Avenge + Fly Blowfly Strike Prevention and Lousicide for Sheep Spray-on Pour-on

Troy Laboratories Pty Ltd

Chemwatch Hazard Alert Code: **3**

Chemwatch: **5445-38**
Version No: **3.1.1.1**
Safety Data Sheet according to WHS and ADG requirements

Issue Date: **23/12/2020**
Print Date: **08/04/2021**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	Avenge + Fly Blowfly Strike Prevention and Lousicide for Sheep Spray-on Pour-on
Chemical Name	Not Applicable
Synonyms	Avenge + Fly; APVMA number 62598
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains imidacloprid)
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	To be used as directed on product label.

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	1800 033 111 (24 hours)

SECTION 2 Hazards identification

Classification of the substance or mixture

COMBUSTIBLE LIQUID, regulated for storage purposes only

Poisons Schedule	S5
Classification [1]	Flammable Liquid Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Specific target organ toxicity - single exposure Category 3 (narcotic effects), Acute Aquatic Hazard Category 2, Chronic Aquatic Hazard Category 2, Reproductive Toxicity Category 1B
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)

AUH019	May form explosive peroxides.
H227	Combustible liquid.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.
H411	Toxic to aquatic life with long lasting effects.
H360Df	May damage the unborn child. Suspected of damaging fertility.

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.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection/hearing protection/...
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.

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.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/... if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.
P302+P352	IF ON SKIN: Wash with plenty of water.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

P501	Dispose of contents/container 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
34590-94-8	>60	dipropylene glycol monomethyl ether

CAS No	%[weight]	Name
872-50-4	10-30	N-methyl-2-pyrrolidone
138261-41-3	1-10	imidacloprid
Not Available	balance	Ingredients determined not to be hazardous

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 neonicotinoid intoxications:

No specific antidotes are known.

It is important to support respiration if signs of paralysis appear and to monitor blood pressure and pulse rate, since bradycardia and hypotonia are possible.

Since the compounds **do NOT** inhibit cholinesterase activity, treatment with a reactivating oxime is not indicated.

Symptoms of poisoning may be mediated by either stimulation or inhibition of nicotinic activity, or by other possible mechanisms. Therefore treatment with a nicotinic antagonist might be either ineffective or contraindicated.

Handbook of Neurotoxicology; Vol 1; Ed Edward J. Massaro, Humana Press, 2001

This compound does not inhibit cholinesterase but toxic symptoms may resemble cholinergic stimulation.

Treat symptomatically.

SECTION 5 Firefighting measures

Extinguishing media

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. Wear full body protective clothing with breathing apparatus.

	Prevent, by any means available, spillage from entering drains or water course. Use water delivered as a fine spray to control fire and cool adjacent area. Avoid spraying water onto liquid pools. **DO NOT** approach containers suspected to be hot. Cool fire exposed containers with water spray from a protected location. If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	Combustible. Slight fire hazard when exposed to heat or flame. Heating may cause expansion or decomposition leading to violent rupture of containers. On combustion, may emit toxic fumes of carbon monoxide (CO). May emit acrid smoke. Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO_2) nitrogen oxides (NOx) other pyrolysis products typical of burning organic material.
HAZCHEM	•3Z

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	Environmental hazard - contain spillage. Clean up all spills immediately. Avoid breathing vapours and contact with skin and eyes. Control personal contact with the substance, by using protective equipment. Contain and absorb spill with sand, earth, inert material or vermiculite. Wipe up. Place in a suitable, labelled container for waste disposal.
Major Spills	Environmental hazard - contain spillage. Moderate hazard. Clear area of personnel and move upwind. Alert Fire Brigade and tell them location and nature of hazard. Wear breathing apparatus plus protective gloves. Prevent, by any means available, spillage from entering drains or water course. No smoking, naked lights or ignition sources. Increase ventilation. Stop leak if safe to do so. Contain spill with sand, earth or vermiculite. 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	**DO NOT** allow clothing wet with material to stay in contact with skin The tendency of many ethers to form explosive peroxides is well documented. Ethers lacking non-methyl hydrogen atoms adjacent to the ether link are thought to be relatively safe **DO NOT** concentrate by evaporation, or evaporate extracts to dryness, as residues may contain explosive peroxides with DETONATION potential. Any static discharge is also a source of hazard. Before any distillation process remove trace peroxides by shaking with excess 5% aqueous ferrous sulfate solution or by percolation through a column of activated alumina. Distillation results in uninhibited ether distillate with considerably increased hazard because of risk of peroxide formation on storage.

	Add inhibitor to any distillate as required. When solvents have been freed from peroxides by percolation through columns of activated alumina, the absorbed peroxides must promptly be desorbed by treatment with polar solvents such as methanol or water, which should then be disposed of safely. The substance accumulates peroxides which may become hazardous only if it evaporates or is distilled or otherwise treated to concentrate the peroxides. The substance may concentrate around the container opening for example. Purchases of peroxidisable chemicals should be restricted to ensure that the chemical is used completely before it can become peroxidised. A responsible person should maintain an inventory of peroxidisable chemicals or annotate the general chemical inventory to indicate which chemicals are subject to peroxidation. An expiration date should be determined. The chemical should either be treated to remove peroxides or disposed of before this date. The person or laboratory receiving the chemical should record a receipt date on the bottle. The individual opening the container should add an opening date. Unopened containers received from the supplier should be safe to store for 18 months. Opened containers should not be stored for more than 12 months. 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 or ignition sources. Avoid contact with incompatible materials. When handling, **DO NOT** eat, drink or smoke. Keep containers securely sealed when not in use. Avoid physical damage to containers. Always wash hands with soap and water after handling. Work clothes should be laundered separately. Use good occupational work practice. Observe manufacturer's storage and handling recommendations contained within this SDS. Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	Consider storage under inert gas. Store in original containers. Keep containers securely sealed. Store in a cool, dry, well-ventilated area. Store away from incompatible materials and foodstuff containers. Protect containers against physical damage and check regularly for leaks. Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	Metal can or drum Packaging as recommended by manufacturer. Check all containers are clearly labelled and free from leaks.
Storage incompatibility	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	dipropylene glycol monomethyl ether	(2-Methoxymethylethoxy) propanol	50 ppm / 308 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	N-methyl-2-pyrrolidone	1-Methyl-2-pyrrolidone	25 ppm / 103 mg/m3	309 mg/m3 / 75 ppm	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
dipropylene glycol monomethyl ether	150 ppm	1700* ppm	9900** ppm
N-methyl-2-pyrrolidone	30 ppm	32 ppm	190 ppm

Ingredient	Original IDLH	Revised IDLH

Ingredient	Original IDLH	Revised IDLH
dipropylene glycol monomethyl ether	600 ppm	Not Available
N-methyl-2-pyrrolidone	Not Available	Not Available
imidacloprid	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
imidacloprid	E	≤ 0.01 mg/m³
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.

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.
An approved self contained breathing apparatus (SCBA) may be required in some situations.
Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

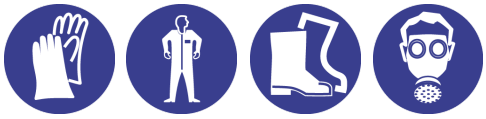
Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Personal 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 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. P.V.C apron. Barrier cream. Skin cleansing cream. Eye wash unit.

Recommended material(s)

GLOVE SELECTION INDEX

Respiratory protection

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:
Avenge + Fly Blowfly Strike Prevention and Lousicide for Sheep Spray-on Pour-on

Material	CPI
BUTYL	A
PE/EVAL/PE	A
NATURAL RUBBER	B
PVA	B

* 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.

Type AK-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 5 x ES	AK-AUS / Class 1 P2	-	AK-PAPR-AUS / Class 1 P2
up to 25 x ES	Air-line*	AK-2 P2	AK-PAPR-2 P2
up to 50 x ES	-	AK-3 P2	-
50+ x ES	-	Air-line**	-

^ - Full-face
A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(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 blue liquid; mixes with water.

Physical state	Liquid	**Relative density (Agua= 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**	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)	88	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Combustible.	**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
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	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 high vapour concentrations of N-methyl-2-pyrrolidone (NMP) may produce mucous membrane irritation, headache, giddiness, mental confusion and nausea. Fatalities were not recorded following inhalation of 180-200 mg/m3 for 2 hours by mice and following a 6 hour exposure to saturated vapours by rats. Laboratory animals exposed to concentrations of 50 ppm for 8 hours daily for 20 days or 370 ppm for 6 hours daily for 10 days showed no gross or histopathological abnormalities Inhalation hazard is increased at higher temperatures. Not normally a hazard due to non-volatile nature of product In fog-laden atmospheres rats exposed to dipropylene glycol monomethyl ether DPME, for 7 hours, exhibited a mild narcosis from which they rapidly recovered. Controlled human exposures to vapour produced CNS impairment at 1000 ppm in one subject Acute effects from inhalation of high vapour concentrations may be chest and nasal irritation with coughing, sneezing, headache and even nausea.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Dipropylene monomethyl ether (DPME) produces marked central nervous system depression in rats. Lethal doses produced respiratory failure within 48 hours. The insecticidal activity of neonicotinoids (nitromethylene, chlorothiazoles, chlorpyridines, spinosads) is attributed to binding of the molecule to nicotinic acetylcholine receptors (nAChR) located in the insect central nervous system (CNS). This group of insecticides have much lower activity in vertebrate tissues due to differences in binding to nAChR subtypes. Poor penetration of the blood-brain barrier is an additional factor that acts to reduce the toxicity of neonicotinoids in vertebrates. Nevertheless at relatively high levels of exposure, these insecticides are neuroactive and produce neurotoxic effects. The principal effect may involve stimulation or inhibition. Tremors have occurred in mice treated with representative compounds. These compounds produce a variety of neurotoxic signs following acute exposure, with complete recovery within several hours or a few days following treatment. The most consistent finding at lower doses is evidence of decreased activity. At higher doses, tremors, impaired pupillary function (either dilated or pin-point pupils) and hypothermia are the most common effects. Finally, at near lethal doses, neurotoxic effects are assorted and include motor incoordination, (uncoordinated gait or impaired aerial righting), autonomic signs (lachrymation, urine staining) and CNS depression (marked decreased motor activity and decreased response to stimuli). Deaths associated with treatment occurred within 4-24 hours. There was no evidence of neuropathology associated with these compounds. Certain findings (e.g tremors, impaired pupillary function and hypothermia) that are evident at sublethal doses are likely associated with nicotinic stimulation or represent nonspecific toxic effects. Sustained dietary exposure to relatively low doses produces little or no evidence of neurotoxicity. These results suggest that cumulative toxicity is not a concern with neonicotinoid insecticides. This outcome is consistent with their rapid metabolism and excretion in rats. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.
Skin Contact	The material produces mild skin irritation; evidence exists, or practical experience predicts, that the material either produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant, but mild, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period.

	Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. Prolonged contact with N-methyl-2-pyrrolidone (NMP) reportedly causes severe dermatitis with redness, cracking, swelling, blisters and oedema. An instance of severe skin irritation after a few days work with NMP shows latex rubber gloves as giving insufficient protection. A review article casts doubts on reliability of animal single patch tests, i.e Draize tests. [Irritant Cutaneous Reaction to NMP, Contact Dermatitis 27: 148-150, 1992] 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. Continuous contact with DPME of the skin of numerous rabbits for 90 days caused only slight scaliness. Patch tests on human volunteers produced no evidence of primary irritation or sensitisation. Sufficient absorption did occur in rabbits to produce narcosis and high doses proved lethal. Pathology revealed gastric distension, occasional gastric irritation and granular and hydropic changes to kidneys Absorption by skin may readily exceed vapour inhalation exposure. Symptoms for skin absorption are the same as for inhalation.
Eye	Direct contact with the liquid N-methyl-2-pyrrolidone (NMP) may produce painful burning or stinging of the eyes and lids, watering and inflammation of the conjunctiva and temporary corneal clouding. When one drop of undiluted dipropylene glycol monomethyl ether (DPME) was placed in a rabbits eyes on each of five consecutive days, a mild transitory irritation of the conjunctival membranes occurred. Fluorescein staining revealed no corneal damage. Direct contact of the substance can produce painful irritation (blepharoconjunctivitis, slight keratitis, and an increase in intra-ocular pressure) which, is however rapidly reversible. Persistent eye lesions do not develop Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. 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: - 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. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects. Studies with some glycol ethers (principally the monoethylene glycols) and their esters indicate reproductive changes, testicular atrophy, infertility and kidney function changes. The metabolic acetic acid derivatives of glycol ethers (alkoxyacetic acids), not the ether itself, have been found to be the proximal reproductive toxin in animals. The potency of these metabolites decreases significantly as the chain length of the ether increases. Consequently glycol ethers with longer substituents (e.g diethylene glycols, triethylene glycols) have not generally been associated with reproductive effects. One of the most sensitive indicators of toxic effects observed from many of the glycol ethers is an increase in the erythrocytic osmotic fragility in rats Which produces haemolytic anaemia). This appears to be related to the development of haemoglobinuria (blood in the urine) at higher exposure levels or as a result of chronic exposure. Glycol ethers based on propylene oxides, propylene glycol ethers, dipropylene glycol ethers and tripropylene glycol ethers are mainly available, commercially, as alpha-isomers (because of thermodynamic considerations); these are incapable of forming alkoxyacetic or alkoxypropionic acids as metabolites and therefore do not produce erythrocyte fragility unless contaminated by ethylene glycol ethers or to a significant degree by the beta-isomer . beta-Isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). The teratogenic potential, subchronic and long term inhalation toxicity of N-methyl-2-pyrrolidone (NMP has been studied in rats. No evidence of nephrotoxicity was seen. No carcinogenic effects were observed. Very high doses are embryotoxic to rats and mice. Reproductive effects have been reported in animals. Rats, rabbits, guinea pigs and monkeys exposed to DPME, 7 hr/day, 5 days a week for periods of 6-8 months to saturated atmospheres (300 ppm), exhibited little effect. Narcotic effects were produced in rats. This concentration of vapour is objectionable to human beings. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals.

Avenge + Fly Blowfly Strike Prevention and Lousicide for Sheep Spray-on Pour-on		
	TOXICITY	**IRRITATION**
	Not Available	Not Available

dipropylene glycol monomethyl ether	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 10.526 mg/kg[1]	Eye (human): 8 mg - mild
	Oral(Rat) LD50; 5.684 mg/kg[1]	Eye (rabbit): 500 mg/24hr - mild
		Skin (rabbit): 238 mg - mild
		Skin (rabbit): 500 mg (open)-mild
N-methyl-2-pyrrolidone	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 20004000 mg/kg[2]	Eye (rabbit): 100 mg - moderate
	Inhalation(Rat) LC50; 3.18.8 mg/l4h[2]	
	Oral(Rabbit) LD50; ~3500 mg/kg[2]	
imidacloprid	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >5000 mg/kg[2]	Eye (rabbit): non-irritating *
	Inhalation(Rat) LC50; >0.069 mg/L4h[2]	Skin (rabbit): non-irritating *
	Oral(Mouse) LD50; 98 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*	

DIPROPYLENE GLYCOL MONOMETHYL ETHER	for propylene glycol ethers (PGEs): Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM). Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids. Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product. Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body. As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces. As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m3 for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m3. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m3), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating None are skin sensitisers. In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested). Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m3 (600 ppm) for PnB and 2,010 mg/m3 (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m3 (43 ppm).

	In this study, the highest tested TPM concentration, 1010 mg/m3 (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members. One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m3) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m3). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m3), with decreased body weights occurring at 3000 ppm (11058 mg/m3). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health. In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity. The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice. The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. 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.
N-METHYL-2-PYRROLIDONE	for N-methyl-2-pyrrolidone (NMP): **Acute toxicity:** In rats, NMP is absorbed rapidly after inhalation, oral, and dermal administration, distributed throughout the organism, and eliminated mainly by hydroxylation to polar compounds, which are excreted via urine. About 80% of the administered dose is excreted as NMP and NMP metabolites within 24 h. A probably dose-dependent yellow coloration of the urine in rodents is observed. The major metabolite is 5-hydroxy-*N*-methyl-2-pyrrolidone. Studies in humans show comparable results. Dermal penetration through human skin has been shown to be very rapid. NMP is rapidly biotransformed by hydroxylation to 5-hydroxy-*N*-methyl-2-pyrrolidone, which is further oxidized to *N*-methylsuccinimide; this intermediate is further hydroxylated to 2-hydroxy-*N*-methylsuccinimide. These metabolites are all colourless. The excreted amounts of NMP metabolites in the urine after inhalation or oral intake represented about 100% and 65% of the administered doses, respectively. NMP has a low potential for skin irritation and a moderate potential for eye irritation in rabbits. Repeated daily doses of 450 mg/kg body weight administered to the skin caused painful and severe haemorrhage and eschar formation in rabbits. These adverse effects have not been seen in workers occupationally exposed to pure NMP, but they have been observed after dermal exposure to NMP used in cleaning processes. No sensitisation potential has been observed. In acute toxicity studies in rodents, NMP showed low toxicity. Uptake of oral, dermal, or inhaled acutely toxic doses causes functional disturbances and depressions in the central nervous system. Local irritation effects were observed in the respiratory tract when NMP was inhaled and in the pyloric and gastrointestinal tracts after oral administration. In humans, there was no irritative effect in the respiratory system after an 8-h exposure to 50 mg/m3. **Repeat dose toxicity:** There is no clear toxicity profile of NMP after multiple administration. In a 28-day dietary study in rats, a compound-related decrease in body weight gain was observed in males at 1234 mg/kg body weight and in females at 2268 mg/kg body weight. Testicular degeneration and atrophy in males and thymic atrophy in females were observed at these dose levels. The no-observed-adverse-effect level (NOAEL) was 429 mg/kg body weight in males and 1548 mg/kg body weight in females. In a 28-day intubation study in rats, a dose-dependent increase in relative liver and kidney weights and a decrease in lymphocyte count in both sexes were observed at 1028 mg/kg body weight. The NOAEL in this study was 514 mg/kg body weight. In another rat study, daily dietary intake for 90 days caused decreased body weights at doses of 433 and 565 mg/kg body weight in males and females, respectively. There were also neurobehavioural effects at these dose levels. The NOAELs in males and females were 169 and 217 mg/kg body weight, respectively. The toxicity profile after exposure to airborne NMP depends strongly on the ratio of vapour to aerosol and on the area of exposure (i.e., head-only or whole-body exposure). Because of higher skin absorption for the aerosol, uptake is higher in animals exposed to aerosol than in those exposed to vapour at similar concentrations. Studies in female rats exposed head only to 1000 mg/m3 showed only minor nasal irritation, but massive mortality and severe effects on major organs were observed when the females were whole-body exposed to the same concentration of coarse droplets at high relative humidity. Several studies in rats following repeated exposure to NMP at concentrations between 100 and 1000 mg/m3 have shown systemic toxicity effects at the lower dose levels. In most of the studies, the effects were not observed after a 4-week observation period. In rats, exposure to 3000 mg NMP/m3 (head only) for 6 h/day, 5 days/week, for 13 weeks caused a decrease in body weight gain, an increase in erythrocytes, haemoglobin, haematocrit, and mean corpuscular volume, decreased absolute testis weight, and cell loss in the germinal epithelium of the testes. The NOAEL was 500 mg/m3. There are no data in humans after repeated-dose exposure. **Carcinogenicity:** NMP did not show any clear evidence for carcinogenicity in rats exposed to concentrations up to 400 mg/m3 in a long-term inhalation study. **Genotoxicity:** The mutagenic potential of NMP is weak. Only a slight increase in the number of revertants was observed when tested in a *Salmonella* assay with base-pair substitution strains. NMP has been shown to induce aneuploidy in yeast *Saccharomyces cerevisiae* cells. No investigations regarding mutagenicity in humans were available. **Reproductive toxicity:** In a two-generation reproduction study in rats, whole-body exposure of both males and females to 478 mg/m3 of NMP vapour for 6 h/day, 7 days/week, for a minimum of 100 days (pre-mating, mating, gestation, and lactation periods)

	resulted in a 7% decrease in fetal weight in the F1 offspring. A 4-11% transient, non-dose-dependent decrease was observed in the average pup weight at all exposure levels tested (41, 206, and 478 mg/m3). **Developmental toxicity:** When NMP was administered dermally, developmental toxicity was registered in rats at 750 mg/kg body weight. The observed effects were increased preimplantation losses, decreased fetal weights, and delayed ossification. The NOAEL for both developmental effects and maternal toxicity (decreased body weight gain) was 237 mg/kg body weight. Inhalation studies in rats (whole-body exposure) demonstrated developmental toxicity as increased preimplantation loss without significant effect on implantation rate or number of live fetuses at 680 mg/m3 and behavioural developmental toxicity at 622 mg/m3. In an inhalation study (whole-body exposure), the NOAEL for maternal effects was 100 mg/m3, and the NOAEL for developmental effects was 360 mg/m3. A tolerable inhalation concentration, 0.3 mg/m3, based on mortality and organ damage, is expected to be protective against any possible reproductive toxicity. Similarly, an oral tolerable intake of 0.6 mg/kg body weight per day, based on a 90-day study, is expected to provide adequate protection against possible reproductive effects. Because of non-existent data on the exposure of the general population and very limited information on occupational exposure, no meaningful risk characterisation can be performed A substance (or part of a group of chemical substances) of very high concern (SVHC) - or product containing an SVHC: It is proposed that use within the European Union be subject to authorisation under the REACH Regulation.Indeed, listing of a substance as an SVHC by the European Chemicals Agency (ECHA) is the first step in the procedure for authorisation or restriction of use of a chemical. The criteria are given in article 57 of the REACH Regulation. A substance may be proposed as an SVHC if it meets one or more of the following criteria: it is carcinogenic *; it is mutagenic *; it is toxic for reproduction *; it is persistent, bioaccumulative and toxic (PBT substances); it is very persistent and very bioaccumulative (vPvB substances); there is "scientific evidence of probable serious effects to human health or the environment which give rise to an equivalent level of concern"; such substances are identified on a case-by-case basis. * Collectively described as CMR substances The "equivalent concern" criterion is significant because it is this classification which allows substances which are, for example, neurotoxic, endocrine-disrupting or otherwise present an unanticipated environmental health risk to be regulated under REACH] Simply because a substance meets one or more of the criteria does not necessarily mean that it will be proposed as an SVHC. Many such substances are already subject to restrictions on their use within the European Union, such as those in Annex XVII of the REACH Regulation SVHCs are substances for which the current restrictions on use (where these exist) might be insufficient. There are three priority groups for assessment: PBT substances and vPvB substances; substances which are widely dispersed during use; substances which are used in large quantities.
IMIDACLOPRID	***[* The Pesticides Manual, Incorporating The Agrochemicals Handbook, 10th Edition, Editor Clive Tomlin, 1994, British Crop Protection Council]*** ADI 0.057 mg/kg bw. *
DIPROPYLENE GLYCOL MONOMETHYL ETHER & N-METHYL-2-PYRROLIDONE	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

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

Avenge + Fly Blowfly Strike Prevention and Lousicide for Sheep Spray-on Pour-on	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

dipropylene glycol monomethyl ether	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72	Algae or other aquatic plants	>969mg/l	2
	NOEC(ECx)	528	Crustacea	>=0.5mg/l	2
	EC50	96	Algae or other aquatic plants	>969mg/l	2
	EC50	48	Crustacea	1930mg/l	2
	LC50	96	Fish	>1000mg/l	2

N-methyl-2-pyrrolidone	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48	Crustacea	ca.4897mg/l	1
	LC50	96	Fish	2.936-3.873mg/L	4
	EC50	72	Algae or other aquatic plants	>500mg/l	1
	NOEC(ECx)	504	Crustacea	12.5mg/l	2

imidacloprid	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48	Crustacea	0.784-1.182mg/L	4
	LC50	96	Fish	>0.868mg/L	4
	EC50	72	Algae or other aquatic plants	>10mg/l	2
	NOEC(ECx)	144	Crustacea	<0.001mg/l	4

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

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
dipropylene glycol monomethyl ether	HIGH	HIGH
N-methyl-2-pyrrolidone	LOW	LOW
imidacloprid	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
dipropylene glycol monomethyl ether	LOW (BCF = 100)
N-methyl-2-pyrrolidone	LOW (BCF = 0.16)
imidacloprid	LOW (LogKOW = 1.4496)

Mobility in soil

Ingredient	Mobility
dipropylene glycol monomethyl ether	LOW (KOC = 10)
N-methyl-2-pyrrolidone	LOW (KOC = 20.94)
imidacloprid	LOW (KOC = 5048)

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. **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 or consult manufacturer for recycling options. Consult State Land Waste Authority for disposal. Bury or incinerate residue at an approved site. Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	•3Z

Land transport (ADG)

UN number	3082
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains imidacloprid)
Transport hazard class(es)	Class: 9 Subrisk: Not Applicable
Packing group	III
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: 274 331 335 375 AU01 Limited quantity: 5 L

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082
are not subject to this Code when transported by road or rail in;
(a) packagings;
(b) IBCs; or
(c) any other receptacle not exceeding 500 kg(L).
- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

Air transport (ICAO-IATA / DGR)

UN number	3082
UN proper shipping name	Environmentally hazardous substance, liquid, n.o.s. * (contains imidacloprid)
Transport hazard class(es)	ICAO/IATA Class: 9 ICAO / IATA Subrisk: Not Applicable ERG Code: 9L
Packing group	III
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: A97 A158 A197 A215

Cargo Only Packing Instructions	964
Cargo Only Maximum Qty / Pack	450 L
Passenger and Cargo Packing Instructions	964
Passenger and Cargo Maximum Qty / Pack	450 L
Passenger and Cargo Limited Quantity Packing Instructions	Y964
Passenger and Cargo Limited Maximum Qty / Pack	30 kg G

Sea transport (IMDG-Code / GGVSee)

UN number	3082
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains imidacloprid)
Transport hazard class(es)	IMDG Class: 9 IMDG Subrisk: Not Applicable
Packing group	III
Environmental hazard	Marine Pollutant
Special precautions for user	EMS Number: F-A , S-F Special provisions: 274 335 969 Limited Quantities: 5 L

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

Not Applicable

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

Product name	Group
dipropylene glycol monomethyl ether	Not Available
N-methyl-2-pyrrolidone	Not Available
imidacloprid	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
dipropylene glycol monomethyl ether	Not Available
N-methyl-2-pyrrolidone	Not Available
imidacloprid	Not Available

SECTION 15 Regulatory information

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

dipropylene glycol monomethyl ether is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

N-methyl-2-pyrrolidone 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 6
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

imidacloprid is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (imidacloprid)
Canada - DSL	No (imidacloprid)
Canada - NDSL	No (dipropylene glycol monomethyl ether; N-methyl-2-pyrrolidone; imidacloprid)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	No (imidacloprid)
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	No (imidacloprid)
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 Other information

Revision Date	23/12/2020
Initial Date	19/12/2020

SDS Version Summary

Version	Issue Date	Sections Updated
3.1.1.1	23/12/2020	Classification, Ingredients

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
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