Moxistar LV Se Low Volume Oral Drench for Sheep with Selenium

The Hunter River Company Pty Ltd

Chemwatch Hazard Alert Code: **2**

Chemwatch: **5667-96**
Version No: **2.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Issue Date: **21/06/2024**
Print Date: **21/06/2024**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	Moxistar LV Se Low Volume Oral Drench for Sheep with Selenium
Chemical Name	Not Applicable
Synonyms	APVMA No. 86229
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	For the treatment and control of moxidectin sensitive gastrointestinal parasites, lungworm and itchmite of sheep. An aid in the control of selenium responsive conditions.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**The Hunter River Company Pty Ltd**
Address	74-76 Drummond Road Shepparton VIC 3630 Australia
Telephone	03 5820 8400
Fax	Not Available
Website	http://www.hunterriverco.com.au/
Email	Not Available

Emergency telephone number

Association / Organisation	**The Hunter River Company Pty Ltd**
Emergency telephone numbers	03 5820 8400 (Mon-Fri 9-5pm)
Other emergency telephone numbers	13 11 26 (24 hours for Poisons Info Centre)

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	S6
Classification [1]	Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2A, Hazardous to the Aquatic Environment Acute Hazard Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI***

Label elements

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

Hazard statement(s)

H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H319	Causes serious eye irritation.
H401	Toxic to aquatic life.
H412	Harmful to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.

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P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P302+P352	IF ON SKIN: Wash with plenty of water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

Not Applicable

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
57-55-6	10-30	propylene glycol
100-51-6	1-10	benzyl alcohol
113507-06-5	<1	moxidectin
13410-01-0	<1	sodium selenate, anhydrous
Not Available	balance	Ingredients determined not to be hazardous
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available***	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Wash out immediately with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice. ▸ Avoid giving milk or oils. ▸ Avoid giving alcohol.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.
To treat poisoning by the higher aliphatic alcohols (up to C7):
- Gastric lavage with copious amounts of water.
- It may be beneficial to instill 60 ml of mineral oil into the stomach.
- Oxygen and artificial respiration as needed.
- Electrolyte balance: it may be useful to start 500 ml. M/6 sodium bicarbonate intravenously but maintain a cautious and conservative attitude toward electrolyte replacement unless shock or severe acidosis threatens.
- To protect the liver, maintain carbohydrate intake by intravenous infusions of glucose.
- Haemodialysis if coma is deep and persistent. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products, Ed 5)

BASIC TREATMENT

- Establish a patent airway with suction where necessary.
- Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- Monitor and treat, where necessary, for shock.
- Monitor and treat, where necessary, for pulmonary oedema.
- Anticipate and treat, where necessary, for seizures.

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- **DO NOT** use emetics. Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.
- Give activated charcoal.

ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- If the patient is hypoglycaemic (decreased or loss of consciousness, tachycardia, pallor, dilated pupils, diaphoresis and/or dextrose strip or glucometer readings below 50 mg), give 50% dextrose.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Treat seizures with diazepam.
- Proparacaine hydrochloride should be used to assist eye irrigation.

EMERGENCY DEPARTMENT

- Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- Acidosis may respond to hyperventilation and bicarbonate therapy.
- Haemodialysis might be considered in patients with severe intoxication.
- Consult a toxicologist as necessary. BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

For C8 alcohols and above.
Symptomatic and supportive therapy is advised in managing patients.
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.).

SECTION 5 Firefighting measures

Extinguishing media

The product contains a substantial proportion of water, therefore there are no restrictions on the type of extinguishing media which may be used. Choice of extinguishing media should take into account surrounding areas.
Though the material is non-combustible, evaporation of water from the mixture, caused by the heat of nearby fire, may produce floating layers of combustible substances.
In such an event consider:

- foam.
- dry chemical powder.
- carbon dioxide.

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.

Advice for firefighters

Fire Fighting	• 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	• The material is not readily combustible under normal conditions. • However, it will break down under fire conditions and the organic component may burn. • Not considered to be a significant fire risk. • Heat may cause expansion or decomposition with violent rupture of containers. • Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). • May emit acrid smoke. Decomposes on heating and produces toxic fumes of: carbon dioxide (CO_2) nitrogen oxides (NO_x) metal oxides other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.
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	• Clean up all spills immediately. • Avoid breathing vapours and contact with skin and eyes. • Control personal contact with the substance, by using protective equipment.

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	▸ Contain and absorb spill with sand, earth, inert material or vermiculite. ▸ Wipe up. ▸ Place in a suitable, labelled container for waste disposal.
Major Spills	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. ▸ Stop leak if safe to do so. ▸ Contain spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ 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	▸ **DO NOT** allow clothing wet with material to stay in contact with skin ▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ **DO NOT** allow material to contact humans, exposed food or food utensils. ▸ 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	▸ 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. Store below 30 deg. C. Store out of direct sunlight

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Glass container is suitable for laboratory quantities ▸ Polyethylene or polypropylene container. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ Glycols and their ethers undergo violent decomposition in contact with 70% perchloric acid. This seems likely to involve formation of the glycol perchlorate esters (after scission of ethers) which are explosive, those of ethylene glycol and 3-chloro-1,2-propanediol being more powerful than glyceryl nitrate, and the former so sensitive that it explodes on addition of water. Alcohols ▸ are incompatible with strong acids, acid chlorides, acid anhydrides, oxidising and reducing agents. ▸ reacts, possibly violently, with alkaline metals and alkaline earth metals to produce hydrogen ▸ react with strong acids, strong caustics, aliphatic amines, isocyanates, acetaldehyde, benzoyl peroxide, chromic acid, chromium oxide, dialkylzincs, dichlorine oxide, ethylene oxide, hypochlorous acid, isopropyl chlorocarbonate, lithium tetrahydroaluminate, nitrogen dioxide, pentafluoroguanidine, phosphorus halides, phosphorus pentasulfide, tangerine oil, triethylaluminium, triisobutylaluminium ▸ should not be heated above 49 deg. C. when in contact with aluminium equipment

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	propylene glycol	Propane-1,2-diol total: (vapour & particulates)	150 ppm / 474 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	propylene glycol	Propane-1,2-diol: particulates only	10 mg/m3	Not Available	Not Available	Not Available
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	TEEL-1	TEEL-2	TEEL-3
propylene glycol	30 mg/m3	1,300 mg/m3	7,900 mg/m3
benzyl alcohol	30 ppm	52 ppm	740 ppm
sodium selenate, anhydrous	1.4 mg/m3	1.6 mg/m3	2 mg/m3

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Ingredient	Original IDLH	Revised IDLH
propylene glycol	Not Available	Not Available
benzyl alcohol	Not Available	Not Available
moxidectin	Not Available	Not Available
sodium selenate, anhydrous	1 mg/m3	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
benzyl alcohol	E	≤ 0.1 ppm
moxidectin	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

For potent pharmacological agents:
Solutions Handling:
- Solutions can be handled outside a containment system or without local exhaust ventilation during procedures with no potential for aerosolisation. If the procedures have a potential for aerosolisation, an air-purifying respirator is to be worn by all personnel in the immediate area.
- Solutions used for procedures where aerosolisation may occur (e.g., vortexing, pumping) are to be handled within a containment system or with local exhaust ventilation.
- In situations where this is not feasible (may include animal dosing), an air-purifying respirator is to be worn by all personnel in the immediate area. If using a ventilated enclosure that has not been validated, wear a half-mask respirator equipped with HEPA cartridges until the enclosure is validated for use.
- Ensure gloves are protective against solvents in use.

Unless written procedures, specific to the workplace are available, the following is intended as a guide:
- **For Laboratory-scale handling of Substances assessed to be toxic by inhalation.** ***Quantities of up to 25 grams*** may be handled in Class II biological safety cabinets *; ***Quantities of 25 grams to 1 kilogram*** may be handled in Class II biological safety cabinets* or equivalent containment systems; ***Quantities exceeding 1 kg*** may be handled either using specific containment, a hood or Class II biological safety cabinet*,
- HEPA terminated local exhaust ventilation should be considered at point of generation of dust, fumes or vapours.
- The need for respiratory protection should also be assessed where incidental or accidental exposure is anticipated. Dependent on levels of contamination, PAPR, full face air purifying devices with P2 or P3 filters or air supplied respirators should be evaluated. When handling: ***Quantities of up to 25 grams***, an approved respirator with HEPA filters or cartridges should be considered; ***Quantities of 25 grams to 1 kilogram,*** a half-face negative pressure, full negative pressure, or powered helmet-type air purifying respirator should be considered. ***Quantities in excess of 1 kilogram*****,** a full face negative pressure, helmet-type air purifying, or supplied air respirator should be considered.

Written procedures, specific to a particular work-place, may replace these recommendations
* For Class II Biological Safety Cabinets, Types B2 or B3 should be considered. Where only Class I, open fronted Cabinets are available, glove panels may be added, Laminar flow cabinets do not provide sufficient protection when handling these materials unless especially designed to do so.
Pilot Plant and Production
- Wear appropriate gloves; lab coat, nylon coveralls or disposable Tyvek suit; safety glasses, safety shoes, and disposable booties. Use good manufacturing practices (i.e., cGMPs).
- Protective garment (coveralls, Tyvek, lab coat) is not to be worn outside the work area.
- Clean/dirty/decontamination areas are to be established.
- Negative/positive air pressure relationships and buffer zones required (i.e., ante-room/degowning room/airlock).
- Area access is to be restricted.
- High-energy operations such as milling, particle sizing, spraying or fluidising should be done within an approved emission control or containment system.
- Develop cleaning procedures and techniques that limit potential exposure

Individual protection measures, such as personal protective equipment

Eye and face protection

When handling very small quantities of the material eye protection may not be required.
For laboratory, larger scale or bulk handling or where regular exposure in an occupational setting occurs:
- Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]
- Face shield. Full face shield may be required for supplementary but never for primary protection of eyes.
- 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].

Skin protection

See Hand protection below

Hands/feet protection

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,

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	· 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. ▸ Rubber gloves (nitrile or low-protein, powder-free latex, latex/ nitrile). Employees allergic to latex gloves should use nitrile gloves in preference. ▸ Double gloving should be considered. ▸ PVC gloves. ▸ Change gloves frequently and when contaminated, punctured or torn. ▸ Wash hands immediately after removing gloves. ▸ Protective shoe covers. [AS/NZS 2210] ▸ Head covering.
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

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:
Moxistar LV Se Low Volume Oral Drench for Sheep with Selenium

Material	CPI
BUTYL	C
NATURAL RUBBER	C
NEOPRENE	C
PE/EVAL/PE	C
PVA	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.

Ansell Glove Selection

Glove — *In order of recommendation*
AlphaTec® Solvex® 37-675
MICROFLEX® 93-260
TouchNTuff® 92-500
TouchNTuff® 92-605
TouchNTuff® 92-600
TouchNTuff® 93-250
TouchNTuff® 93-700
AlphaTec® 15-554
AlphaTec® Solvex® 37-185
AlphaTec® 38-612

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required. Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

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

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

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

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The suggested gloves for use should be confirmed with the glove supplier.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Pale yellow liquid with mild odour; miscible with water.

Physical state	Liquid	**Relative density (Water = 1)**	Not Available
Odour	Mild odour	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Applicable
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	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	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	Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. Exposure to aliphatic alcohols with more than 3 carbons may produce central nervous system effects such as headache, dizziness, drowsiness, muscle weakness, delirium, CNS depression, coma, seizure, and neurobehavioural changes. Symptoms are more acute with higher alcohols. Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, respiratory depression secondary to CNS depression, pulmonary oedema, chemical pneumonitis and bronchitis. Cardiovascular involvement may result in arrhythmias and hypotension. Gastrointestinal effects may include nausea and vomiting. Kidney and liver damage may result following massive exposures. The alcohols are potential irritants being, generally, stronger irritants than similar organic structures that lack functional groups (e.g. alkanes) but are much less irritating than the corresponding amines, aldehydes or ketones. Alcohols and glycols (diols) rarely represent serious hazards in the workplace, because their vapour concentrations are usually less than the levels which produce significant irritation which, in turn, produce significant central nervous system effects as well.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Effects on the nervous system characterise over-exposure to higher aliphatic alcohols. These include headache, muscle weakness, giddiness, ataxia, (loss of muscle coordination), confusion, delirium and coma. Gastrointestinal effects may include nausea, vomiting and diarrhoea. In the absence of effective treatment, respiratory arrest is the most common cause of death in animals acutely poisoned by the higher alcohols. Aspiration of liquid alcohols produces an especially toxic response as they are able to penetrate deeply in the lung where they are absorbed and may produce pulmonary injury. Those possessing lower viscosity elicit a greater response. The result is a high blood level and prompt death at doses otherwise tolerated by ingestion without aspiration. In general the secondary alcohols are less toxic than the corresponding primary isomers. As a general observation, alcohols are more powerful central nervous system depressants than their aliphatic analogues. In sequence of decreasing depressant potential, tertiary alcohols with multiple substituent OH groups are more potent than secondary alcohols, which, in turn, are more potent than primary alcohols. The potential for overall systemic toxicity increases with molecular weight (up to C7), principally because the water solubility is diminished and lipophilicity is increased. Within the homologous series of aliphatic alcohols, narcotic potency may increase even faster than lethality Only scanty toxicity information is available about higher homologues of the aliphatic alcohol series (greater than C7) but animal data establish that lethality does not continue to increase with increasing chain length. Aliphatic alcohols with 8 carbons are less toxic than those immediately preceding them in the series. 10 -Carbon n-decyl alcohol has low toxicity as do the solid fatty alcohols (e.g. lauryl, myristyl, cetyl and stearyl). However the rat aspiration test suggests that decyl and melted dodecyl (lauryl) alcohols are dangerous if they enter the trachea. In the rat even a small quantity (0.2 ml) of these behaves like a hydrocarbon solvent in causing death from pulmonary oedema. Primary alcohols are metabolised to corresponding aldehydes and acids; a significant metabolic acidosis may occur. Secondary alcohols are converted to ketones, which are also central nervous system depressants and which, in he case of the higher homologues persist in the blood for many hours. Tertiary alcohols are metabolised slowly and incompletely so their toxic effects are generally persistent. Ingestion of propylene glycol produced reversible central nervous system depression in humans following ingestion of 60 ml. Symptoms included increased heart-rate (tachycardia), excessive sweating (diaphoresis) and grand mal seizures in a 15 month child who ingested

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	large doses (7.5 ml/day for 8 days) as an ingredient of vitamin preparation. Excessive repeated ingestions may cause hypoglycaemia (low levels of glucose in the blood stream) among susceptible individuals; this may result in muscular weakness, incoordination and mental confusion. Very high doses given during feeding studies to rats and dogs produce central nervous system depression (although one-third of that produced by ethanol), haemolysis and insignificant kidney changes. In humans propylene glycol is partly excreted unchanged in the urine and partly metabolised as lactic and pyruvic acid. Lactic acidosis may result.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. 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. A single prolonged exposure is not likely to result in the material being absorbed in harmful amounts. However the material may be absorbed in potentially harmful amounts when applied in large quantities to severe burns (second or third degree) over large areas of the body as part of a cream, other topical application or by prolonged contact with clothing accidentally wetted by the material. Absorption under such circumstances can elevated serum osmolality and may result in osmotic shock. Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man.
Eye	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 temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Propylene glycol is though, by some, to be a sensitising principal following the regular use of topical creams by eczema patients. A study of 866 persons using a formulation containing propylene glycol in a patch test indicated that propylene glycol caused primary irritation in 16% of exposed individuals probably caused by dehydration. Undiluted propylene glycol was tested on 1556 persons in a 24 hour patch test. 12.5% showed reactions which were largely toxic (70%) or allergic in nature (30%). Reaction responses reached their maximum on the second day or later. Reactions were seasonal in nature ranging from 17.8% in winter to 9.2% in other seasons. In a patch-test using 25 standard allergens conducted on 500 individuals, propylene glycol ranked fourth in sensitising response. 84 subjects were patch tested using 100% propylene glycol. as well as 2% and 5% in water. With undiluted material, 15% demonstrated a reaction, with 40% of the reactions being allergic in nature and 60% being irritant. In dilute solutions 5 of 248 subjects exhibited a reaction. Undiluted propylene glycol tested on the skin of man produced no irritation under open conditions but when applied under occlusive conditions, for 2 weeks, it produced severe erythema, oedema and vesicles, probably due to sweat retention and weak primary irritation. Predictive contact skin sensitisation tests indicate that propylene glycol is an intermediate grade sensitiser with an index of 1% of tested subjects. Groups of cats fed 5 gm/kg/day of propylene glycol for 14 weeks showed a significant dose-related increase in red blood cell Heinz body formation without any marked signs of haemolytic anaemia. The no-effect-level for cats without formation of Heinz bodies is 100-500 ml/kg. There is no evidence of anaemia or degenerative change. Groups of rats dosed orally with 0.5 or 10 mg/kg/day for 12 weeks had lowered food intake but no adverse effects on body weights. Erythrocytes were more fragile. Heinz bodies were not apparent.

Moxistar LV Se Low Volume Oral Drench for Sheep with Selenium	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

propylene glycol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 11890 mg/kg[2]	Eye (rabbit): 100 mg - mild
	Inhalation (Rat) LC50: >44.9 mg/l4h[1]	Eye (rabbit): 500 mg/24h - mild
	Oral (Rat) LD50: 20000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin(human):104 mg/3d Intermit Mod
		Skin(human):500 mg/7days mild
		Skin: no adverse effect observed (not irritating)[1]

benzyl alcohol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 2000 mg/kg[2]	Eye (rabbit): 0.75 mg open SEVERE
	Inhalation (Rat) LC50: >4.178 mg/L4h[2]	Eye: adverse effect observed (irritating)[1]
	Oral (Rat) LD50: 1230 mg/kg[2]	Skin (man): 16 mg/48h-mild
		Skin (rabbit):10 mg/24h open-mild
		Skin: no adverse effect observed (not irritating)[1]

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moxidectin	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye (rabbit): slight irritant *(recovery within 72 hours)
	Oral (Mouse) LD50; 42 mg/kg[2]	Skin (rabbit): non-irritant *
sodium selenate, anhydrous	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: >0.052<=0.51 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: 1.6 mg/kg[2]	Skin: adverse effect observed (irritating)[1]

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

PROPYLENE GLYCOL	The acute oral toxicity of propylene glycol is very low, and large quantities are required to cause perceptible health damage in humans. Serious toxicity generally occurs only at plasma concentrations over 1 g/L, which requires extremely high intake over a relatively short period of time. It would be nearly impossible to reach toxic levels by consuming foods or supplements, which contain at most 1 g/kg of PG. Cases of propylene glycol poisoning are usually related to either inappropriate intravenous administration or accidental ingestion of large quantities by children.The potential for long-term oral toxicity is also low. Because of its low chronic oral toxicity, propylene glycol was classified by the U. S. Food and Drug Administration as "generally recognized as safe" (GRAS) for use as a direct food additive. Prolonged contact with propylene glycol is essentially non-irritating to the skin. Undiluted propylene glycol is minimally irritating to the eye, and can produce slight transient conjunctivitis (the eye recovers after the exposure is removed). Exposure to mists may cause eye irritation, as well as upper respiratory tract irritation. Inhalation of the propylene glycol vapours appears to present no significant hazard in ordinary applications. However, limited human experience indicates that inhalation of propylene glycol mists could be irritating to some individuals It is therefore recommended that propylene glycol not be used in applications where inhalation exposure or human eye contact with the spray mists of these materials is likely, such as fogs for theatrical productions or antifreeze solutions for emergency eye wash stations. Propylene glycol is metabolised in the human body into pyruvic acid (a normal part of the glucose-metabolism process, readily converted to energy), acetic acid (handled by ethanol-metabolism), lactic acid (a normal acid generally abundant during digestion), and propionaldehyde (a potentially hazardous substance). Propylene glycol shows no evidence of being a carcinogen or of being genotoxic. Research has suggested that individuals who cannot tolerate propylene glycol probably experience a special form of irritation, but that they only rarely develop allergic contact dermatitis. Other investigators believe that the incidence of allergic contact dermatitis to propylene glycol may be greater than 2% in patients with eczema. One study strongly suggests a connection between airborne concentrations of propylene glycol in houses and development of asthma and allergic reactions, such as rhinitis or hives in children Another study suggested that the concentrations of PGEs (counted as the sum of propylene glycol and glycol ethers) in indoor air, particularly bedroom air, is linked to increased risk of developing numerous respiratory and immune disorders in children, including asthma, hay fever, eczema, and allergies, with increased risk ranging from 50% to 180%. This concentration has been linked to use of water-based paints and water-based system cleansers. Patients with vulvodynia and interstitial cystitis may be especially sensitive to propylene glycol. Women suffering with yeast infections may also notice that some over the counter creams can cause intense burning. Post menopausal women who require the use of an eostrogen cream may notice that brand name creams made with propylene glycol often create extreme, uncomfortable burning along the vulva and perianal area. Additionally, some electronic cigarette users who inhale propylene glycol vapor may experience dryness of the throat or shortness of breath . As an alternative, some suppliers will put Vegetable Glycerin in the "e-liquid" for those who are allergic (or have bad reactions) to propylene glycol. Adverse responses to intravenous administration of drugs which use PG as an excipient have been seen in a number of people, particularly with large dosages thereof. Responses may include "hypotension, bradycardia... QRS and T abnormalities on the ECG, arrhythmia, cardiac arrest, serum hyperosmolality, lactic acidosis, and haemolysis". A high percentage (12% to 42%) of directly-injected propylene glycol is eliminated/secreted in urine unaltered depending on dosage, with the remainder appearing in its glucuronide-form. The speed of renal filtration decreases as dosage increases, which may be due to propylene glycol's mild anesthetic / CNS-depressant -properties as an alcohol. In one case, intravenous administration of propylene glycol-suspended nitroglycerin to an elderly man may have induced coma and acidosis. Propylene glycol is an approved food additive for dog food under the category of animal feed and is generally recognized as safe for dogs with an LD50 of 9 mL/kg. The LD50 is higher for most laboratory animals (20 mL/kg) Similarly, propylene glycol is an approved food additive for human food as well. The exception is that it is prohibited for use in food for cats due to links to Heinz body anemia.
BENZYL ALCOHOL	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. Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and connubial contact dermatitis occur. Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes. Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits. Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water. Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis. Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a suffcient 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

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contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure.
Hands: Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema may often be complicated by sensitisation .Fragrance allergy may be a relevant problem in patients with hand eczema; perfumes are present in consumer products to which their hands are exposed. A significant relationship between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe) chronic hand eczema may not be clear.
Axillae Bilateral axillary (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy.
Face Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of of being fragrance allergic.
Irritant reactions (including contact urticaria): Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this, Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported . The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested , but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen.
Pigmentary anomalies: The term “pigmented cosmetic dermatitis” was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified.. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil.
Photo-reactions Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon . Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. Phototoxic reactions still occur but are rare.
General/respiratory: Fragrances are volatile and therefore, in addition to skin exposure, a perfume also exposes the eyes and naso-respiratory tract. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma . Asthma-like symptoms can be provoked by sensory mechanisms. In an epidemiological investigation, a significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.
Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A prehapten is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems. A prohapten 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 prehapten or as a prohapten, 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 sensitisers has not been studied in detail. Skin sensitising prohaptens can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity.
QSAR prediction: The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptens) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptens. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation
CYP1A2 is a member of the cytochrome P450 super family, is one of the best characterized. It is responsible for the metabolism of commonly drugs belonging to classes such as antidepressants, antipsychotics, mood stabilizers, beta blockers and sedative/hypnotics
CYP1A2 also metabolises a number of procarcinogens (such as those in cigarettes). Cigarette smoking may lead to three fold increase in 1A2 activity, which explains why smokers require higher doses of beta blockers than than non-smokers
Drugs that inhibit CYP1A2 will predictably increase the plasma concentrations of the medications or decrease in clearance of substrates. Drugs such as ciprofloxacin, fluvoxamine, verapamil cimetidine , caffeine and isoniazid are inhibitors of CYP1A2 enzyme. Vegetables such as grape fruit juice, cumic and turmeric are inhibitors of the CYP1A2 enzyme which may leads to increase plasma concentration of psychotropics
Inhibition of NF-kB in vivo can be detrimental. NF-kB controls multiple functions in homeostasis including a functional immune response, cell cycle, and cell death. Genetic studies in mice and analysis of naturally occurring mutations in humans point to specific developmental and immune consequences due to altering NF-kB activity.
The same functions that make NF-kB attractive for developing inhibitors for treating disease also play a role in homeostasis,and disruption of the NF-kB pathway during development or in adults leads to unfavorable and potentially unhealthy consequences.
NF-kB plays a role in multiple homeostatic cellular processes including response to stimuli,cell proliferation, and death, regulating communication between cells, but is also tightly linked with other signaling pathways within the cell, such a p38 and JNK. In addition to mediating proinflammatory responses, NF-kB may regulate apoptotic and cell cycle changes induced by cellular stress, DNA damage or oncogenes by communication with the tumor suppressor p53. Disruption of normal cellular responses by inhibiting NF-kB can have adverse consequences such as immune suppression and tissue damage.

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Understanding the consequences of lack of NF-kB activity in adult humans comes from observation of naturally occurring genetic deficiencies in this pathway. Mutations have been discovered in humans in signaling molecules upstream of NF-kB resulting in defects in development or immunity. Genetic defects have also been discovered in genes that immediately affect NF-kB activation including IKK gamma (NEMO), a subunit of the IKK complex, and IkBalpha.The IKK gamma mutations result in a defective IKK complex and the IkBalpha mutation results in an IkBalpha protein that cannot be phosphorylated and degraded. Both genetic defects result in suppressed NF-kB activation and ectodermal dysplasia with immunodeficiency.In general patients with these genetic defects have multiple immunological defects including impaired innate immunity,impaired antibody production, and ultimately severe bacterial infections. Understanding the immune defects and susceptibilities in patients with genetic defects in the NF-kB pathway will help prepare for potential adverse effects of pharmacologic NF-kB inhibitors
The requirement for NF-kB in the development and maintenance of the immune system is well documented. NF-kB is required for survival during fetal development and for normal lymphocyte generation in adult mice. Removal of the p65 (RelA) subunit of NF-kB or the IKKbeta gene results in death during fetal development primarily due to massive liver apoptosis
Fetal liver stem cells from p65 or IKKbeta deficient mice have been transplanted into irradiated hosts revealing a specific requirement of NF-kB for T-cells, B-cells, and common lymphoid progenitor development but not for myeloid cells or stem cells. The failure to produce lymphocytes is mediated through hypersensitivity to TNF due to lack of NF-kB activity. Lymphocyte depletion with chemical or genetic inhibition of NF-kB have implications for therapeutic potential use in humans. The double-sided nature of NF-kB inhibition is clear in this instance where chemical inhibition in vivo mimics genetic experiments inducing rapid TNF-dependent apoptosis. Rapid induction of apoptosis may be an advantage for treating some forms of cancer, but at the same time cause depletion of some lymphocyte populations.
In addition to controlling lymphocyte development, NF-kB plays a major role in both adaptive and innate immunity. Various signaling pathways responding to receptor recognition of immune challenge converge on NF-kB which then regulates genes that control the immune response. Both T-cell receptor and B-cell receptors activate NF-kB through phosphorylation of CARMA1 by PKC theta and PKC beta respectively, resulting in recruitment and activation of IKK and ultimately expression of genes that control cellular activation, proliferation, and survival. In addition, NF-kB plays a role in T-cell response to costimulatory signals. Cells respond to pathogenic microorganisms in part through recognition by Toll-like receptors (TLRs).TLR-family members recognize different molecular structures present in microbes and respond by activating signaling pathways including NF-kB leading to expression of anti-microbial effector molecules, as well as molecules that help in development of the adaptive immune response. Inhibition of NF-kB during TLR stimulation can lead to macrophage apoptosis, a mechanism used by some pathogens to help evade immune response. NF-kB is clearly required for normal mature B-cell and T-cell maintenance and function, including regulatory, memory, and natural killer-like T cells. Inhibition of NF-kB activation in lymphocytes results in defects in growth, survival, and cytokine production and blocks multiple steps in germinal center formation.Given the diverse roles NF-kB plays in immune response to pathogens it is not surprising to find mice genetically deficient in components of the NF-kB pathway are susceptible to parasitic and bacterial infection.
The role of NF-kB in inhibition of apoptosis is one of the factors that make it a potential target for cancer therapy. NF-kB deficient mice die during embryogenesis in part due to TNF-mediated liver damage. Adult mice with impaired NF-kB targeted to the liver have normal liver function, but have severe liver damage after challenge with concanavalin A, a pan-T cell activator.Liver damage occurs due to sustained activation of JNK due to accumulation of reactive oxygen species (ROS) in the absence of normal NF-kB activation.
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
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 derivate 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)
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

Continued...

	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.
MOXIDECTIN	May produce developmental toxicity in rat offspring at maternally toxic doses. This does not occur in rabbits. * * Cyanamid The ADI for Moxidectin is set at 0.01mg/kg/day. The corresponding NOEL is set at 1mg/kg/day. ADI means Acceptable Daily Intake and NOEL means No-observable-effect-level. In rats given oral doses of moxidectin, decreased activity, prostration, tremors, chromodacryorrhea, decreased respiration, diarrhoea, hypersensitivity to touch and sound, and epistaxis occurred. Congestion of the liver, kidneys and lungs were observed in animals that died, but animals which were sacrificed at the end of the 14-day observation period showed no abnormalities. No overt signs of toxicity were noted in rabbits treated dermally with moxidectin. Studies of moxidectin show the side effects vary by animal and may be affected by the product's formulation, application method and dosage. An overdose of moxidectin enhances the effect of gamma-aminobutyric acid (GABA) in the central nervous system. In horses, overdose may lead to depression, drooping of the lower lip, tremor, lack of coordination when moving (ataxia), decreased rate of breathing (respiratory rate), stupor and coma. If a dog licks moxidectin from the skin which was applied as a "spot-on" (topical) treatment, this has the same effect as an overdose, and may cause vomiting, salivation and neurological signs such as ataxia, tremor, and nystagmus Collie dogs cannot be administered moxidectin For avermectins: Technical avermectin exhibits high mammalian acute toxicity. In vertebrates, the effects occur via poisoning of the central nervous system (CNS) through reactions at the receptor for the inhibitory neurotransmitter GABA. The avermectins open the GABAA receptor chloride channel by binding to the GABA recognition site (receptor protein) and act as partial agonists.. Chloride ions then flow into the postsynaptic neuron. This chloride permeability increase can significantly hyperpolarize (make more negative) the membrane potential, which has a dampening effect on nerve impulse firing. There is also a reversible dose-dependent increase in chloride ion permeability in response to very low doses of avermectins. In GABA-insensitive neurons with no inhibitory innervation, the avermectins induce an irreversible increase in chloride ion conductance through interacting with voltage-dependent chloride channels. Avermectin intoxication in mammals begins with hyperexcitability, tremors, and incoordination and later develops into ataxia and coma-like sedation. This is similar to the mode of action of ethanol and barbiturates and benzodiazepine sedatives However, the avermectins are less specific in their action and can affect a variety of other ligand- and voltage-gated chloride channels. The general safety of the avermectins depends on the presence of an intact P-glycoprotein blood-brain barrier Avermectin 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 photodegradate 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. 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. 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 dot appear to produce birth defects. Abamectin is non-mutagenic in the Ames test and the micronucleus test. 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. 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).
SODIUM SELENATE, ANHYDROUS	Eye effects, general anaesthesia, convulsions, muscle weakness, spasticity, cardiac EKG changes, cyanosis, lung tumours, diarrhoea, impaired liver function tests, leumaemia, specific developmental changes, effects on newborn recorded. The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
PROPYLENE GLYCOL & BENZYL ALCOHOL	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

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***

Continued...

– Data available to make classification

SECTION 12 Ecological information

Toxicity

Moxistar LV Se Low Volume Oral Drench for Sheep with Selenium

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

propylene glycol

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	336h	Algae or other aquatic plants	<5300mg/l	1
EC50	96h	Algae or other aquatic plants	19000mg/l	2
EC50	72h	Algae or other aquatic plants	19300mg/l	2
EC50	48h	Crustacea	>114.4mg/L	4
LC50	96h	Fish	710mg/L	4

benzyl alcohol

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	500mg/l	2
LC50	96h	Fish	10mg/l	2
EC50	48h	Crustacea	230mg/l	2
NOEC(ECx)	336h	Fish	5.1mg/l	2
EC50	96h	Algae or other aquatic plants	76.828mg/l	2

moxidectin

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	0.001mg/L	Not Available
EC50	72h	Algae or other aquatic plants	>0.087mg/L	Not Available
EC50	48h	Crustacea	<0.001mg/L	Not Available
NOEC(ECx)	96h	Fish	<0.001mg/L	Not Available

sodium selenate, anhydrous

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	0.55-0.85mg/L	4
EC50	72h	Algae or other aquatic plants	15.57mg/L	2
EC50	48h	Crustacea	0.52-0.63mg/l	4
NOEC(ECx)	4320h	Fish	<0.005mg/l	2
EC50	96h	Algae or other aquatic plants	0.032mg/L	2

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

Toxic to aquatic organisms.
Harmful 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
propylene glycol	LOW	LOW
benzyl alcohol	LOW	LOW
sodium selenate, anhydrous	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
propylene glycol	LOW (BCF = 1)
benzyl alcohol	LOW (LogKOW = 1.1)
sodium selenate, anhydrous	LOW (LogKOW = -3.1818)

Mobility in soil

Ingredient	Mobility
propylene glycol	HIGH (Log KOC = 1)
benzyl alcohol	LOW (Log KOC = 15.66)
sodium selenate, anhydrous	LOW (Log KOC = 48.64)

SECTION 13 Disposal considerations

Continued...

Waste treatment methods

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

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
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

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

Not Applicable

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

Product name	Group
propylene glycol	Not Available
benzyl alcohol	Not Available
moxidectin	Not Available
sodium selenate, anhydrous	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
propylene glycol	Not Available
benzyl alcohol	Not Available
moxidectin	Not Available
sodium selenate, anhydrous	Not Available

SECTION 15 Regulatory information

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

propylene glycol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

benzyl alcohol is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

moxidectin is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
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
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7

sodium selenate, anhydrous is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

Continued...

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (moxidectin)
Canada - DSL	No (moxidectin)
Canada - NDSL	No (propylene glycol; benzyl alcohol; moxidectin; sodium selenate, anhydrous)
China - IECSC	No (moxidectin)
Europe - EINEC / ELINCS / NLP	No (moxidectin)
Japan - ENCS	No (moxidectin)
Korea - KECI	No (moxidectin)
New Zealand - NZIoC	Yes
Philippines - PICCS	No (moxidectin)
USA - TSCA	No (moxidectin)
Taiwan - TCSI	Yes
Mexico - INSQ	No (moxidectin)
Vietnam - NCI	No (moxidectin)
Russia - FBEPH	No (moxidectin; sodium selenate, anhydrous)
Legend:	***Yes = All CAS declared ingredients are on the inventory*** ***No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.***

SECTION 16 Other information

Revision Date	21/06/2024
Initial Date	21/06/2024

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	21/06/2024	Toxicological information - Acute Health (inhaled), Toxicological information - Acute Health (skin), Toxicological information - Acute Health (swallowed), First Aid measures - Advice to Doctor, Physical and chemical properties - Appearance, Toxicological information - Chronic Health, Hazards identification - Classification, Ecological Information - Environmental, Exposure controls / personal protection - Exposure Standard, First Aid measures - First Aid (eye), First Aid measures - First Aid (skin), Composition / information on ingredients - Ingredients, Exposure controls / personal protection - Personal Protection (other), Accidental release measures - Spills (major), Accidental release measures - Spills (minor), Handling and storage - Storage (storage requirement), Toxicological information - Toxicity and Irritation (Other), Identification of the substance / mixture and of the company / undertaking - Use

Other information

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

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

Definitions and abbreviations

- PC—TWA: Permissible Concentration-Time Weighted Average
- PC—STEL: Permissible Concentration-Short Term Exposure Limit
- IARC: International Agency for Research on Cancer
- ACGIH: American Conference of Governmental Industrial Hygienists
- STEL: Short Term Exposure Limit
- TEEL: Temporary Emergency Exposure Limit。
- IDLH: Immediately Dangerous to Life or Health Concentrations
- 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
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration

- 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

Continued...

- 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

Continued...

end of SDS