotoxicity assays on terpenoid tertiary alcohols and related esters, this group of flavouring substances would not be expected to exhibit a low genotoxic potential in vivo

Metabolic fate: Based on the results of hydrolysis, the reactivity of linalool in aqueous media, and data on metabolism it is concluded that members of this chemical category exhibit similar chemical and biochemical fate. The esters are readily hydrolyzed to the corresponding alcohols, linalool and alpha-terpineol. Linalool is then partial converted to alpha-terpineol mainly under acidic conditions. Alicyclic and aliphatic tertiary alcohols are efficiently detoxicated by two principal pathways: conjugation primarily with glucuronic acid and excretion primarily in urine, and omega-oxidation to eventually yield diacids and their reduced or hydrated analogs. These polar metabolites will be efficiently excreted primarily in the urine either unchanged or as the glucuronic acid conjugates. The physicochemical and toxicological properties of these substances are consistent with their known reactivity and common metabolic fate.

Esters belonging to this category can be hydrolysed to their corresponding terpenoid alcohol and organic acid. Hydrolysis can also be catalysed by a class of esters known as carboxylesterases or B-type esterases that predominated in hepatocytes.

Esters of tertiary terpenoid alcohols are readily hydrolyzed in animals, including fish. Once hydrolysed, the resulting alcohols undergo excretion unchanged or as the glucuronic acid conjugate. To a minor extent, CYP-450 mediated oxidation at the omega or omega-1 position yields polar oxidized metabolites capable of excretion primarily in the urine. Terpenoid alcohols formed in the gastrointestinal tract are readily absorbed. During hydrolysis under acidic condition cyclisation may occur.

In humans and animals, terpenoid tertiary alcohols primarily conjugate with glucuronic acid and are excreted in the urine and feces. Terpenoid alcohols with unsaturation may also undergo allylic oxidation to form polar diol metabolites that may be excreted either free or conjugated. If the diol contains a primary alcohol function, it may undergo further oxidation to the corresponding carboxylic acid. In a minor pathway, the endocyclic alkene of alpha-terpineol is epoxidised and then hydrolyzed to yield a triol metabolite 1,2,8-trihydroxy-p-menthane which also has been reported in humans following inadvertent oral ingestion of a pine oil disinfectant containing alpha-terpineol.

Bicyclic tertiary alcohols are conjugated with glucuronic acid and excreted primarily in the urine. In rabbits the structurally related bicyclic tertiary alcohols thujyl alcohol (4-methyl-1-(1-methylethyl)bicyclo[3.1.0]hexan-3-ol) and beta-santenol (2,3,7-trimethylbicyclo[2.2.1]heptan-2-ol) are conjugated with glucuronic acid. In a metabolism study using the terpenoid tertiary alcohol trans-sobrerol, in humans, dogs, and rats, ten metabolites were isolated in urine, eight of which were characterised in humans. Two principle modes of metabolism were observed, allylic oxidation of the ring positions and alkyl substituents, and conjugation of the tertiary alcohol fractions with glucuronic acid. These metabolic patterns are common modes of converting tertiary and secondary terpenoid alcohols to polar metabolites, which are easily excreted in the urine and faeces. Menthol forms similar conjugation products in rats

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

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

Prehaptens

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

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

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themselves. Linalool and linalyl acetate are the main components of lavender oil. They autoxidise on air exposure also when present in the essential oil, and form the same oxidation products found in previous studies of the pure synthetic terpenes. Experimental sensitisation studies showed that air exposure of lavender oil increased the sensitisation potency. Patch test results in dermatitis patients showed a connection between positive reactions to oxidised linalool, linalyl acetate and lavender oil.

Prohaptens

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

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

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

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

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

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

Renal tumours induced by limonene in male rats is 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 an NOAEL for these effects. Limonene is neither genotoxic or teratogenic nor toxic to the reproductive system.

ETHANOL & N,N-DIETHYL-M-TOLUAMIDE & 2-ETHYLHEXYL BICYCLOHEPTENE DICARBOXIMIDE	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.
PIPERONYL BUTOXIDE & PERMETHRIN	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.
PERMETHRIN & LAVENDER OIL	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.

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

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

SECTION 12 Ecological information**Toxicity**

Flyaway Insecticidal Spray for Horses (Flyaway Insecticidal Spray for Horses)	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
ethanol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	96h	Algae or other aquatic plants	<0.001mg/L	4
	EC50	72h	Algae or other aquatic plants	275mg/l	2
	EC50	48h	Crustacea	>79mg/L	4
	LC50	96h	Fish	>100mg/l	2

Continued...

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	EC50	96h	Algae or other aquatic plants	<0.001mg/L	4
N,N-diethyl-m-toluamide	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1008h	Fish	0.8-2.4	7
	EC50	48h	Crustacea	55.776-99.6mg/L	4
	NOEC(ECx)	504h	Crustacea	3.7mg/l	4
	LC50	96h	Fish	70.965mg/L	4
piperonyl butoxide	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	48h	Crustacea	0.01mg/l	4
	EC50	72h	Algae or other aquatic plants	0.85mg/l	2
	EC50	48h	Crustacea	0.46-0.674mg/L	4
permethrin	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	504h	Crustacea	<0.001mg/L	4
	EC50	48h	Crustacea	<0.001mg/L	4
2-ethylhexyl bicycloheptene dicarboximide	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>1.63<2.7mg/l	2
	ErC50	72h	Algae or other aquatic plants	>4.38mg/l	2
	EC50	48h	Crustacea	1.995-4.83mg/L	4
	NOEC(ECx)	96h	Crustacea	<0.077mg/l	2
dibutyl phthalate	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1344h	Fish	3.1-21.2	7
	ErC50	72h	Algae or other aquatic plants	1.2mg/l	1
	NOEC(ECx)	72h	Algae or other aquatic plants	0.5mg/l	1
	EC50	72h	Algae or other aquatic plants	1.2mg/l	1
	EC50	48h	Crustacea	3.4mg/l	1
	LC50	96h	Fish	0.28-0.44mg/l	4
lavender oil	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
Legend:	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				

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

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

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ethanol	LOW (Half-life = 2.17 days)	LOW (Half-life = 5.08 days)
N,N-diethyl-m-toluamide	HIGH	HIGH
piperonyl butoxide	HIGH	HIGH
permethrin	HIGH	HIGH
2-ethylhexyl bicycloheptene dicarboximide	HIGH	HIGH
dibutyl phthalate	LOW (Half-life = 23 days)	LOW (Half-life = 3.08 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
ethanol	LOW (LogKOW = -0.31)
N,N-diethyl-m-toluamide	LOW (BCF = 2.4)
piperonyl butoxide	HIGH (LogKOW = 4.75)
permethrin	LOW (LogKOW = 7.4267)

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Ingredient	Bioaccumulation
2-ethylhexyl bicycloheptene dicarboximide	LOW (LogKOW = 3.7)
dibutyl phthalate	LOW (BCF = 176)

Mobility in soil

Ingredient	Mobility
ethanol	HIGH (KOC = 1)
N,N-diethyl-m-toluamide	LOW (KOC = 536.6)
piperonyl butoxide	LOW (KOC = 69.74)
permethrin	LOW (KOC = 178400)
2-ethylhexyl bicycloheptene dicarboximide	LOW (KOC = 10410)
dibutyl phthalate	LOW (KOC = 1460)



SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Recycle wherever possible. ▶ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▶ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material). ▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.
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SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	*2YE

Land transport (ADG)

UN number	1170				
UN proper shipping name	ETHANOL (ETHYL ALCOHOL) or ETHANOL SOLUTION (ETHYL ALCOHOL SOLUTION)				
Transport hazard class(es)	<table border="1"> <tr> <td>Class</td> <td>3</td> </tr> <tr> <td>Subrisk</td> <td>Not Applicable</td> </tr> </table>	Class	3	Subrisk	Not Applicable
Class	3				
Subrisk	Not Applicable				
Packing group	II				
Environmental hazard	Environmentally hazardous				
Special precautions for user	<table border="1"> <tr> <td>Special provisions</td> <td>144</td> </tr> <tr> <td>Limited quantity</td> <td>1 L</td> </tr> </table>	Special provisions	144	Limited quantity	1 L
Special provisions	144				
Limited quantity	1 L				

Air transport (ICAO-IATA / DGR)

UN number	1170						
UN proper shipping name	Ethanol or Ethanol. solution						
Transport hazard class(es)	<table border="1"> <tr> <td>ICAO/IATA Class</td> <td>3</td> </tr> <tr> <td>ICAO / IATA Subrisk</td> <td>Not Applicable</td> </tr> <tr> <td>ERG Code</td> <td>3L</td> </tr> </table>	ICAO/IATA Class	3	ICAO / IATA Subrisk	Not Applicable	ERG Code	3L
ICAO/IATA Class	3						
ICAO / IATA Subrisk	Not Applicable						
ERG Code	3L						
Packing group	II						
Environmental hazard	Environmentally hazardous						
Special precautions for user	<table border="1"> <tr> <td>Special provisions</td> <td>A3 A58 A180</td> </tr> <tr> <td>Cargo Only Packing Instructions</td> <td>364</td> </tr> <tr> <td>Cargo Only Maximum Qty / Pack</td> <td>60 L</td> </tr> </table>	Special provisions	A3 A58 A180	Cargo Only Packing Instructions	364	Cargo Only Maximum Qty / Pack	60 L
Special provisions	A3 A58 A180						
Cargo Only Packing Instructions	364						
Cargo Only Maximum Qty / Pack	60 L						

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Passenger and Cargo Packing Instructions	353
Passenger and Cargo Maximum Qty / Pack	5 L
Passenger and Cargo Limited Quantity Packing Instructions	Y341
Passenger and Cargo Limited Maximum Qty / Pack	1 L

Sea transport (IMDG-Code / GGVSee)

UN number	1170	
UN proper shipping name	ETHANOL (ETHYL ALCOHOL) or ETHANOL SOLUTION (ETHYL ALCOHOL SOLUTION)	
Transport hazard class(es)	IMDG Class	3
	IMDG Subrisk	Not Applicable
Packing group	II	
Environmental hazard	Marine Pollutant	
Special precautions for user	EMS Number	F-E, S-D
	Special provisions	144
	Limited Quantities	1 L

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

Not Applicable

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

Product name	Group
ethanol	Not Available
N,N-diethyl-m-toluamide	Not Available
piperonyl butoxide	Not Available
permethrin	Not Available
2-ethylhexyl bicycloheptene dicarboximide	Not Available
dibutyl phthalate	Not Available
lavender oil	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
ethanol	Not Available
N,N-diethyl-m-toluamide	Not Available
piperonyl butoxide	Not Available
permethrin	Not Available
2-ethylhexyl bicycloheptene dicarboximide	Not Available
dibutyl phthalate	Not Available
lavender oil	Not Available

SECTION 15 Regulatory information

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

ethanol is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

N,N-diethyl-m-toluamide is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

piperonyl butoxide is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

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

permethrin is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2

Australian Inventory of Industrial Chemicals (AIIC)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

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

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

2-ethylhexyl bicycloheptene dicarboximide is found on the following regulatory lists

Continued...

Flyaway Insecticidal Spray for Horses (Flyaway Insecticidal Spray for Horses)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australian Inventory of Industrial Chemicals (AIIC)

dibutyl phthalate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C

Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

lavender oil is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (permethrin)
Canada - NDSL	No (ethanol; N,N-diethyl-m-toluamide; piperonyl butoxide; permethrin; 2-ethylhexyl bicycloheptene dicarboximide; dibutyl phthalate; lavender oil)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (lavender oil)
Korea - KECI	No (2-ethylhexyl bicycloheptene dicarboximide)
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	No (permethrin; 2-ethylhexyl bicycloheptene dicarboximide)
Taiwan - TCSI	Yes
Mexico - INSQ	No (lavender oil)
Vietnam - NCI	Yes
Russia - FBEPH	No (2-ethylhexyl bicycloheptene dicarboximide)
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	12/10/2021
Initial Date	03/29/2010

SDS Version Summary

Version	Date of Update	Sections Updated
4.1	11/01/2019	One-off system update. NOTE: This may or may not change the GHS classification
5.1	12/10/2021	Classification change due to full database hazard calculation/update.

Other information

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

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

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

Flyaway Insecticidal Spray for Horses (Flyaway Insecticidal Spray for Horses)

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

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