

Flukamec Plus Selenium Anthelmintic for Sheep

Virbac (Australia) Pty Limited

Chemwatch Hazard Alert Code: 2

Chemwatch: 62-7955

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Safety Data Sheet according to WHS and ADG requirements

L.GHS.AUS.EN

SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	Flukamec Plus Selenium Anthelmintic for Sheep
Synonyms	APVMA No: 54108
Other means of identification	Not Available

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

Relevant identified uses	Use according to manufacturer's directions.
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Details of the supplier of the safety data sheet

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

Emergency telephone number

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

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

Poisons Schedule	S6
Classification ^[1]	Acute Toxicity (Oral) Category 4, Acute Toxicity (Inhalation) Category 4, Skin Sensitizer Category 1, Carcinogenicity Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HSIS; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
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SIGNAL WORD **WARNING**

Hazard statement(s)

H302	Harmful if swallowed.
H332	Harmful if inhaled.
H317	May cause an allergic skin reaction.
H351	Suspected of causing cancer.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P281	Use personal protective equipment as required.
P261	Avoid breathing mist/vapours/spray.
P270	Do not eat, drink or smoke when using this product.

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P272	Contaminated work clothing should not be allowed out of the workplace.
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Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/attention.
P363	Wash contaminated clothing before reuse.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P301+P312	IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P330	Rinse mouth.

Precautionary statement(s) Storage

P405	Store locked up.
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Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.
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SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
68786-66-3	<5	triclabendazole
100-51-6	<1	benzyl alcohol
71751-41-2	<1	abamectin
13410-01-0	<1	sodium selenate, anhydrous
	balance	Ingredients determined not to be hazardous

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prosthesis such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor.
Ingestion	<ul style="list-style-type: none"> ▶ IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY. ▶ For advice, contact a Poisons Information Centre or a doctor. ▶ Urgent hospital treatment is likely to be needed. ▶ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. ▶ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist. ▶ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS. <p>Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:</p> <ul style="list-style-type: none"> ▶ INDUCE vomiting with fingers down the back of the throat, ONLY IF CONSCIOUS. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. <p>NOTE: Wear a protective glove when inducing vomiting by mechanical means.</p>

Indication of any immediate medical attention and special treatment needed

For abamectin (avermectins):

Toxicity following accidental ingestion may be minimised by emesis-induction within one half hour of exposure. Since abamectin is thought to bind to glutamate-gated chloride ion channels, it is probably wise to avoid drugs that also interact with other ligand-gated chloride channels, including those that enhance GABA activity in patients with potentially toxic abamectin exposure. Avoid drugs that enhance GABA activity (barbiturate, benzodiazepines, valproic acid, etc.).

Following exposures to chlorophenoxy compounds:

- ▶ Acute toxic reactions are rare. The by-product of production, dioxin, may be implicated in subacute features such as hepatic enlargement, chloracne, neuromuscular symptoms and deranged

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- ▶ porphyrin metabolism.
- ▶ Large intentional overdoses result in coma, metabolic acidosis, myalgias, muscle weakness, elevated serum creatine kinase, myoglobinuria, irritation of the skin, eyes, respiratory tract and gut and mild renal and hepatic dysfunction.
- ▶ Several cases of sensorimotor peripheral neuropathies have been associated with chronic dermal exposure to 2,4-D. For acute exposures the usual methods of gut and skin contamination (lavage, charcoal, cathartic) are recommended in the first several hours. Alkalisiation of the urine and generous fluid replacement have the added benefit of treating any myoglobinuria present. Monitor metabolic acidosis, hyperthermia, hyperkalaemia, myoglobinuria and hepatic/renal dysfunction. for 2,4-dichlorophenoxyacetic acid (2,4-D) and its derivatives
- ▶ Gastric lavage if there are no signs of impending convulsions.
- ▶ Cautious administration of short-acting anticonvulsant drug if convulsions appear imminent.
- ▶ General supportive measures for central nervous system depression.
- ▶ If hypotension appears, search vigorously for a contributing cause (e.g. dehydration, electrolyte balance, acidosis, myocardial disturbances and hyperpyrexia).
- ▶ As appropriate, treat dehydration, electrolyte disturbances, acidosis, and hyperexia.
- ▶ To promote excretion of 2,4-D, initiate alkaline diuresis, as in salicylate poisoning by injecting sodium bicarbonate, intravenously, until the urine pH exceeds 7.5 and then infuse mannitol; renal clearance rises sharply as urine pH rises above 7.5 - above pH 8.0, it is said to be 100-fold greater than pH 6.0.
- ▶ If cardiac disturbances are suspected, monitor ECG continuously when possible. Prepare to deliver defibrillating shocks in the event of ventricular fibrillation.
- ▶ If hypotension intensifies, a trial with a vasopressor drug may be appropriate. Adrenalin (epinephrine) should be avoided because of possible fibrillation.
- ▶ If myotonia appears, a trial with quinidine may be helpful.
- ▶ Physiotherapy may be necessary for motion disorders associated with peripheral neuritis, myopathy or brain stem dysfunction.

GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products, 5th Ed.

In general, chlorophenoxy herbicides are rapidly absorbed from the gastrointestinal tract and evenly distributed throughout the body; accumulation in human tissues is not expected. A steady-state level in the human body will be achieved within 3–5 days of exposure. The herbicides are eliminated mainly in the urine, mostly unchanged, although fenoprop may be conjugated to a significant extent. Biological half-lives of chlorophenoxy herbicides in mammals range from 10 to 33 h; between 75% and 95% of the ingested amount is excreted within 96 h. Dogs appear to retain chlorophenoxy acids longer than other species as a result of relatively poor urinary clearance and thus may be more susceptible to their toxic effects. Metabolic conversions occur only at high doses. The salt and ester forms are rapidly hydrolysed and follow the same pharmacokinetic pathways as the free acids

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

- ▶ There is no restriction on the type of extinguisher which may be used.
- ▶ Use extinguishing media suitable for surrounding area.

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	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves in the event of a fire. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Use fire fighting procedures suitable for surrounding area. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Non combustible. ▶ Not considered to be a significant fire risk. ▶ Expansion or decomposition on heating may lead to violent rupture of containers. ▶ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. <p>Decomposition may produce toxic fumes of:</p> <ul style="list-style-type: none"> ‘ carbon dioxide (CO₂) ‘ other pyrolysis products typical of burning organic material. <p>May emit poisonous fumes. May emit corrosive fumes.</p>
HAZCHEM	Not Applicable

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	<ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb spill with sand, earth, inert material or vermiculite. ▶ Wipe up. ▶ Place in a suitable, labelled container for waste disposal.
Major Spills	<p>Moderate hazard.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling.

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- ▶ Neutralise/decontaminate residue (see Section 13 for specific agent).
- ▶ Collect solid residues and seal in labelled drums for disposal.
- ▶ Wash area and prevent runoff into drains.
- ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.
- ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Avoid contact with moisture. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ Polyethylene or polypropylene container. ▶ Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Avoid reaction with oxidising agents

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	sodium selenate, anhydrous	Selenium compounds (as Se) excluding hydrogen selenide	0.1 mg/m3	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
benzyl alcohol	Benzyl alcohol	30 ppm	52 ppm	740 ppm
sodium selenate, anhydrous	Sodium selenate; (Disodium selenate)	1.4 mg/m3	1.6 mg/m3	2 mg/m3

Ingredient	Original IDLH	Revised IDLH
triclabendazole	Not Available	Not Available
benzyl alcohol	Not Available	Not Available
abamectin	Not Available	Not Available
sodium selenate, anhydrous	1 mg/m3	Not Available

MATERIAL DATA

Exposure controls

Appropriate engineering controls	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. 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.</p>
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	<table border="1"> <tr> <td>Type of Contaminant:</td> <td>Air Speed:</td> </tr> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td> <td>0.25-0.5 m/s (50-100 f/min.)</td> </tr> <tr> <td>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)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </table> <p>Within each range the appropriate value depends on:</p> <table border="1"> <tr> <td>Lower end of the range</td> <td>Upper end of the range</td> </tr> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	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.)	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use	4: Large hood or large air mass in motion	4: Small hood-local control only
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Personal protection																					
Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] 																				
Skin protection	See Hand protection below																				
Hands/feet protection	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber <p>NOTE:</p> <ul style="list-style-type: none"> ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturizer is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> - frequency and duration of contact, - chemical resistance of glove material, - glove thickness and - dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> - When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. - When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. - Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. - Contaminated gloves should be replaced. <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> - Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. - Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p>																				
Body protection	See Other protection below																				

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Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C. apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit.
Thermal hazards	Not Available

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

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

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Material	CPI
BUTYL	C
VITON	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Respiratory protection

Type A 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	-	A-PAPR-AUS / Class 1
up to 50 x ES	-	A-AUS / Class 1	-
up to 100 x ES	-	A-2	A-PAPR-2 ^

^ - 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.

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Liquid.		
Physical state	Liquid	Relative density (Water = 1)	1.05-1.15
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Not Available	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. The material is not thought to produce respiratory irritation (as classified by EC Directives using animal models). Nevertheless inhalation of vapours, fumes or aerosols, especially for prolonged periods, may produce respiratory discomfort and occasionally, distress.
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. Ingestion may result in nausea, abdominal irritation, pain and vomiting
Skin Contact	The material is not thought to be a skin irritant (i.e. is unlikely to produce irritant dermatitis as described in EC Directives using animal models). Temporary discomfort, however, may result from prolonged dermal exposures. Good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. 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	Limited evidence exists, or practical experience suggests, that the material may cause eye irritation in a substantial number of individuals and/or is expected to 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 temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. 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. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, on the basis that similar materials tested in appropriate animal studies provide some suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects. Workers exposed to chlorophenoxy herbicides show a significant increase in soft-tissue sarcoma, malignant lymphomas and bronchial carcinomas. Prolonged or repeated contact with solutions may result in non-allergic dermatoses. Until recently, most epidemiological studies of the effects of chlorophenoxy herbicides dealt with populations exposed in the 1950s and 1960s, when the trichlorophenol-based herbicides 2,4,5-T and fenoprop were contaminated with polychlorinated dioxins and furans, including 2,3,7,8-tetrachlorodibenzodioxin (TCDD); the effects observed may therefore have been a consequence of the presence of the dioxin contaminants. In addition, most epidemiological studies on chlorophenoxy herbicides conducted to date have involved multiple exposures to chemical agents, including other pesticides and synthetic organic compounds. In a series of case-referent studies conducted in Sweden in the late 1970s and early 1980s, strong associations were noted between soft tissue sarcomas (STS) and multiple lymphomas (including Hodgkin disease (HD) and non-Hodgkin lymphoma (NHL)) and the use of chlorophenoxy herbicides by agricultural or forestry workers. The association between STS and chlorophenoxy herbicide use observed in the Swedish studies has not been confirmed in other case-referent studies. Although a number of cohort studies of occupationally exposed workers have been conducted, the small size of many of them limits their usefulness in assessing the relationship between STS and the herbicides. The risk for malignant lymphoma (HD + NHL) was almost five times greater for agricultural and forestry workers exposed to a mixture of chlorophenoxy herbicides than for controls in the case-referent study in Sweden but was not significantly elevated in a Danish cohort study of 3390 workers in a chemical plant manufacturing MCPA, dichlorprop, mecoprop, and 2,4-D, as well as other industrial chemicals and dyes Chronic exposure to 2,4-dichlorophenoxyacetic acid(2,4-D), its salts and its esters and its analogues may result in nausea, liver function changes, contact toxic dermatitis, irritation of the airways and eyes, as well as neurological changes. Persons with chronic diseases of the central nervous system, liver, heart, kidneys, lungs and skin, as well as those with endocrinological or immunological disturbances should not be exposed to herbicides (ILO Encyclopaedia). Groups of rats receiving 2,4-D in their diets for 13 weeks showed growth retardation and decreased food intake at 150 mg/kg/day dosage and an increased serum glutamic pyruvic transaminase (SGPT). A statistically significant incidence of astrocytoma was seen in the brains of male rats receiving 45 mg/kg/day for 104 weeks suggesting a possible carcinogenic effect although the prevalence of naturally occurring tumours in controls makes this result equivocal. A controversial study implicating 2,4-D as the cause of non-Hodgkin's lymphoma among male Kansas residents, aged 21 years or older, was difficult to evaluate because of a number of confounding factors. Agent Orange, a mixture of 2,4-D and 2,4,5-T, with contamination from 2,3,7,8-tetrachlorodibenzo-p-dioxin (also referred to as "dioxin" or TCDD) has been studied due to exposure of military personnel during its use as a herbicide in Vietnam. Neurological, reproductive and carcinogenic effects, purported to have occurred amongst veterans may be related to 2,4-D and 2,4,5-T but given the toxicity of the other components this remains the subject of conjecture. Most, if not all, occupational illnesses associated with 2,4,5-trichlorophenoxyacetic acids (2,4,5-T) and its derivatives actually result from TCDD contamination. Repeated overexposure to phenoxy herbicides may cause liver, kidney, gastrointestinal and muscular effects. Subchronic exposure by dogs to phenoxy herbicides produced a reduction in circulating lymphocytes Teratogenic response was exhibited in mice (but not rats). Cleft palate was demonstrated. No such findings occurred in non-human primates given up to 10 mg/kg/day (containing 0.05 ppm TCDD) from gestation day 22 to 38. The no-observed effect level (NOAEL) in hamsters was 2 mg/kg 2,4,5-T A number of benzimidazoles have been shown to also inhibit mammalian tubulin polymerisation and to be aneugenic <i>in vivo</i> . Aneugens affect cell division and the mitotic spindle apparatus resulting in loss or gain of whole chromosomes, thereby inducing an "aneuploidy". Mitotic aneuploidy is a characteristic of many types of tumorigenesis (in cancer). Several benzimidazoles have been shown to be genotoxic. Genotoxicity may arise as aneugens may also be clastogens, or may produce clastogenic metabolites. Clastogens increase the rate of genetic mutation by interfering with the function of nucleic acids. A clastogen is a specific mutagen that causes breaks in chromosomes.

Flukamec Plus Selenium Anthelmintic for Sheep	TOXICITY	IRRITATION
	Not Available	Not Available
triclabendazole	TOXICITY	IRRITATION
	dermal (rat) LD50: >4000 mg/kg ^[2]	Eye: slight *
	Inhalation (rat) LC50: >0.5 mg/l/4h ^[2]	
	Oral (rat) LD50: >8000 mg/kg ^[2]	

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benzyl alcohol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 2000 mg/kg ^[2]	Eye (rabbit): 0.75 mg open SEVERE
	Inhalation (rat) LC50: >4.178 mg/l/4h ^[2]	Skin (man): 16 mg/48h-mild
	Oral (rat) LD50: 1230 mg/kg ^[2]	Skin (rabbit):10 mg/24h open-mild
abamectin	TOXICITY	IRRITATION
	dermal (rat) LD50: >330 mg/kg ^[2]	Eye (rabbit): slight *
	Inhalation (rat) LC50: 1.1 mg/l/4h ^[2]	Skin (rabbit): non irritating*
	Oral (rat) LD50: 1.5 mg/kg ^[2]	
sodium selenate, anhydrous	TOXICITY	IRRITATION
	Oral (rat) LD50: 1.6 mg/kg ^[2]	Not Available

Legend:

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

TRICLABENDAZOLE

For chlorophenoxy pesticides:
551chlph

WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.

Side-reactions during manufacture of the parent compound may result in the production of trace amounts of polyhalogenated aromatic hydrocarbon(s). Halogenated phenols, and especially their alkali salts, can condense above 300 deg. C . to form polyphenoxyphenols or, in a very specific reaction, to form dibenzo-p-dioxins

Polyhalogenated aromatic hydrocarbons (PHAHs) comprise two major groups. The first group represented by the halogenated derivatives of dibenzodioxins (the chlorinated form is PCDD), dibenzofurans (PCDF) and biphenyls (PCB) exert their toxic effect (as hepatocarcinogens, reproductive toxicants, immunotoxicants and procarcinogens) by interaction with a cytosolic protein known as the Ah receptor. In guinea pigs the Ah receptor is active in a mechanism which "pumps" PHAH into the cell whilst in humans the reverse appears to be true. This, in part, may account for species differences often cited in the literature. This receptor exhibits an affinity for the planar members of this group and carries these to the cellular nucleus where they bind, reversibly, to specific genomes on DNA. This results in the regulation of the production of certain proteins which elicit the toxic response. The potency of the effect is dependent on the strength of the original interaction with the Ah receptor and is influenced by the degree of substitution by the halogen and the position of such substitutions on the parent compound.

The most potent molecule is 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) while the coplanar PCBs (including mono-ortho coplanars) possess approximately 1% of this potency. Nevertheless, all are said to exhibit "dioxin-like" behaviour and in environmental and health assessments it has been the practice to assign each a TCDD-equivalence value.

The most subtle and important biological effects of the PHAHs are the effects on endocrine hormones and vitamin homeostasis. TCDD mimics the effect of thyroxine (a key metamorphosis signal during maturation) and may disrupt patterns of embryonic development at critical stages. Individuals from exposed wildlife populations have been observed to have altered sexual development, sexual dysfunction as adults and immune system suppression. Immunotoxic effects of the PHAHs (including the brominated congener, PBB) have been the subject of several studies. No clear pattern emerges in human studies however with T-cell numbers and function (a blood marker for immunological response) increasing in some and decreasing in others.

Developmental toxicity (e.g. cleft palate, hydronephrosis) occurs in relatively few species; functional alterations following TCDD exposure leads to deficits in cognitive functions in monkeys and to adverse effects in the male reproductive system of rats.

Three incidences have occurred which have introduced abnormally high levels of dioxin or dioxin-like congeners to humans. The explosion at a trichlorophenol-manufacturing plant in Seveso, Italy distributed TCDD across a large area of the country-side, whilst rice-oil contaminated with heat-transfer PCBs (and dioxin-like contaminants) has been consumed by two groups, on separate occasions (one in Yusho, Japan and another in Yu-cheng, Taiwan). The only symptom which can unequivocally be related to all these exposures is the development of chloracne, a disfiguring skin condition, following each incident. Contaminated oil poisonings also produced eye-discharge, swelling of eyelids and visual disturbances. The Babies born up to 3 years after maternal exposure (so-called "Yusho-babies") were characteristically brown skinned, coloured gums and nails and (frequently) produced eye-discharges. Delays in intellectual development have been noted. It has been estimated that Yu-cheng patients consumed an average level of 0.06 mg/kg body weight/day total PCB and 0.0002 mg/kg/day of PCDF before the onset of symptoms after 3 months. When the oil was withdrawn after 6 months they had consumed 1 gm total PCB containing 3.8 mg PCDF. Taiwanese patients consumed 10 times as much contaminated oil as the Japanese patients (because of later withdrawal); however since PCB/PCDF concentration in the Japanese oil was 10 times that consumed in Taiwan, patients from both countries consumed about the same amount of PCBs/PCDFs. Preliminary data from the Yusho cohort suggests a six-fold excess of liver cancer mortality in males and a three-fold excess in women.

Recent findings from Seveso indicate that the biological effects of low level exposure (BELLEs), experienced by a cohort located at a great distance from the plant, may be protective AGAINST the development of cancer. The PHAHs do not appear to be genotoxic - they do not alter the integrity of DNA. This contrasts with the effects of the many polycyclic aromatic hydrocarbons (PAHs) (or more properly, their reactive metabolites). TCDD induces carcinogenic effects in the laboratory in all species, strains and sexes tested. These effects are dose-related and occur in many organs. Exposures as low as 0.001 ug/kg body weight/day produce carcinoma. Several studies implicate PCBs in the development of liver cancer in workers as well as multi-site cancers in animals. The second major group of PHAH consists of the non-planar PCB congeners which possess two or more ortho-substituted halogens. These have been shown to produce neurotoxic effects which are thought to reduce the concentration of the brain neurotransmitter, dopamine, by inhibiting certain enzyme-mediated processes. The specific effect elicited by both classes of PHAH seems to depend on the as much on the developmental status of the organism at the time of the exposure as on the level of exposure over a lifetime.

NOTE: Some jurisdictions require that health surveillance be conducted on workers occupationally exposed to polycyclic aromatic hydrocarbons. Such surveillance should emphasise

- ▶ demography, occupational and medical history
- ▶ health advice, including recognition of photosensitivity and skin changes
- ▶ physical examination if indicated
- ▶ records of personal exposure including photosensitivity

Foetotoxicity recorded. * Transchem MSDS

Flukamec Plus Selenium Anthelmintic for Sheep

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.

For benzyl alkyl alcohols:

Unlike benzylic alcohols, the beta-hydroxyl group of the members of this cluster is unlikely to undergo phase II metabolic activation. Instead, the beta-hydroxyl group is expected to contribute to detoxification via oxidation to hydrophilic acid. Despite structural similarity to carcinogenic ethyl benzene, only a marginal concern has been assigned to phenethyl alcohol due to limited mechanistic analogy.

For benzoates:

Acute toxicity: Benzyl alcohol, benzoic acid and its sodium and potassium salt can be considered as a single category regarding human health, as they are all rapidly metabolised and excreted via a common pathway within 24 hrs. Systemic toxic effects of similar nature (e.g. liver, kidney) were observed. However with benzoic acid and its salts toxic effects are seen at higher doses than with benzyl alcohol.

The compounds exhibit low acute toxicity as for the oral and dermal route. The LD50 values are > 2000 mg/kg bw except for benzyl alcohol which needs to be considered as harmful by the oral route in view of an oral LD50 of 1610 mg/kg bw. The 4 hrs inhalation exposure of benzyl alcohol or benzoic acid at 4 and 12 mg/l as aerosol/dust respectively gave no mortality, showing low acute toxicity by inhalation for these compounds.

Benzoic acid and benzyl alcohol are slightly irritating to the skin, while sodium benzoate was not skin irritating. No data are available for potassium benzoate but it is also expected not to be skin irritating. Benzoic acid and benzyl alcohol are irritating to the eye and sodium benzoate was only slightly irritating to the eye. No data are available for potassium benzoate but it is expected also to be only slightly irritating to the eye.

Sensitisation: The available studies for benzoic acid gave no indication for a sensitising effect in animals, however occasionally very low positive reactions were recorded with humans (dermatological patients) in patch tests. The same occurs for sodium benzoate. It has been suggested that the very low positive reactions are non-immunologic contact urticaria. Benzyl alcohol gave positive and negative results in animals. Benzyl alcohol also demonstrated a maximum incidence of sensitization of only 1% in human patch testing. Over several decades no sensitization with these compounds has been seen among workers.

Repeat dose toxicity: For benzoic acid repeated dose oral toxicity studies give a NOAEL of 800 mg/kg/day. For the salts values > 1000 mg/kg/day are obtained. At higher doses increased mortality, reduced weight gain, liver and kidney effects were observed.

For benzyl alcohol the long-term studies indicate a NOAEL > 400 mg/kg bw/d for rats and > 200 mg/kg bw/d for mice. At higher doses effects on bodyweights, lesions in the brains, thymus, skeletal muscle and kidney were observed. It should be taken into account that administration in these studies was by gavage route, at which saturation of metabolic pathways is likely to occur.

Mutagenicity: All chemicals showed no mutagenic activity in *in vitro* Ames tests. Various results were obtained with other *in vitro* genotoxicity assays. Sodium benzoate and benzyl alcohol showed no genotoxicity *in vivo*. While some mixed and/or equivocal *in vitro* chromosomal/chromatid responses have been observed, no genotoxicity was observed in the *in vivo* cytogenetic, micronucleus, or other assays. The weight of the evidence of the *in vitro* and *in vivo* genotoxicity data indicates that these chemicals are not mutagenic or clastogenic. They also are not carcinogenic in long-term carcinogenicity studies.

In a 4-generation study with benzoic acid no effects on reproduction were seen (NOAEL: 750 mg/kg). No compound related effects on reproductive organs (gross and histopathology examination) could be found in the (sub) chronic studies in rats and mice with benzyl acetate, benzyl alcohol, benzaldehyde, sodium benzoate and supports a non-reprotoxic potential of these compounds. In addition, data from reprotoxicity studies on benzyl acetate (NOAEL >2000 mg/kg bw/d; rats and mice) and benzaldehyde (tested only up to 5 mg/kg bw; rats) support the non-reprotoxicity of benzyl alcohol and benzoic acid and its salts.

Developmental toxicity: In rats for sodium benzoate dosed via food during the entire gestation developmental effects occurred only in the presence of marked maternal toxicity (reduced food intake and decreased body weight) (NOAEL = 1400 mg/kg bw). For hamster (NOEL: 300 mg/kg bw), rabbit (NOEL: 250 mg/kg bw) and mice (CD-1 mice, NOEL: 175 mg/kg bw) no higher doses (all by gavage) were tested and no maternal toxicity was observed. For benzyl alcohol: NOAEL= 550 mg/kg bw (gavage; CD-1 mice). LOAEL = 750 mg/kg bw (gavage mice). In this study maternal toxicity was observed e.g. increased mortality, reduced body weight and clinical toxicology. Benzyl acetate: NOEL = 500 mg/kg bw (gavage rats). No maternal toxicity was observed. Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and conjugal contact dermatitis occur. Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes.

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

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

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

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

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

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

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

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

BENZYL ALCOHOL

Flukamec Plus Selenium Anthelmintic for Sheep

increased risk of being fragrance allergic.

Irritant reactions (including contact urticaria): Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this. Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported. The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested, but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen.

Pigmentary anomalies: The term "pigmented cosmetic dermatitis" was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil.

Photo-reactions Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon. Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. Phototoxic reactions still occur but are rare.

General/respiratory: Fragrances are volatile and therefore, in addition to skin exposure, a perfume also exposes the eyes and naso-respiratory tract. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma. Asthma-like symptoms can be provoked by sensory mechanisms. In an epidemiological investigation, a significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.

Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A prohaptens is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems. A prohaptens is a chemical that itself is non- or low-sensitising but that is transformed into a hapten in the skin (bioactivation) usually via enzyme catalysis. It is not always possible to know whether a particular allergen that is not directly reactive acts as a prohaptens or as a prohaptens, or both, because air oxidation and bioactivation can often give the same product (geraniol is an example). Some chemicals might act by all three pathways.

Prohaptens

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

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

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

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

A member or analogue of a group of benzyl derivatives generally regarded as safe (GRAS) based in part on their self-limiting properties as flavouring substances in food; their rapid absorption, metabolic detoxification, and excretion in humans and other animals, their low level of flavour use, the wide margin of safety between the conservative estimates of intake and the no-observed-adverse effect levels determined from chronic and subchronic studies and the lack of significant genotoxic and mutagenic potential. This evidence of safety is supported by the fact that the intake of benzyl derivatives as natural components of traditional foods is greater than the intake as intentionally added flavouring substances.

All members of this group are aromatic primary alcohols, aldehydes, carboxylic acids or their corresponding esters or acetals. The substances in this group:

- contain a benzene ring substituted with a reactive primary oxygenated functional group or can be hydrolysed to such a functional group
- the major pathway of metabolic detoxification involves hydrolysis and oxidation to yield the corresponding benzoic acid derivative which is excreted either as the free acid or the glycine conjugate
- they show a consistent pattern of toxicity in both short- and long-term studies and
- they exhibit no evidence of genotoxicity in standardised batteries of in vitro and in vivo assays.

The benzyl derivatives are rapidly absorbed through the gut, metabolised primarily in the liver, and excreted in the urine as glycine conjugates of benzoic acid derivatives.

In general, aromatic esters are hydrolysed in vivo through the catalytic activity of carboxylesterases, the most important of which are the A-esterases. Hydrolysis of benzyl and benzoate esters to yield corresponding alcohols and carboxylic acids and hydrolysis of acetals to yield benzaldehyde and simple alcohols have been reported in several experiments.

The alcohols and aldehydes are rapidly oxidised to benzoic acid while benzoate esters are hydrolysed to benzoic acid.

Flavor and Extract Manufacturers Association (FEMA)

The aryl alkyl alcohol (AAA) fragrance ingredients are a diverse group of chemical structures with similar metabolic and toxicity profiles.

The AAA fragrances demonstrate low acute and subchronic dermal and oral toxicity.

At concentrations likely to be encountered by consumers, AAA fragrance ingredients are non-irritating to the skin.

The potential for eye irritation is minimal.

With the exception of benzyl alcohol and to a lesser extent phenethyl and 2-phenoxyethyl AAA alcohols, human sensitization studies, diagnostic patch tests and human induction studies, indicate that AAA fragrance ingredients generally have no or low sensitization potential. Available data indicate that the potential for photosensitization is low.

NOAELs for maternal and developmental toxicity are far in excess of current human exposure levels.

No carcinogenicity in rats or mice was observed in 2-year chronic testing of benzyl alcohol or a-methylbenzyl alcohol; the latter did induce species and gender-specific renal adenomas in male rats at the high dose. There was no to little genotoxicity, mutagenicity, or clastogenicity in the mutagenicity in vitro bacterial assays, and in vitro mammalian cell assays. All in vivo micronucleus assays were negative.

It is concluded that these materials would not present a safety concern at current levels of use as fragrance ingredients

The Research Institute for Fragrance Materials (RIFM) Expert Panel

Flukamec Plus Selenium Anthelmintic for Sheep

ABAMECTIN	<p>No significant acute toxicological data identified in literature search.</p> <p>For avermectins:</p> <p>Technical avermectin exhibits high mammalian acute toxicity. It is not considered to be mutagenic and does not sensitise skin. It is not readily absorbed by mammals and the majority of the residue is excreted in the faeces within 2 days. The 24-month rat chronic feeding/ oncogenicity study and 94-week mouse chronic toxicity oncogenicity study were negative for oncogenic potential. The results of a series of developmental toxicity studies (rat, rabbit, mouse) have been evaluated and showed that avermectin B1 produces developmental toxicity (cleft palate) in the CF1 mouse. Toxicology data were also evaluated for the delta-8,9-isomer of avermectin B1 which is a plant photodegrade that can range between 5 and 20 percent of the residue on/in cottonseed. This isomer possesses avermectin-like toxicological activity. It was concluded that the delta 8,9-isomer also produces developmental toxicity (cleft palate) in mice, but not in rats. In addition to avermectin and its delta 8,9-isomer, toxicology data were also evaluated for the "polar degradates" of avermectin, which constitute a large percentage (up to 70%) of the total residue on cottonseed. Review of the toxicology data indicated that these polar degradates do not possess avermectin-like toxicological activity and for this reason need not be included in the tolerance expression for residues in/on cottonseed.</p> <p>Abamectin (a mixture of avermectin isomers) is a reproductive toxin in laboratory animals at doses which are acutely toxic to the mother. In development toxicity studies with abamectin, cleft palates were seen in mice and rabbits and clubbing of the forepaws was seen in rabbits. The no-observed-adverse-effect-level (NOAEL) for maternal and developmental toxicity in rabbits was 1 mg/kg/day. In CF-1 mice, a strain recognised to be particularly sensitive to avermectins, the NOAEL for maternal toxicity was 0.05 mg/kg/day and the NOAEL for malformations was 0.2 mg/kg/day. Studies show that the sensitivity of a subpopulation of CF-1 mice to avermectins is due to the absence of a transmembrane P-glycoprotein, a significant component of the blood-brain interface that normally acts as a non-selective protective barrier in a wide range of species including humans. CF-1 mice are therefore an unlikely candidate for assessing human risk. No evidence of developmental toxicity was seen in oral studies in rats in the absence of maternal toxicity (NOAEL = 1.6 mg/kg/day). In a rat multigenerational reproduction study, pup toxicity and deaths were seen at 0.4 mg/kg/day (NOAEL = 0.12 mg/kg/day). Neonatal rats are not an appropriate model for assessing human risk in humans because (a) rat milk has a greater fat content than human breast milk and abamectin concentrates in fat; (b) on a weight basis, the neonatal rat consumes significantly greater quantities of milk than the newborn human and (c) the blood brain barrier in rodents is formed post-natally (as evidenced by low P-glycoprotein levels) while in humans this membrane is formed pre-natally.</p> <p>Ivermectin, a close structural analogue, has been used extensively in the treatment of human onchocerciasis at an oral therapeutic dose of 0.2 mg/kg, without serious drug-related effects. Despite its wide usage in animals and humans, ivermectin does not appear to produce birth defects.</p> <p>Abamectin is non-mutagenic in the Ames test and the micronucleus test.</p> <p>Dietary carcinogenicity studies in mice and rats showed negative results. In a 14-week oral study in monkeys no effects were seen at 0.2, 0.5 or 1.0 mg/kg/day; emesis was seen at 2.0 mg/kg/day; delayed pupillary obstruction at 6 and 8 mg/kg/day and mydriasis at 12 mg/kg/day.</p> <p>In chronic oral toxicity, abamectin produced decreased body weight gain in mice (no-observed-adverse-effect-level (NOAEL) = 1.5 mg/kg/day); tremors in rats (NOAEL = 1.5 mg/kg/day), weight loss, tremors, mydriasis, liver and gall bladder changes and death in dogs (NOAEL = 0.25 mg/kg/day); and emesis, mydriasis and sedation in monkeys (NOAL = 1 mg/kg/day).</p> <p>Oral (rat) LD50: 8.7-12.8 mg/kg (14 day) * ADI 0.0001 mg/kg Toxicity Class EPA IV Non-mutagenic in the Ames test ADI: 0.4 mg/day [Manufacturer] Convulsions recorded.</p>
SODIUM SELENATE, ANHYDROUS	<p>The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans.</p> <p>Evidence of carcinogenicity may be inadequate or limited in animal testing.</p> <p>Eye effects, general anaesthesia, convulsions, muscle weakness, spasticity, cardiac EKG changes, cyanosis, lung tumours, diarrhoea, impaired liver function tests, leukaemia, specific developmental changes, effects on newborn recorded.</p>

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 available but does not fill the criteria for classification
✔ – Data available to make classification
⊘ – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Flukamec Plus Selenium Anthelmintic for Sheep	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
triclabendazole	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
benzyl alcohol	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96		Fish	10mg/L	4
abamectin	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
EC50	96		Algae or other aquatic plants	7.3096mg/L	4
sodium selenate, anhydrous	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96		Fish	0.69mg/L	4
EC50	48		Crustacea	0.083mg/L	4
EC50	96		Algae or other aquatic plants	0.199mg/L	4
BCF	336		Algae or other aquatic plants	10mg/L	4
NOEC	168		Algae or other aquatic plants	0.0091mg/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

Flukamec Plus Selenium Anthelmintic for Sheep

(Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

for chlorophenoxy herbicides:

Environmental fate:

Residues of chlorophenoxy herbicides in the environment are the consequence of the direct application of these compounds to agricultural and non-agricultural areas. Biodegradation is the primary route of elimination from the environment; photolysis and hydrolysis also contribute to their removal.

The chlorophenoxy herbicides are considered to have only marginal potential for leaching to groundwater. In basic waters, phenoxy herbicide esters are hydrolysed to the anionic forms; in acidic waters, photodegradation or vapourisation predominates, depending on the ester.

Chlorophenoxy herbicides may be transported in the atmosphere in the form of droplets, vapour, or powder following application by spraying.

Chlorophenoxy herbicides may be present in food as a result of their direct application to crops; however, concentrations are normally low.

The group of acidic herbicides, including the phenoxy acids, possess functional groups that ionise in aqueous systems yielding pKa values of less than 4. The behaviour of these materials is closely correlated with their acid character. The most significant factor with respect to soil mobility is the organic content of the soil which readily absorbs these compounds. Furthermore in acidic systems these compounds are also absorbed by clay particles. The esters and ethers are expected to behave differently from the acid forms although hydrolysis may influence subsequent binding. In general the esters and ethers are considered non-persistent in the environment.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
benzyl alcohol	LOW	LOW
sodium selenate, anhydrous	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
benzyl alcohol	LOW (LogKOW = 1.1)
sodium selenate, anhydrous	LOW (LogKOW = -3.1818)

Mobility in soil

Ingredient	Mobility
benzyl alcohol	LOW (KOC = 15.66)
sodium selenate, anhydrous	LOW (KOC = 48.64)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▶ 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. <p>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.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> ▶ 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.
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SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

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

Not Applicable

SECTION 15 REGULATORY INFORMATION**Safety, health and environmental regulations / legislation specific for the substance or mixture****TRICLABENDAZOLE(68786-66-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

BENZYL ALCOHOL(100-51-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

ABAMECTIN(71751-41-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists

SODIUM SELENATE, ANHYDROUS(13410-01-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Inventory of Chemical Substances (AICS)

Australia Hazardous Substances Information System - Consolidated Lists

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

National Inventory	Status
Australia - AICS	N (abamectin)
Canada - DSL	N (abamectin; triclabendazole)
Canada - NDSL	N (benzyl alcohol; abamectin; triclabendazole; sodium selenate, anhydrous)
China - IECSC	N (abamectin; triclabendazole)
Europe - EINEC / ELINCS / NLP	N (abamectin; triclabendazole)
Japan - ENCS	N (abamectin; triclabendazole)
Korea - KECI	N (triclabendazole)
New Zealand - NZIoC	Y
Philippines - PICCS	N (abamectin; triclabendazole)
USA - TSCA	N (abamectin; triclabendazole)
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION**Other information****Ingredients with multiple cas numbers**

Name	CAS No
abamectin	71751-41-2, 86753-29-9

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
 OSF: Odour Safety Factor
 NOAEL: No Observed Adverse Effect Level
 LOAEL: Lowest Observed Adverse Effect Level
 TLV: Threshold Limit Value
 LOD: Limit Of Detection
 OTV: Odour Threshold Value
 BCF: BioConcentration Factors
 BEI: Biological Exposure Index

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