Troy Repel-X Insecticidal and Repellent spray

Troy Laboratories Pty Ltd

Chemwatch Hazard Alert Code: **3**

Chemwatch: **5398-32**
Version No: **6.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **09/02/2022**
Print Date: **09/02/2022**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	Troy Repel-X Insecticidal and Repellent spray
Chemical Name	Not Applicable
Synonyms	APVMA number: 52274
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	Kills and repels flies and other biting insects for horses, dogs, cattle and pigs. To be used as directed on product label. Use according to manufacturer's directions.

Details of the supplier of the safety data sheet

Registered company name	**Troy Laboratories Pty Ltd**
Address	37 Glendenning Road Glendenning NSW 2761 Australia
Telephone	02 8808 3600
Fax	02 9677 9300
Website	www.Troylab.com.au
Email	admin@troylab.com.au

Emergency telephone number

Association / Organisation	**Troy Laboratories Pty Ltd**
Emergency telephone numbers	02 8808 3600 (Office hours (8am – 4pm, Monday to Friday))
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Flammable Liquids Category 2, Serious Eye Damage/Eye Irritation Category 2A, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
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**

Hazard statement(s)

H225	Highly flammable liquid and vapour.
H319	Causes serious eye irritation.
H412	Harmful to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

Page 1 continued...

P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P233	Keep container tightly closed.
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.
P273	Avoid release to the environment.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.
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.

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
64-17-5	>60	ethanol
134-62-3	<3	N,N-diethyl-m-toluamide
51-03-6	<1	piperonyl butoxide
8000-29-1	<1	citronella oil
8003-34-7	<1	pyrethrum
Not Available	<10	mulgofen on-870 (emulsifier) ,proprietary
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	If this product comes in contact with the eyes: ▸ Wash out immediately with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ 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. ▸ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

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).

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- 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

Advice for firefighters

Fire Fighting	▸ 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	▸ 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). Combustion products include: carbon dioxide (CO_2) nitrogen oxides (NOx) other pyrolysis products typical of burning organic material.
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	▸ Remove all ignition sources. ▸ 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	▸ 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

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Safe handling	▸ Containers, even those that have been emptied, may contain explosive vapours. ▸ Do NOT cut, drill, grind, weld or perform similar operations on or near containers. ▸ **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	▸ Store in original containers in approved flame-proof area. ▸ No smoking, naked lights, heat or ignition sources. ▸ **DO NOT** store in pits, depressions, basements 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 SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ 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. ▸ Polyethylene or polypropylene container. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ Avoid oxidising agents, acids, acid chlorides, acid anhydrides, chloroformates. ▸ Avoid strong bases. * ▸ Avoid reaction with oxidising agents, bases and strong reducing agents. ▸ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

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	pyrethrum	Pyrethrum	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

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
citronella oil	Not Available	Not Available
pyrethrum	5,000 mg/m3	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
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.*	

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Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
N,N-diethyl-m-toluamide	E	≤ 0.1 ppm
citronella 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

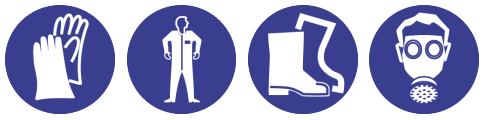
Exposure controls

Appropriate engineering controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.

- Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area.
- Work should be undertaken in an isolated system such as a "glove-box" . Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system.
- Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within.
- Open-vessel systems are prohibited.
- Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation.
- Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system.
- For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood.
- Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas).
- Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air.
- Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed.

Personal protection



Eye and face protection

- Safety glasses with side shields.
- Chemical goggles.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection

See Hand protection below

Hands/feet protection

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

NOTE:

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

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
· frequency and duration of contact,
· chemical resistance of glove material,
· glove thickness and
· dexterity
Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
· Contaminated gloves should be replaced.
As defined in ASTM F-739-96 in any application, gloves are rated as:
· Excellent when breakthrough time > 480 min
· Good when breakthrough time > 20 min
· Fair when breakthrough time < 20 min
· Poor when glove material degrades
For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.
It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation

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	efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Body protection	See Other protection below
Other protection	▸ 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:
Troy Repel-X Insecticidal and Repellent spray

Material	CPI
BUTYL	A
NEOPRENE	A
NITRILE	A
NITRILE+PVC	A
PE/EVAL/PE	A
PVC	B
NATURAL RUBBER	C
NATURAL+NEOPRENE	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)

- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Clear light yellow highly flammable liquid with citronella odour; mixes with water.

Physical state	Liquid	**Relative density (Water = 1)**	0.835
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**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	78	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	13	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	HIGHLY FLAMMABLE.	**Oxidising properties**	Not Available

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Upper Explosive Limit (%)	19	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	3.5	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (Not Available%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	▸ 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

Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.
Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.
Inhalation 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.
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.
Airborne piperidine at 2-5 ppm did not cause irritation amongst workers, but the pungent odour could be tolerated by an unacclimated individual for only a brief time.
Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination

Ingestion

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

Ingestion of ethanol may produce nausea, vomiting, gastrointestinal bleeding, abdominal pain and diarrhoea. Systemic effects:

Blood concentration:	Effects:
<1.5 g/l	Mild: Impaired visual acuity, coordination and reaction time, emotional lability
1.5-3.0 g/l	Moderate: Slurred speech, confusion, ataxia, emotional lability, perceptual and sensation disturbances possible blackout spells, and incoordination with impaired objective performance in standardised tests. Possible diplopia, flushing, tachycardia, sweating and incontinence. Bradypnoea may occur early and tachypnoea may develop in cases of metabollic acidosis, hypoglycaemia and hypokalaemia. CNS depression may progress to coma.
3-5 g/l	Severe: Cold clammy skin, hypothermia and hypotension. Atrial fibrillation and atrioventricular block have been reported. Respiratory depression may occur, respiratory failure may follow serious intoxication, aspiration of vomitus may result in pneumonitis and pulmonary oedema. Convulsions due to severe hypoglycaemia may also occur Acute hepatitis may develop.

Piperidines produces a pressor effect (blood-pressure increase) and respiratory stimulation in a manner similar to their analogue, nicotine. The piperidine alkaloids (e.g. coniine), extracted from poison hemlock, produce ataxia, salivation, convulsions and coma. Because of structural similarities with nicotine, various mammalian receptors may bind these substances. As a consequence, clinical findings may include initial stimulation (tremor, ataxia, mydriasis), nausea, vomiting, sore throat followed by cardiorespiratory depression (bradycardia, paralysis, coma) and ascending paralysis. Death may result from respiratory failure. Stimulation of nicotinic receptors primarily affects the autonomic ganglia, adrenal medulla, and the motor end-plate of striated muscle; nicotinic agonists primarily produce actions affecting the neurosmuscular junctions (producing, for example, fasciculations, weakness and paralysis) and muscarinic effects (producing postganglionic stimulation and, as a result, cardiac inhibition, vasodilation, salivation, lachrymation, bronchoconstriction and gastrointestinal stimulation). In animals, near lethal doses of piperidine may produce increased excitability to sound and touch and cause contraction of the smooth muscle and increased blood pressure. The piperidines may exert an inotropic and chronotropic action on the heart. Large doses block ganglionic conduction. Small doses cause both parasympathetic and sympathetic stimulation due to action on the ganglia. Signs of intoxication include increased blood pressure and heart rate, nausea, vomiting, salivation, laboured breathing, muscular weakness, paralysis and convulsions.

Skin Contact

The material may produce moderate skin irritation; limited evidence or practical experience suggests, that the material either:
▸ 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.
Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The

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	dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Skin exposure to the piperidine for less than 3 minutes is reported to cause severe burns. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	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. When instilled into the eyes of rabbits, piperidine caused severe injury with permanent corneal damage. Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. On the basis of epidemiological data, the material is regarded as carcinogenic to humans. There is sufficient data to establish a causal association between human exposure to the material and the development of cancer. Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Long-term exposure to ethanol may result in progressive liver damage with fibrosis or may exacerbate liver injury caused by other agents. Repeated ingestion of ethanol by pregnant women may adversely affect the central nervous system of the developing foetus, producing effects collectively described as foetal alcohol syndrome. These include mental and physical retardation, learning disturbances, motor and language deficiency, behavioural disorders and reduced head size. Consumption of ethanol (in alcoholic beverages) may be linked to the development of Type I hypersensitivities in a small number of individuals. Symptoms, which may appear immediately after consumption, include conjunctivitis, angioedema, dyspnoea, and urticarial rashes. The causative agent may be acetic acid, a metabolite (1). (1) Boehncke W.H., & H.Gall, Clinical & Experimental Allergy, 26, 1089-1091, 1996 Rats and rabbits exposed at 2.87 ppm piperidine for 4 hours/day for 4 months, showed alterations in brain electrical activity, cardiovascular system and spermatogenesis, decreased body weight gain and dystrophic changes in liver and kidney. At 0.57 ppm the animals showed decreased arterial pressure, increased permeability of skin capillaries and increased neuromuscular irritability. Normal human urine from non-smokers contains piperidine in amounts between 3 and 20 mg/litre. The substance produces a pressor effect (blood-pressure increase) and respiratory stimulation in a manner similar to its analogue, nicotine. In rats exposed to the cold significant rises in the levels of piperidine were observed in the brain and peripheral endocrine glands suggesting a neuroendocrine function. The substance is found in small amounts in some black peppers. Principal routes of exposure are usually by inhalation of mists or vapours from heated material and skin contact/absorption. 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 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). An increased incidence sperm head abnormalities and period nausea, vomiting and nasal exudate were observed in animals following chronic exposure

Troy Repel-X Insecticidal and Repellent spray	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

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]

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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/l4h[1]	
	Oral (Rat) LD50; >2000 mg/kg[1]	
citronella oil	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 4700 mg/kg[2]	Not Available
	Oral (Rat) LD50; 7200 mg/kg[2]	
pyrethrum	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 300 mg/kg[2]	Not Available
	Oral (Rat) LD50; 200 mg/kg[2]	
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

For N,N-diethyl-m-toluamide (Deet)
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.
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 .
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.
Repeated dermal application to horses produced hypersteatosis, an overactivity of the selacious glands, when the solution of Deet was 15% or higher.
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 .
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 (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.
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 .
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.
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
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).
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

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	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 . 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 . **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 Dee **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. **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. 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 fetal 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 **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 **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 The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. Reproductive effector in rats
PIPERONYL BUTOXIDE	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. Dermal (rabbit) LD50: >1880 mg/kg [Handbook of Toxicology] *Published value - probably not peer-reviewed ADI: 0.03 mg/kg
CITRONELLA 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. Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production. Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure. The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration. d-Limonene is readily absorbed by inhalation and ingestion. Dermal absorption is reported to be lower than by the inhalation route. d-Limonene is rapidly distributed to different tissues in the body, readily metabolised and eliminated primarily through the urine. Limonene exhibits low acute toxicity by all three routes in animals. Limonene is a skin irritant in both experimental animals and humans. Limited data are available on the potential to cause eye and respiratory irritation. Autooxidised products of d-limonene have the potential to be skin sensitisers. Limited data are available in humans on the potential to cause respiratory sensitisation. Autooxidation of limonene occurs readily in the presence of light and air forming a variety of oxygenated monocyclic terpenes. Risk of skin sensitisation is high in situations where contact with oxidation products of limonene occurs. Renal tumours induced by limonene in male rats is though to be sex and species specific and are not considered relevant to humans. Repeated exposure affects the amount and activity of liver enzymes, liver weight, blood cholesterol levels and bile flow in animals. Increase in liver weight is considered a physiological adaption as no toxic effects on the liver have been reported. From available data it is not possible to identify an NOAEL for these effects. Limonene is neither genotoxic or teratogenic nor toxic to the reproductive system. 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.[

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	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 inalool is below 1% in naïve 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.
PYRETHRUM	ADI: 0.04 mg/kg/day For pyrethrins The term "pyrethrin" refers to all six isomers found in pyrethrum, extracts which are obtained from the dried and ground flowers of the pyrethrum plant, Chrysanthemum cinerariaefolium. The CAS Registry No. for the mixture is 8003-34-7. The individual isomers are referred to by the common names of the acid followed by an Arabic number 1 or 2 (i.e., pyrethrin 1, pyrethrin 2, cinerin 1, cinerin 2, jasmolin 1, jasmolin 2). If the term pyrethrins is followed by a roman numerical designation, than it refers to all of the isomers of that number in the pyrethrum extract (e.g., pyrethrins I includes pyrethrin 1, cinerin 1, and jasmolin 1). Pyrethrins have low to moderate acute toxicity via the oral, dermal, and inhalation routes . Mammalian toxicity data suggest that pyrethrins are slightly toxic to small mammals on an acute oral basis (LD50 = 700 mg/kg body weight). They are a moderate eye irritant , a mild dermal irritant , and are not a skin sensitisers. **Toxic Effects** The critical toxicological effects of pyrethrins are ▸ neurobehavioral effects (tremors, labored breathing, hyperactivity, secretory signs, matted coats), following acute, short-term, and chronic exposure, with nervous system lesions observed in the rat and mouse following acute exposure; ▸ thyroid effects, following chronic exposure in the rat and dog; and ▸ liver effects, following short- and long-term exposure in the rat, dog, and mouse. Following inhalation exposure, neurobehavioral effects were observed initially, and respiratory tract lesions were observed at all dose levels. The neurobehavioral effects and the mode of action on the sodium channel are considered relevant to humans because the effects are observed in both the rat and mouse, and the mode of action affects a basic function of the nervous system that is common to all animals. **Toxic Mixtures Effects:** The U.S.EPA considered the possibility for increased toxicity due to the presence of synergists such as MGK-264 and piperonyl butoxide in pyrethrins formulations. In order for synergistic effects to be observed in humans, absorbed doses high enough to significantly affect the mixed function oxidase enzymes would be required. It is unlikely that these levels would occur based on the registered uses of pyrethrins. **Neurotoxicity:** There is a concern for neurotoxicity resulting from exposure to pyrethrins, based on ▸ tremors in female rats, decreased motor activity in male rats, and neuropathology in both sexes in a rat acute neurotoxicity study; ▸ clinical signs (excessive salivation and head arched backward) in a female rabbit following exposure during gestation; and ▸ tremors in female rats in a subchronic inhalation study. In the range-finding developmental toxicity studies in rats and rabbits, tremors/convulsions were observed in those that died during the study. In the mouse 90-day range-finding study, tremors and increased/decreased activity were observed at dose levels that also resulted in mortality. Pyrethrins are axonic poisons. **Reproductive toxicity:** In the two generation rat reproduction study, parental male systemic and reproductive toxicity were detected at 1000 ppm (65 mg/kg body weight per day) and parental female systemic toxicity was detected at 3000 ppm (196 mg/kg body weight per day). The NOAEL for parental systemic (male) and reproductive toxicity was 100 ppm (6.4 mg/kg body weight-day). **Cancer:** Pyrethrins are classified as “Suggestive Evidence of Carcinogenicity, but Not Sufficient to Assess Human Carcinogenic Potential,” based on the weight-of-the-evidence including ▸ the occurrence of benign liver tumors in female rats, ▸ no treatment-related increase in liver tumors in male rats, ▸ no treatment-related increase in tumors in either sex of mice, and ▸ no concern for mutagenicity. **Endocrine disruption:** There is evidence that pyrethrins are associated with endocrine disruption. Direct measurements of serum thyroid hormones [T3, T4, and TSH], as well as histopathological alterations in the thyroid (i.e. follicular cell hypertrophy, follicular cell hyperplasia, follicular cell adenomas and/or carcinomas) indicate there is concern regarding the potential for endocrine disruption When the appropriate screening and/or testing protocols have been developed, pyrethrins may be subject to additional screening and/or testing. **Pyrethrins and pyrethroids:** Pyrethrins are botanical insecticides that come from the pyrethrum flower, *Chrysanthemum cinerariaefolium*. Pyrethrins have limitations because of the cost of production and instability in sunlight; therefore, many synthetic pyrethrins-like compounds were developed to be more stable in sunlight and cost effective. These compounds are referred to as synthetic pyrethroids. Although all pyrethroids interact with sodium channels, there are multiple types of sodium channels and it is currently unknown whether the pyrethrins and pyrethroids have similar effects on all channels
ETHANOL & N,N-DIETHYL-M-TOLUAMIDE	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.
CITRONELLA OIL & PYRETHRUM	No significant acute toxicological data identified in literature search.

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

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

SECTION 12 Ecological information

Toxicity

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Troy Repel-X Insecticidal and Repellent spray	**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
	LC50	96h	Fish	>100mg/l	2
	EC50	72h	Algae or other aquatic plants	275mg/l	2
	EC50	48h	Crustacea	>79mg/L	4
	EC50	96h	Algae or other aquatic plants	<0.001mg/L	4
N,N-diethyl-m-toluamide	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	NOEC(ECx)	504h	Crustacea	3.7mg/l	4
	BCF	1008h	Fish	0.8-2.4	7
	LC50	96h	Fish	70.965mg/L	4
	EC50	48h	Crustacea	55.776-99.6mg/L	4
piperonyl butoxide	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	NOEC(ECx)	48h	Crustacea	0.01mg/l	4
	LC50	96h	Fish	1-3.3mg/l	4
	EC50	72h	Algae or other aquatic plants	0.85mg/l	2
	EC50	48h	Crustacea	0.46-0.674mg/L	4
citronella oil	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50(ECx)	48h	Crustacea	11.7-32.4mg/L	4
	LC50	96h	Fish	11.7-19.44mg/L	4
	EC50	48h	Crustacea	11.7-32.4mg/L	4
pyrethrum	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	NOEC(ECx)	504h	Crustacea	0.001mg/L	4
	LC50	96h	Fish	0.003-0.004mg/L	4
	EC50	48h	Crustacea	0.01-0.014mg/L	4
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*				

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
When ethanol is released into the soil it readily and quickly biodegrades but may leach into ground water; most is lost by evaporation. When released into water the material readily evaporates and is biodegradable.
Ethanol does not bioaccumulate to an appreciable extent.
The material is readily degraded by reaction with photochemically produced hydroxy radicals; release into air will result in photodegradation and wet deposition.
Environmental Fate:
TERRESTRIAL FATE: An estimated Koc value of 1 indicates that ethanol is expected to have very high mobility in soil. Volatilisation of ethanol from moist soil surfaces is expected to be an important fate process given a Henry's Law constant of 5X10-6 atm-m3/mole. The potential for volatilisation of ethanol from dry soil surfaces may exist based upon an extrapolated vapor pressure of 59.3 mmHg. Biodegradation is expected to be an important fate process for ethanol based on half-lives on the order of a few days for ethanol in sandy soil/groundwater microcosms.
AQUATIC FATE: An estimated Koc value of 1 indicates that ethanol is not expected to adsorb to suspended solids and sediment. Volatilisation from water surfaces is expected based upon a Henry's Law constant of 5X10-6 atm-m3/mole. Using this Henry's Law constant and an estimation method, volatilisation half-lives for a model river and model lake are 3 and 39 days, respectively.An estimated BCF= 3, from a log Kow of -0.31 suggests bioconcentration in aquatic organisms is low. Hydrolysis and photolysis in sunlit surface waters is not expected to be an important environmental fate process for ethanol since this compound lacks functional groups that hydrolyse or absorb light under environmentally relevant conditions. Ethanol was degraded with half-lives on the order of a few days in aquatic studies conducted using microcosms constructed with a low organic sandy soil and groundwater, indicating it is unlikely to be persistent in aquatic environments(8).
ATMOSPHERIC FATE: Ethanol, which has an extrapolated vapor pressure of 59.3 mm Hg at 25 deg C, is expected to exist solely as a vapor in the ambient atmosphere. Vapour-phase ethanol is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 5 days, calculated from its rate constant of 3.3X10-12 m3/molecule-sec at 25 deg C.
Ecotoxicity:
log Kow: -0.31- -0.32
Half-life (hr) air: 144
Half-life (hr) H2O surface water: 144
Henry's atm m3 /mol: 6.29E-06
BOD 5 if unstated: 0.93-1.67,63%
COD: 1.99-2.11,97%
ThOD: 2.1
For piperidine :
log Kow : 0.84
Half-life (hr) air : 81.6
Henry's atm m3 /mol: 4.45E-06
Bioaccumulation : not sig
Environmental fate;
Terrestrial fate:: An estimated Koc value of 68 determined using a log Kow of 0.84, indicates that piperidine is expected to have high mobility in soil. However, the pKa of piperidine is 11.28, indicating that this compound will primarily exist in the cation form in the environment and cations generally adsorb more strongly to soils containing organic carbon and clay

Continued...

than their neutral counterparts. Cations do not volatilise from moist soil surfaces. Piperidine is expected to volatilise from dry soil surfaces based upon a vapor pressure of 32.1 mm Hg. A 66.9 % theoretical BOD in 2 weeks using an activated sludge inoculum and the Japanese MITI test indicates that biodegradation may be an important environmental fate process in soil.
Aquatic fate:: Based on the estimated Koc value of piperidine is not expected to adsorb to suspended solids and sediment. The pKa indicates piperidine will exist almost entirely in the cation form at pH values of 5 to 9 and therefore volatilisation from water surfaces is not expected to be an important fate process. An estimated BCF of 3 (using the log Kow) suggests the potential for bioconcentration in aquatic organisms is low. Piperidine was found to degrade anaerobically via denitrification in 12-15 days in microbial consortia from freshwater sediments, estuarine sediments and activated sludge.
Atmospheric fate: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere, piperidine, which has a vapor pressure of 32.1 mm Hg at 25 deg C, is expected to exist solely as a vapor in the ambient atmosphere. Vapor-phase piperidine is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 4 hours, calculated from its rate constant of 8.9 x10-11 cu cm/molecule-sec at 25 deg C(that was derived using a structure estimation method
Ecotoxicity:
Daphnia magna LC50 948 h): 8.234 mg/l
Fish LC50 (96 h): fathead minnow (Pimephales promelas) 129.6 mg/l
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

Bioaccumulative potential

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

Mobility in soil

Ingredient	Mobility
ethanol	HIGH (KOC = 1)
N,N-diethyl-m-toluamide	LOW (KOC = 536.6)
piperonyl butoxide	LOW (KOC = 69.74)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible. ▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▸ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material). ▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	NO

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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)	Class: 3 Subrisk: Not Applicable
Packing group	II
Environmental hazard	Not Applicable
Special precautions for user	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)	ICAO/IATA Class: 3 ICAO / IATA Subrisk: Not Applicable ERG Code: 3L
Packing group	II
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: A3 A58 A180 Cargo Only Packing Instructions: 364 Cargo Only Maximum Qty / Pack: 60 L 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	Not Applicable
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
citronella oil	Not Available
pyrethrum	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
citronella oil	Not Available
pyrethrum	Not Available

Continued...

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

citronella oil is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

pyrethrum 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 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2
Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (ethanol; N,N-diethyl-m-toluamide; piperonyl butoxide; citronella oil; pyrethrum)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (citronella oil)
Japan - ENCS	No (citronella oil; pyrethrum)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	No (pyrethrum)
Taiwan - TCSI	Yes
Mexico - INSQ	No (citronella oil)
Vietnam - NCI	Yes
Russia - FBEPH	No (citronella oil)
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	09/02/2022
Initial Date	30/04/2020

SDS Version Summary

Version	Date of Update	Sections Updated
5.1	10/12/2021	Classification change due to full database hazard calculation/update.
6.1	09/02/2022	Acute Health (eye), Acute Health (inhaled), Acute Health (skin), Acute Health (swallowed), Advice to Doctor, Chronic Health, Classification, Disposal, Engineering Control, Environmental, First Aid (inhaled), Handling Procedure, Ingredients, Personal Protection (other), Personal Protection (hands/feet), Storage (storage incompatibility), Storage (suitable container), Use

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

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ES: Exposure Standard
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index
AIIC: Australian Inventory of Industrial Chemicals
DSL: Domestic Substances List
NDSL: Non-Domestic Substances List
IECSC: Inventory of Existing Chemical Substance in China
EINECS: European INventory of Existing Commercial chemical Substances
ELINCS: European List of Notified Chemical Substances
NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory
KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals
PICCS: Philippine Inventory of Chemicals and Chemical Substances
TSCA: Toxic Substances Control Act
TCSI: Taiwan Chemical Substance Inventory
INSQ: Inventario Nacional de Sustancias Químicas
NCI: National Chemical Inventory
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances