OzCrop Fipronil 200 SC Insecticide

OzCrop Pty Ltd

Chemwatch Hazard Alert Code: **2**

Chemwatch: **5667-33**
Version No: **4.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Issue Date: **26/04/2024**
Print Date: **13/06/2024**
S.GHS.AUS.EN.E

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

Product Identifier

Product name	OzCrop Fipronil 200 SC Insecticide
Chemical Name	Not Applicable
Synonyms	APVMA Approval No.: 94294
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains fipronil)
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	Agricultural Insecticide.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**OzCrop Pty Ltd**
Address	5.08, 12 Century Circuit Norwest NSW 2153 Australia
Telephone	+61 2 8123 0170
Fax	+61 2 8123 0171
Website	http://www.ozcrop.com.au
Email	enquiries@ozcrop.com.au

Emergency telephone number

Association / Organisation	**In Transport Emergency DIAL 000**
Emergency telephone numbers	1800 033 111 (24 hours - Australia wide)
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	S6
Classification [1]	Acute Toxicity (Oral) Category 4, Acute Toxicity (Dermal) Category 4, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2A, Acute Toxicity (Inhalation) Category 4, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Specific Target Organ Toxicity - Repeated Exposure Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 1
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI***

Label elements

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

Hazard statement(s)

H302	Harmful if swallowed.
H312	Harmful in contact with skin.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H332	Harmful if inhaled.
H335	May cause respiratory irritation.
H372	Causes damage to organs through prolonged or repeated exposure.

Page 1 continued...

H410	Very toxic to aquatic life with long lasting effects.
AUH019	May form explosive peroxides.

Precautionary statement(s) Prevention

P260	Do not breathe mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P280	Wear protective gloves, protective clothing, eye protection and face protection.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of water.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P330	Rinse mouth.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
120962-03-0	30-60	canola oil
120068-37-3	10-30	fipronil
61791-12-6	1-10	castor oil, hydrogenated, ethoxylated
68131-39-5	1-10	alcohols C12-15 ethoxylated
57-55-6	<1	propylene glycol
4719-04-4	<1	hexahydro-1,3,5-tris(hydroxyethyl)triazine
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: ▸ Immediately hold eyelids apart and flush the eye continuously with 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. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ 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, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY.** ▸ For advice, contact a Poisons Information Centre or a doctor. ▸ Urgent hospital treatment is likely to be needed.

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	▸ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. ▸ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist. ▸ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS. **Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:** ▸ **INDUCE** vomiting with fingers down the back of the throat, **ONLY IF CONSCIOUS**. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. **NOTE:** Wear a protective glove when inducing vomiting by mechanical means.

Indication of any immediate medical attention and special treatment needed

As in all cases of suspected poisoning, follow the ABCDEs of emergency medicine (airway, breathing, circulation, disability, exposure), then the ABCDEs of toxicology (antidotes, basics, change absorption, change distribution, change elimination).
For poisons (where specific treatment regime is absent):

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 pulmonary oedema.
- Monitor and treat, where necessary, for shock.
- Anticipate seizures.
- **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.

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.
- Drug therapy should be considered for pulmonary oedema.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Treat seizures with diazepam.
- Proparacaine hydrochloride should be used to assist eye irrigation.

BRONSTEIN, A.C. and CURRANCE, P.L.
EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994
Treat symptomatically.

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 full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ Avoid spraying water onto liquid pools. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	The emulsion is not combustible under normal conditions. However, it will break down under fire conditions and the hydrocarbon component will burn. Combustion products include: carbon dioxide (CO_2) acrolein hydrogen chloride phosgene hydrogen fluoride nitrogen oxides (NOx) sulfur oxides (SOx) silicon dioxide (SiO_2) other pyrolysis products typical of burning organic material. **CARE**: Water in contact with hot liquid may cause foaming and a steam explosion with wide scattering of hot oil and possible severe burns. Foaming may cause overflow of containers and may result in possible fire.
HAZCHEM	•3Z

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

Continued...

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. Slippery when spilt. ▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Control personal contact with the substance, by using protective equipment. ▸ Contain and absorb spill with sand, earth, inert material or vermiculite. ▸ Wipe up. ▸ Place in a suitable, labelled container for waste disposal.
Major Spills	Environmental hazard - contain spillage. Slippery when spilt. Moderate hazard. ▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ No smoking, naked lights or ignition sources. ▸ Increase ventilation. ▸ Stop leak if safe to do so. ▸ Contain spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Absorb remaining product with sand, earth or vermiculite. ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	Rags wet / soaked with unsaturated hydrocarbons / drying oils may auto-oxidise; generate heat and, in-time, smoulder and ignite. This is especially the case where oil-soaked materials are folded, bunched, compressed, or piled together - this allows the heat to accumulate or even accelerate the reaction Oily cleaning rags should be collected regularly and immersed in water, or spread to dry in safe-place away from direct sunlight or stored, immersed, in solvents in suitably closed containers. ▸ **DO NOT** allow clothing wet with material to stay in contact with skin ▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ Avoid smoking, naked lights or ignition sources. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ No smoking, naked lights or ignition sources. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ **DO NOT** use aluminium or galvanised containers ▸ Metal can or drum ▸ Packaging as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Polyol esters of fatty acids become unstable with water and high temperatures, and the instability is enhanced in the presence of alkaline substances. The presence of an alkali or acid results in the partial hydrolysis of fatty acids and the formation of free polyglycerol. ▸ Glycol ethers may form peroxides under certain conditions; the potential for peroxide formation is enhanced when these substances are used in processes such as distillation where they are concentrated or even evaporated to near-dryness or dryness; storage under a nitrogen atmosphere is recommended to minimise the possible formation of highly reactive peroxides ▸ Nitrogen blanketing is recommended if transported in containers at temperatures within 15 deg C of the flash-point and at or above the flash-point - large containers may first need to be purged and inerted with nitrogen prior to loading ▸ In the presence of strong bases or the salts of strong bases, at elevated temperatures, the potential exists for runaway reactions. ▸ Contact with aluminium should be avoided; release of hydrogen gas may result- glycol ethers will corrode scratched aluminium surfaces. ▸ May discolour in mild steel/ copper; lined containers, glass or stainless steel is preferred ▸ 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 . Investigation of the hazards associated with use of 2-butoxyethanol for alloy electropolishing showed that mixtures with 50-95% of acid at 20 deg C, or 40-90% at 75 C, were explosive and initiable by sparks. Sparking caused mixtures with 40-50% of acid to become explosive, but 30% solutions appeared safe under static conditions of temperature and concentration.

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	▸ Avoid strong acids, bases. · Materials soaked with plant/ vegetable derived (and rarely, animal) oils may undergo spontaneous combustion · The more unsaturated is the fatty acid component, the more susceptible is the oil to oxidation and spontaneous combustion. · Many vegetable and animal oils absorb oxygen from the air to form oxidation products. This oxidation process produces heat and the resultant increase in temperature accelerates the oxidation process. · Drying oils such as linseed, tung, poppy and sunflower oils and semi-drying oils such as soya bean, tall oil, corn, cotton and castor oils all absorb oxygen readily and thus experience the self-heating process. · Cotton fibres are readily ignited and if contaminated with an oxidisable oil, may ignite unless heat can be dissipated · Vegetable oils and some animal fats undergo undesirable deterioration reactions in the presence of oxygen from the air becoming rancid accompanying off-flavours and smells. The mechanism of autoxidation of vegetable oils is classically regarded as following a number of stages being: · a usually slow initiation phase · a usually rapid propagation · and a termination phase The initiation phase involves the formation of a free radical from a triglyceride molecule in the fat: this may be promoted by the presence of heavy metals in the oil, or by heat or light. The next stage is the reaction of the triglyceride free radical with oxygen to produce a peroxide free radical, which can react with another triglyceride to produce a hydroperoxide and another triglyceride free radical. Steps 2 and 3 can repeat in a chain reaction until two peroxy free radicals collide and neutralise each other. Some drying oils produce cyclic peroxides instead of hydroperoxides. Autooxidation may also occur in saturated fatty acids and their esters. Monohydroperoxides are formed. Although all carbon atoms are subject to oxidation, preferential oxidation appears to occur towards the centre of the molecule. Autoxidation is assisted by higher ambient temperatures (the rate doubling for every ten degrees Centigrade rise) and by the presence of heavy metal ions, especially copper. The degree of unsaturation of the oil is also relevant to shelf-life; oils with a high linolenic fatty acid content (3 double bonds) being more prone that those with a higher saturated fatty acid content. Autoxidation can be minimized by the presence of anti-oxidants, which can act as free-radical inhibitors. Vegetable oils should therefore be stored in a cool place away from heat and light, and should only come into contact with inert (glass of stainless steel) containers which will not leach heavy metals. Blanketing under nitrogen should be considered in bulk storages.

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: particulates only	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	propylene glycol	Propane-1,2-diol total: (vapour & particulates)	150 ppm / 474 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
hexahydro-1,3,5-tris(hydroxyethyl)triazine	2.3 mg/m3	25 mg/m3	150 mg/m3

Ingredient	Original IDLH	Revised IDLH
canola oil	Not Available	Not Available
fipronil	Not Available	Not Available
castor oil, hydrogenated, ethoxylated	Not Available	Not Available
alcohols C12-15 ethoxylated	Not Available	Not Available
propylene glycol	Not Available	Not Available
hexahydro-1,3,5-tris(hydroxyethyl)triazine	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
canola oil	E	≤ 0.1 ppm
fipronil	C	> 0.1 to ≤ milligrams per cubic meter of air (mg/m³)
castor oil, hydrogenated, ethoxylated	E	≤ 0.1 ppm
alcohols C12-15 ethoxylated	E	≤ 0.1 ppm
hexahydro-1,3,5-tris(hydroxyethyl)triazine	E	≤ 0.1 ppm
Notes:	***Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.***	

Exposure controls

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

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An approved self contained breathing apparatus (SCBA) may be required in some situations.
Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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

Within each range the appropriate value depends on:

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

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

Requirements of State Authorities concerning conditions for tank entry must be met. Particularly with regard to training of crews for tank entry; work permits; sampling of atmosphere; provision of rescue harness and protective gear as needed

Individual protection measures, such as personal protective equipment

Eye and face protection

- Safety glasses with side shields.
- Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]
- 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

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

NOTE:

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

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
· frequency and duration of contact,
· chemical resistance of glove material,
· glove thickness and
· dexterity
Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
· Contaminated gloves should be replaced.
As defined in ASTM F-739-96 in any application, gloves are rated as:
· Excellent when breakthrough time > 480 min
· Good when breakthrough time > 20 min
· Fair when breakthrough time < 20 min
· Poor when glove material degrades
For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.
It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.
Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.
Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:
· Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.

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	· 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. ▸ Neoprene rubber gloves
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:
OzCrop Fipronil 200 SC Insecticide

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

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 5 x ES	A-AUS / Class 1 P2	-	A-PAPR-AUS / Class 1 P2
up to 25 x ES	Air-line*	A-2 P2	A-PAPR-2 P2
up to 50 x ES	-	A-3 P2	-
50+ x ES	-	Air-line**	-

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

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	White to yellow liquid; mixes with water.

Physical state	Liquid	**Relative density (Water = 1)**	1.03
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Applicable
pH (as supplied)	6-9	**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)	>100	**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.

Continued...

	▸ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. The material can cause respiratory irritation in some persons. The body's response to such irritation can cause further lung damage. Inhalation hazard is increased at higher temperatures. Inhalation of oil droplets or aerosols may cause discomfort and may produce chemical inflammation of the lungs. Fine mists generated from plant/ vegetable (or more rarely from animal) oils may be hazardous. Extreme heating for prolonged periods, at high temperatures, may generate breakdown products which include acrolein and acrolein-like substances.
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. The material may simulate the actions of GABA (gamma-aminobutyric acid), a major inhibitory neurotransmitter of the brain. Hence it inhibits the electrical activity of certain parts of the nervous systems. Certain substances similar to GABA may produce lightheadedness, inco-ordination, and mood elevation.
Skin Contact	Skin contact with the material may be harmful; systemic effects may result following absorption. The material may cause moderate inflammation of the skin either following direct contact or after a delay of some time. Repeated exposure can cause contact dermatitis which is characterised by redness, swelling and blistering. Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Non-ionic surfactants cause less irritation than other surfactants as they have less ability to denature protein in the skin. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream, through, for example, cuts, abrasions or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals. Prolonged eye contact may cause inflammation characterised by a temporary redness of the conjunctiva (similar to windburn). Non-ionic surfactants can cause numbing of the cornea, which masks discomfort normally caused by other agents and leads to corneal injury. Irritation varies depending on the duration of contact, the nature and concentration of the surfactant.
Chronic	Long-term exposure to respiratory irritants may result in airways disease, involving difficulty breathing and related whole-body problems. Skin contact with the material is more likely to cause a sensitisation reaction in some persons compared to the general population. Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. This material can cause serious damage if one is exposed to it for long periods. It can be assumed that it contains a substance which can produce severe defects. There has been some concern that this material can cause cancer or mutations but there is not enough data to make an assessment. Based on experience with similar materials, there is a possibility that exposure to the material may reduce fertility in humans at levels which do not cause other toxic effects. Based on experience with animal studies, there is a possibility that exposure to the material may result in toxic effects to the development of the foetus, at levels which do not cause significant toxic effects to the mother. Some glycol esters and their ethers cause wasting of the testicles, reproductive changes, infertility and changes to kidney function. Shorter chain compounds are more dangerous. Glyceryl triesters (triglycerides) undergo metabolism to become free fatty acids and glycerol. Animal studies show that there is no toxicity when given by mouth unless the material takes up a large proportion of energy intake. Prolonged or repeated skin contact may cause degreasing, followed by drying, cracking and skin inflammation. Common side effects of treatment with HIV-I protease inhibitors (PI) include diarrhoea, nausea, vomiting, gastrointestinal discomfort, headache, weakness, fatigue and taste disturbances. Renal stones are seen occasionally. Alpha-linolenic acid (ALA) is metabolised to precursors of substances that have an effect of countering inflammation and formation of plaques in arteries. Interactions may occur between ALA and aspirin, other non-steroid anti-inflammatory drugs, and some herbs including garlic and ginkgo. Such interactions might include nosebleeds and easy bruising. If these occur, consider lowering or stopping intake. Repeated swallowing of linoleic acid may alter platelet function. Animal testing showed weight loss, anaemia, loss of white cells and damage to the membrane of red and white cells.

OzCrop Fipronil 200 SC Insecticide	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
canola oil	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
fipronil	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 354 mg/kg[2]	Eye: slight *[** = Aventis]
	Inhalation (Rat) LC50: 0.001 mg/L4h[2]	Skin: non-irritating *
	Oral (Rat) LD50: 97 mg/kg[2]	
castor oil, hydrogenated, ethoxylated	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >20000 mg/kg[2]	Skin (human): non irritant * [BASF]
		Skin: no adverse effect observed (not irritating)[1]
alcohols C12-15 ethoxylated	**TOXICITY**	**IRRITATION**

Continued...

	Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: 1600 mg/kg[2]	Eye: SEVERE *
		Skin: no adverse effect observed (not irritating)[1]
		Skin: slight
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]
hexahydro-1,3,5-tris(hydroxyethyl)triazine	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[2]	Eye (rabbit): slight (OECD 405) [Manufacturer 2]
	Inhalation (Rat) LC50: 0.338 mg/l4h[1]	Eye (rabbit):moderate to SEVERE
	Oral (Mouse) LD50; 1.99 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
		Skin (rabbit): 0.15 mg/3d-I-mild
		Skin (rabbit):not irritating(OECD 403)
		Skin: no adverse effect observed (not irritating)[1]

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

CANOLA OIL

Polyunsaturated fats (PUFAs) protect against heart disease by providing more membrane fluidity than monounsaturated fats (MUFAs), but they are more vulnerable to being oxidized and therefore rancid.
Foods containing monounsaturated fats reduce low-density lipoprotein (LDL) cholesterol, while possibly increasing high-density lipoprotein (HDL) cholesterol.
Levels of oleic, and other monounsaturated fatty acids in red blood cell membranes were positively associated with breast cancer risk. In children, consumption of monounsaturated oils is associated with healthier blood lipid profiles.
The diet in Mediterranean countries consists of more total fat than the diets of Northern European countries, but most of the fat is made up of monounsaturated fatty acids from olive oil and omega-3 fatty acids (PUFAs) from fish and vegetables, and very little saturated fat.
For aliphatic fatty acids (and salts)
Acute oral (gavage) toxicity:
The acute oral LD50 values in rats for both were greater than >2000 mg/kg bw Clinical signs were generally associated with poor condition following administration of high doses (salivation, diarrhoea, staining, piloerection and lethargy).There were no adverse effects on body weight in any study In some studies, excess test substance and/or irritation in the gastrointestinal tract was observed at necropsy.
Skin and eye irritation potential, with a few stated exceptions, is chain length dependent and decreases with increasing chain length
According to several OECD test regimes the animal skin irritation studies indicate that the C6-10 aliphatic acids are severely irritating or corrosive, while the C12 aliphatic acid is irritating, and the C14-22 aliphatic acids generally are not irritating or mildly irritating.
Human skin irritation studies using more realistic exposures (30-minute,1-hour or 24-hours) indicate that the aliphatic acids have sufficient, good or very good skin compatibility.
Animal eye irritation studies indicate that among the aliphatic acids, the C8-12 aliphatic acids are irritating to the eye while the C14-22 aliphatic acids are not irritating.
Eye irritation potential of the ammonium salts does not follow chain length dependence; the C18 ammonium salts are corrosive to the eyes.
Dermal absorption:
The in vitro penetration of C10, C12, C14, C16 and C18 fatty acids (as sodium salt solutions) through rat skin decreases with increasing chain length. At 86.73 ug C16/cm2 and 91.84 ug C18/cm2, about 0.23% and less than 0.1% of the C16 and C18 soap solutions is absorbed after 24 h exposure, respectively.
Sensitisation:
No sensitisation data were located.
Repeat dose toxicity:
Repeated dose oral (gavage or diet) exposure to aliphatic acids did not result in systemic toxicity with NOAELs greater than the limit dose of 1000 mg/kg bw. .
Mutagenicity
Aliphatic acids do not appear to be mutagenic or clastogenic in vitro or in vivo
Carcinogenicity
No data were located for carcinogenicity of aliphatic fatty acids.
Reproductive toxicity
No effects on fertility or on reproductive organs, or developmental effects were observed in studies on aliphatic acids and the NOAELs correspond to the maximum dose tested. The weight of evidence supports the lack of reproductive and developmental toxicity potential of the aliphatic acids category.

Given the large number of substances in this category, their closely related chemical structure, expected trends in physical chemical properties, and similarity of toxicokinetic properties, both mammalian and aquatic endpoints were filled using read-across to the closest structural analogue, and selecting the most conservative supporting substance effect level.
Structure-activity relationships are not evident for the mammalian toxicity endpoints.That is, the low mammalian toxicity of this category of substances limits the ability to discern structural effects on biological activity. Regardless, the closest structural analogue with the most conservative effect value was selected for read across. Irritation is observed for chain lengths up to a cut-off" at or near 12 carbons).
Metabolism:
The aliphatic acids share a common degradation pathway in which they are metabolized to acetyl-CoA or other key metabolites in all living systems. Common biological pathways result in structurally similar breakdown products, and are, together with the physico-chemical properties, responsible for similar environmental behavior and essentially identical hazard profiles with regard to human health.
Differences in metabolism or biodegradability of even and odd numbered carbon chain compounds or saturated/ unsaturated compounds are not expected; even-and odd-numbered carbon chain compounds, and the saturated and unsaturated compounds are naturally occurring and are expected to be metabolized and biodegraded in the same manner.
The acid and alkali salt forms of the homologous aliphatic acid are expected to have many similar physicochemical and toxicological properties when they become bioavailable; therefore,data read across is used for those instances where data are available for the acid

Continued...

form but not the salt, and vice versa. In the gastrointestinal tract, acids and bases are absorbed in the undissociated (non-ionised) form by simple diffusion or by facilitated diffusion. It is expected that both the acids and the salts will be present in (or converted to) the acid form in the stomach. This means that for both aliphatic acid or aliphatic acid salt,the same compounds eventually enter the small intestine, where equilibrium, as a result of increased pH, will shift towards dissociation (ionised form).
Hence, the situation will be similar for compounds originating from acids and therefore no differences in uptake are anticipated
Note that the saturation or unsaturation level is not a factor in the toxicity of these substances and is not a critical component of the read across process..
Toxicokinetics:
The turnover of the [14C] surfactants in the rat showed that there was no significant difference in the rate or route of excretion of 14C given by intraperitoneal or subcutaneous administration. The main route of excretion was as 14CO2 in the expired air at 6 h after administration. The remaining material was incorporated in the body. Longer fatty acid chains are more readily incorporated than shorter chains. At ca. 1.55 and 1.64 mg/kg bw, 71% of the C16:0 and 56% of the C18:0 was incorporated and 21% and 38% was excreted as 14CO2, respectively.

Glycidyl fatty acid esters (GEs), one of the main contaminants in processed oils, are mainly formed during the deodorisation step in the refining process of edible oils and therefore occur in almost all refined edible oils. GEs are potential carcinogens, due to the fact that they readily hydrolyze into the free form glycidol in the gastrointestinal tract, which has been found to induce tumours in various rat tissues. Therefore, significant effort has been devoted to inhibit and eliminate the formation of GEs
GEs contain a common terminal epoxide group but exhibit different fatty acid compositions. This class of compounds has been reported in edible oils after overestimation of 3-monochloropropane-1,2-diol (3-MCPD) fatty acid esters analysed by an indirect method , 3-MCPD esters have been studied as food processing contaminants and are found in various food types and food ingredients, particularly in refined edible oils. 3-Monochloropropane-1,2-diol (3-MCPD) and 2-monochloropropane-1,3-diol (2-MCPD) are chlorinated derivatives of glycerol (1,2,3-propanetriol). 3- and 2-MCPD and their fatty acid esters are among non-volatile chloropropanols, Glycidol is associated with the formation and decomposition of 3- and 2-MCPD. It forms monoesters with fatty acids (GE) during the refining of vegetable oils.
Chloropropanols are formed in HVP during the hydrochloric acid-mediated hydrolysis step of the manufacturing process. In food production, chloropropanols form from the reaction of endogenous or added chloride with glycerol or acylglycerol.
Although harmful effects on humans and animals have not been demonstrated, the corresponding hydrolysates, 3-MCPD and glycidol, have been identified as rodent genotoxic carcinogens, ultimately resulting in the formation of kidney tumours (3-MCPD) and tumours at other tissue sites (glycidol). Therefore, 3-MCPD and glycidol have been categorised as ‘‘possible human carcinogens (group 2B) and “probably carcinogenic to humans (group 2A), respectively, by the International Agency for Research on Cancer (IARC).
Diacylglyceride (DAG) based oils produced by one company were banned from the global market due to “high levels” of GEs.
Several reports have also suggested that a bidirectional transformation process may occur not only between glycidol and 3-MCPD but also their esterified forms in the presence of chloride ions. The transformation rate of glycidol to 3-MCPD was higher than that of 3-MCPD to glycidol under acidic conditions in the presence of chloride ion.
Precursors of GEs in refined oils have been identified as partial acylglycerols, that is, DAGs and monoacylglycerides (MAGs); however, whether they also originate from triacylglycerides (TAGs) is still a topic of controversial debates. Several authors noted that pure TAGs were stable during heat treatment (such as 235 deg C) for 3 h and were therefore not involved in the formation of GEs. However, experimental results have shown that small amounts of GEs are present in a heat-treated oil model consisting of almost 100% TAGs. The formation of GEs from TAGs can be attributed to the pyrolysis of TAGs to DAGs and MAGs. In contrast, 3-MCPD esters in refined oils can be obtained from TAG . Presently, the mechanism for the formation of GE intermediates and the relationship between GEs and 3-MCPD esters are still unknown.
A high consumption of oxidised polyunsaturated fatty acids (PUFAs), which are found in most types of vegetable oil, may increase the likelihood that postmenopausal women will develop breast cancer. Similar effect was observed on prostate cancer, but the study was performed on mice Another "analysis suggested an inverse association between total polyunsaturated fatty acids and breast cancer risk, but individual polyunsaturated fatty acids behaved differently [from each other]. [...] a 20:2 derivative of linoleic acid [...] was inversely associated with the risk of breast cancer"
PUFAs are prone to spontaneous oxidation/ peroxidation. The feeding of lipid oxidation products and oxidised fats has been reported to cause adverse biological effects on laboratory animals, including growth retardation, teratogenicity, tissue damage and increased liver and kidney weights, as well as cellular damage to the testes and epididymes, increased peroxidation of membrane and tissue lipids and induction of cytochrome P450 activities in the colon and liver.
The propensity for PUFAs to oxidise leads to the generation of free radicals and eventually to rancidity.
Culinary oils, when heated, undergo important chemical reaction involving self-sustaining, free radical-mediated oxidative deterioration of PUFAs. Such by-products may be cytotoxic, mutagenic, reproductive toxins and may produce chronic disease. Samples of repeatedly used oils collected from fast-food retail outlets and restaurants have confirmed the production of aldehydic lipid oxidation products (LOPs) at levels exceeding 10 exp-2 moles per kilogram (mol/kg) during “on-site” frying episodes. Volatile emissions from heated culinary oils used in Chinese-style cooking are mutagenic; exposure to such indoor air pollution may render humans more susceptible to contracting lung or further cancers, together with rhinitis and diminished lung function. The high temperatures used in standard (especially Chinese) frying result in fumes that are rich in volatile LOPs, including acrolein.
The end products of lipid peroxidation are reactive aldehydes, such as malondialdehyde (MDA) and 4-hydroxynonenal (HNE), the second one being known also as "second messenger of free radicals" and major bioactive marker of lipid peroxidation, due to its numerous biological activities resembling activities of reactive oxygen species. end-products of lipid peroxidation may be mutagenic and carcinogenic malondialdehyde reacts with deoxyadenosine and deoxyguanosine in DNA, forming DNA adducts. Malondialdehyde produces mutagenic effects in several bioassays.
Side products of lipid peroxidation can also exert toxic effects, even at sites distant from the primary oxidation site. Such products (typically malondialdehyde and a large group of hydroxyalkenals - alpha-beta-unsaturated aldehydes) may interact with protein thiols (producing intermolecular cross-links) and, as a result produce functional impairment to enzyme systems, receptors and structural proteins. Aldehydes may also inhibit protein biosynthesis and increase osmotic fragility of lysosymes (releasing hydrolytic enzymes) and other subcellular organelles. They may also react with nucleic acids.
The toxicity of lipid hydroperoxides to animals is best illustrated by the lethal phenotype of glutathione peroxidase 4 (GPX4) knockout mice. These animals do not survive past embryonic day 8, indicating that the removal of lipid hydroperoxides is essential for mammalian life.
Peroxidised linoleic acid applied to the shaved skin of guinea pigs, in a patch test experiment, produced necrosis and bleeding. When the abdominal skin of guinea pig was patched for 8 days with a cream containing 25 nmol (in terms of malondialdehyde) of lipid peroxides per gram, a thickening of the epidermis was found
Lipid peroxidation in cellular membranes may produce several morphological alterations resulting, for example, in membrane aggregation, deformation or breakage. This may result in the release of hydrolytic enzymes which in turn may degrade functional macromolecules and cause secondary damage. In addition membrane-bound enzyme systems may be disrupted.
Epoxidation of double bonds is a common bioactivation pathway for alkenes. The allylic epoxides formed were found to be sensitizing. Research has shown that conjugated dienes in or in conjunction with a six-membered ring are prohaptens, while related dienes containing isolated double bonds or an acrylic conjugated diene were weak or non-sensitising.
For polyunsaturated fatty acids and oils (triglycerides):
Animal studies have shown a link between polyunsaturated fat and the incidence of tumours, which increased with increasing intake of polyunsaturated fats. This may be partly due to the propensity for polyunsaturated fats to oxidize, leading to generation of free radicals. Research evidence shows that consuming high amounts of polyunsaturated fat may increase the risk of cancer spreading.
Culinary oils, when heated, leads to self-sustaining oxidation f polyunsaturated fatty acids (PUFAs), which may produce oxidation products that are toxic to the cell and reproduction and which may cause mutations and chronic disease.
Samples of repeatedly used oils collected from fast-food retail outlets and restaurants have confirmed the production of aldehydic lipid oxidation products (LOPs) during frying. Volatile emissions from heated culinary oils used in Chinese-style cooking may cause mutations; exposure to such indoor air pollution may make humans more susceptible to contracting lung or other cancers, together with inflammation of the nose, and reduced lung function. The high temperatures used in standard (especially Chinese) frying result in fumes that are rich in volatile LOPs, including acrolein. Shallow frying appears to generate more LOPs than deep frying.
Birth defects: Animal testing shows that LOPs increase the rate of birth defects, whether or not the mother had diabetes. Further investigation reveals that safflower oil subjected to high temperatures markedly increased its propensity to increase birth defects.

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	Further adverse health effects of LOPs in the diet: Animal testing shows that other documented effects of LOPs include peptic ulcer and high blood pressure. Atherosclerosis: Investigations have revealed that LOPs derived from the diet can accelerate all three stages of the development of atherosclerosis, including endothelial injury, accumulation of plaque, and thrombosis. Mutation- and cancer-causing potential: Since they are powerful alkylating agents, alpha,beta-unsaturated aldehydes can covalently modify DNA base units and therefore cause mutations. These LOPs can inactivate DNA replicating systems, a process that can increase the extent of DNA damage. Malondialdehyde (MDA) is also generated by thermally stressing culinary oils, although at lower concentrations than alpha,beta-unsaturated aldehydes. MDA and other aldehydes arising from lipid peroxidation (especially acrolein) present a serious cancer hazard. The most obvious solution to the generation of LOPs in culinary oils during frying is to avoid consuming food in PUFA-rich oils as much as possible. Consumers and those involved in the fast-food industry could employ culinary oils of only a low PUFA content, or monounsaturated fatty acids (MUFA) such as canola or olive oil, or coconut oils (a saturated fatty acid). Acrylamide (which can exert toxic effects on the nervous system and fertility, and may also cause cancer) can be generated when asparagines-rich foods are deep-fried in PUFA-rich oils. For triglycerides: Carboxylic acid esters will undergo enzymatic hydrolysis by ubiquitously expressed Gl esterases. The rate of hydrolysis is dependant on the structure of the ester, and may therefore be rapid or rather slow. Thus, due to hydrolysis, predictions on oral absorption based on the physico-chemical characteristics of the intact parent substance alone may no longer apply. When considering the hydrolysis product glycerol, absorption is favoured based on passive and active absorption of glycerol. The Cosmetic Ingredient Review (CIR) Expert Panel has issued three final reports on the safety of 25 triglycerides, i.e., fatty acid triesters of glycerin High purity is needed for the triglycerides. Previously the Panel published a final report on a diglycerides, and concluded that the ingredients in the diglyceride family are safe in the present practices of use and concentration provided the content of 1,2-diesters is not high enough to induce epidermal hyperplasia. The Panel discussed that there was an increased level of concern because of data regarding the induction of protein kinase C (PKC) and the tumor promotion potential of 1,2-diacylglycerols. The Panel noted that, nominally, glyceryl-1,3-diesters contain 1,2-diesters, raising the concern that 1,2-diesters could potentially induce hyperplasia. The Panel did note that these compounds are more likely to cause these effects when the fatty acid chain length is <=14 carbons, when one fatty acid is saturated and one is not, and when given at high doses, repeatedly. Although minimal percutaneous absorption of triolein has been demonstrated in vivo using guinea pigs (but not hairless mice) and in vitro using full-thickness skin from hairless mice, the Expert Panel recognizes that, reportedly, triolein and tricaprylin can enhance the skin penetration of other chemicals, and recommends that care should be exercised in using these and other glyceryl triesters in cosmetic products. The Panel acknowledged that some of the triglycerides may be formed from plant-derived or animal-derived constituents. The Panel thus expressed concern regarding pesticide residues and heavy metals that may be present in botanical ingredients. They stressed that the cosmetics industry should continue to use the necessary procedures to sufficiently limit amounts of such impurities in an ingredient before blending them into cosmetic formulations. Additionally, the Panel considered the risks inherent in using animal-derived ingredients, namely the transmission of infectious agents. Although tallow may be used in the manufacture of glyceryl tallowate and is clearly animal-derived, the Panel notes that tallow is highly processed, and tallow derivatives even more so. The Panel agrees with determinations by the U.S. FDA that tallow derivatives are not risk materials for transmission of infectious agents. Finally, the Panel discussed the issue of incidental inhalation exposure, as some of the triglycerides are used in cosmetic sprays and could possibly be inhaled. For example, triethylhexanoin and triisostearin are reported to be used at maximum concentrations of 36% and 30%, respectively, in perfumes, and 14.7% and 10.4%, respectively, in face powders.The Panel noted that in aerosol products, 95% – 99% of droplets/particles would not be respirable to any appreciable amount. Furthermore, droplets/particles deposited in the nasopharyngeal or bronchial regions of the respiratory tract present no toxicological concerns based on the chemical and biological properties of these ingredients. Coupled with the small actual exposure in the breathing zone and the concentrations at which the ingredients are used, the available information indicates that incidental inhalation would not be a significant route of exposure that might lead to local respiratory or systemic effects Cosmetic Ingredient Review (CIR) : Amended Safety Assessment of Triglycerides as Used in Cosmetics August 2017 Glyceryl triesters are also known as triglycerides; ingested triglycerides are metabolized to monoglycerides, free fatty acids, and glycerol, all of which are absorbed in the intestinal mucosa and undergo further metabolism. Dermal absorption of Triolein in mice was nil; the oil remained at the application site. Only slight absorption was seen in guinea pig skin. Tricaprylin and other glyceryl triesters have been shown to increase the skin penetration of drugs. Little or no acute, subchronic, or chronic oral toxicity was seen in animal studies unless levels approached a significant percentage of caloric intake. Subcutaneous injections of Tricaprylin in rats over a period of 5 weeks caused a granulomatous reaction characterized by oil deposits surrounded by macrophages. Dermal application was not associated with significant irritation in rabbit skin. Ocular exposures were, at most, mildly irritating to rabbit eyes. No evidence of sensitization or photosensitization was seen in a guinea pig maximization test. Most of the genotoxicity test systems were negative. Tricaprylin, Trioctanoin, and Triolein have historically been used as vehicles in carcinogenicity testing of other chemicals. In one study, subcutaneous injection of Tricaprylin in newborn mice produced more tumors in lymphoid tissue than were seen in untreated animals, whereas neither subcutaneous or intraperitoneal injection in 4- to 6-week-old female mice produced any tumors in another study. Trioctanoin injected subcutaneously in hamsters produced no tumors. Trioctanoin injected intraperitoneally in pregnant rats was associated with an increase in mammary tumors in the offspring compared to that seen in offspring of untreated animals, but similar studies in pregnant hamsters and rabbits showed no tumors in the offspring. One study of Triolein injected subcutaneously in rats showed no tumors at the injection site. As part of an effort to evaluate vehicles used in carcinogenicity studies, the National Toxicology Program conducted a 2-year carcinogenicity study in rats given Tricaprylin by gavage. This treatment was associated with a statistically significant dose-related increase in pancreatic acinar cell hyperplasia and adenoma, but there were no acinar carcinomas, the incidence of mononuclear leukemia was less, and nephropathy findings were reduced, all compared to corn oil controls. Overall, the study concluded that Tricaprylin did not offer significant advantages over corn oil as vehicles in carcinogenicity studies. Trilaurin was found to inhibit the formation of neoplasms initiated by dimethylbenzanthracene (DMBA) and promoted by croton oil. Tricaprylin was not teratogenic in mice or rats, but some reproductive effects were seen in rabbits. A low level of fetal eye abnormalities and a small percentage of abnormal sperm were reported in mice injected with Trioctanoin as a vehicle control. Clinical tests of Trilaurin at 36.3% in a commercial product applied to the skin produced no irritation reactions. Trilaurin, Tristearin, and Tribehenin at 40%, 1.68%, and 0.38%, respectively, in commercial products were also negative in repeated-insult patch tests. Tristearin at 0.32% in a commercial product induced transient, mild to moderate, ocular irritation after instillation into the eyes of human subjects. Based on the enhancement of penetration of other chemicals by skin treatment with glyceryl triesters, it is recommended that care be exercised in using them in cosmetic products. Cosmetic Ingredient Review (CIR) Expert Panel: Final Report on the Safety Assessment of Trilaurin etc: Int J Toxicol, 20 Suppl 4, 61-94 2001 The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. For group E aliphatic esters (polyol esters): The polyol esters, including trimethylolpropane (TMP). Pentaerythritol (PE) and dipentaerythritol (diPE) are unique in their chemical characteristics since they lack beta-tertiary hydrogen atoms, thus leading to stability against oxidation and elimination. Therefore their esters with C5-C10 fatty acids have applications as artificial lubricants. Because of their stability at high temperatures, they are also used in high temperature applications such as industrial oven chain oils, high temperature greases, fire resistant transformer coolants and turbine engines. Polyol esters that are extensively esterified also have greater polarity, less volatility and enhanced lubricating properties. Acute toxicity: Animal studies show relatively low toxicity by swallowing. These esters are hydrolysed in the gastrointestinal tract, and studies have not shown evidence of these accumulating in body tissues. Acute toxicity by skin contact was also found to be low. Repeat dose toxicity: According to animal testing, polyol esters show a low level of toxicity following repeated application, either by swallowing or by skin contact. Reproductive and developmental toxicity: This group should not produce profound reproductive effects in animals. Genetic toxicity: Tests have shown this group to be inactive. It is unlikely that these substances cause mutations. Cancer-causing potential: No association between this group of substances and cancer.
FIPRONIL	Negative in Ames and chromosome aberration tests. * ADI: 0.0002 mg/kg/day NOEL: 0.02 mg/kg/day technical fipronil For fipronil

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	Acute toxicity: Clinical signs and symptoms reported after ingestion of fipronil by humans include sweating, nausea, vomiting, headache, abdominal pain, dizziness, agitation, weakness, and tonic clonic-seizures. Clinical signs of exposure to fipronil are generally reversible and resolve spontaneously Fipronil targets the nervous system. Signs of toxicity during an acute mouse feeding study with 87.4-97.2% fipronil included overactivity, irritability, convulsions, and death The primary metabolite of fipronil in army worms, mice, and humans is fipronil-sulfone, which binds to the GABA receptor with an affinity 6 times greater than the parent compound. Fipronil and its sulfone have similar toxicity in mammals; the mouse ip LD50 24 h after treatment is 41 and 50 mg/kg for fipronil and its sulfone, respectively. Fipronil-desulfinyl, the primary photoproduct in the environment, is 9-10 fold more potent and more acutely toxic than fipronil with an ip LD50 of 23 mg/kg in mice **Distribution:** After exposure fipronil is widely distributed in mammals and is found predominantly in fatty tissues. Rats given a single oral dose had the highest concentrations of fipronil in the stomach, GI tract, fat, and adrenals. Moderate levels were found in the liver, pancreas, thyroid, and ovaries. Low levels were present in the muscle, brain, heart, and cardiac blood . A spot-on treatment study with 14C-fipronil on dogs and cats reported radioactivity 2 months after treatment concentrated in the sebaceous glands, epithelial layers surrounding the hairs, and exposed part of the hair shaft, suggesting the passive diffusion of fipronil in the sebum covering hair and skin . Researchers applied a spot-on fipronil product to dogs and vigorously petted them for 5 minutes every day with cotton gloves to mimic normal exposures to treated animals. Residues transferred to the gloves peaked at 589+/-206 ppm fipronil 24 h after treatment, decreased steadily over time (448 +/- 118 ppm after 8 days) , and were undetectable after 36 days **Absorption:** In an ***in vitro*** study of 14C-fipronil absorption through human, rabbit, and rat epithelial membranes, researchers recorded penetration rates after 8 hours of 0.08% (rat), 0.07% (rabbit), and 0.01% (human) of the dose of 200 g/L fipronil solution. Researchers reported greater absorption from a 0.2 g/L solution of fipronil, with 0.9% (rat), 13.9% (rabbit), 0.9% (humans) of the dose being absorbed **Metabolism:** The whole-blood half-life of fipronil in rats ranged from about 6.2-8.3 days after a single 4 mg/kg oral dose and decreased significantly to 2.1-2.3 days after a single 150 mg/kg oral dose. The primary metabolite of fipronil in animals is the fipronil-sulfone derivative. Researchers injected mice with fipronil and detected the sulfone derivative in the brain, liver, kidney, fat, and faeces . Fipronil-desulfinyl, the primary photodegradate of fipronil, has been measured in the fat, brain, liver, kidney, skin, and feces of mice, rats and lactating goats after oral exposure or injection . **Excretion:** Rats given an oral dose of fipronil excreted 45-75% in the faeces and 5-25% in the urine. The parent compound and the oxidation product, fipronil-sulfone, were present in both. **Chronic toxicity:** Signs of toxicity during a chronic rat feeding study included reduced feeding, reduced body weight gain, seizures (including seizures resulting in death), alterations in thyroid hormones, and alterations in the mass and function of the liver, thyroid, and kidneys. No signs of systemic toxicity (NOEL) were observed in rats ingesting 0.5 ppm (0.019-0.025 mg/kg/day) during a 52-week chronic dietary study. The lowest dosage at which effects were observed (LOEL) was 1.5 ppm (0.059 mg/kg/day males, 0.078 mg/kg/day females), and included increased incidence of seizures and death, alteration in clinical chemistry (protein), and alterations in thyroid hormones **Carcinogenicity:** Mice given fipronil in their diet for 2 years showed no evidence of carcinogenicity at doses of 30 ppm .• Researchers administered fipronil in the diet of rats for 2 years. Carcinogenicity was observed at 12.68 mg/kg/day in males and 16.75 mg/kg/day in females based on an increased incidence of clinical signs and alterations in clinical chemistry and thyroid parameters. In one study, rats were fed 0, 0.5, 2, 6, and 10 ppm (0, 0.025, 0.098, and 0.050 mg/kg/day males, and 0, 0.032, 0.13, and 0.55 mg/kg/day females) fipronil-desulfinyl (the primary photodegradate), for 2 years. Male rats at 10 ppm and female rats at 2, 6, and 10 ppm developed clinical signs of toxicity with no evidence of carcinogenicity (13). The US EPA classified fipronil as a Group C (possible human) carcinogen, based on increased thyroid follicular cell tumors in both sexes of rats. **Mutagenicity**: Fipronil did not cause mutations in human lymphocytes, Chinese hamster V79 cells, salmonella (Ames test), or mouse micronuclei **Reproductive and developmental toxicity:** In one study with rats, no observable effects were recorded at 30 ppm (2.54 mg/kg/day in males, and 2.74 mg/kg/day in females; route of exposure not included). The lowest dosage at which reproductive effects were recorded was 300 ppm (26.0 mg/kg/day in males and 28.4 mg/kg/day in females; route of exposure not included) based on clinical signs of toxicity, decreased litter size, decreased body weights, decrease in percentage of animals mating, reduction in fertility index, reduced post-implantation survival and offspring postnatal survivability, and delay in physical development. Other experimental studies with ingestion of fipronil have not reported significant alterations on animal development. There were no observable adverse effects within the limits of two studies performed using rats and rabbits. The Lowest Observable Adverse Effect Levels (LOAELs) were the highest doses tested: .20 and .1.0 mg/kg/day in rats and rabbits, respectively ***[* The Pesticides Manual, Incorporating The Agrochemicals Handbook, 10th Edition, Editor Clive Tomlin, 1994, British Crop Protection Council]***
CASTOR OIL, HYDROGENATED, ETHOXYLATED	This product contains partially hydrogenated fatty acids and/ or trans fatty acids. The consumption of trans fats increases the risk of coronary heart disease by raising levels of LDL cholesterol and lowering levels of "good" HDL cholesterol. There is an ongoing debate about a possible differentiation between trans fats of natural origin and trans fats of man-made origin but so far no scientific consensus has been found. Two Canadian studies have shown that the natural trans fat vaccenic acid, found in beef and dairy products, may have an opposite health effect and could actually be beneficial compared to hydrogenated vegetable shortening, or a mixture of pork lard and soy fat, by lowering total and LDL cholesterol and triglyceride levels. In lack of recognized evidence and scientific agreement, nutritional authorities consider all trans fats as equally harmful for health and recommend that consumption of trans fats be reduced to trace amounts. The use of hydrogenated oils in foods has never been completely satisfactory. Because the center arm of the triglyceride is shielded somewhat by the end fatty acids, most of the hydrogenation occurs on the end fatty acids, While full hydrogenation produces largely saturated fatty acids, partial hydrogenation results in the transformation of unsaturated cis fatty acids to trans fatty acids in the oil mixture due to the heat used in hydrogenation. Partially hydrogenated oils and their trans fats have increasingly been viewed as "unhealthy". Trans fat is the common name for unsaturated fat with trans-isomer (E-isomer) fatty acid(s). Because the term refers to the configuration of a double carbon-carbon bond, trans fats are sometimes monounsaturated or polyunsaturated, but never saturated. Trans fats do exist in nature but also occur during the processing of polyunsaturated fatty acids in food production. Trans fats occur naturally in a limited number of cases: vaccenyl and conjugated linoleyl (CLA) containing trans fats occur naturally in trace amounts in meat and dairy products from ruminants. The exact biochemical methods by which trans fats produce specific health problems are a topic of continuing research. One theory is that the human lipase enzyme works only on the cis configuration and cannot metabolise a trans fat. A lipase is a water-soluble enzyme that helps digest, transport, and process dietary lipids such as triglycerides, fats, and oils in most - if not all - living organisms. While the mechanisms through which trans fats contribute to coronary heart disease are fairly well understood, the mechanism for trans fat's effect on diabetes is still under investigation. Trans fatty acids may impair the metabolism of long-chain polyunsaturated fatty acids (LCPUFAs), but maternal pregnancy trans fatty acid intake has been inversely associated with LCPUFAs levels in infants at birth thought to underlie the positive association between breastfeeding and intelligence. There are suggestions that the negative consequences of trans fat consumption go beyond the cardiovascular risk. In general, there is much less scientific consensus asserting that eating trans fat specifically increases the risk of other chronic health problems: It has been suggested that the intake of both trans fats and saturated fats promote the development of Alzheimer disease, although not confirmed in an animal model. It has been found that trans fats impaired memory and learning in middle-age rats. The rats' brains of trans-fat eaters had fewer proteins critical to healthy neurological function. Inflammation in and around the hippocampus, the part of the brain responsible for learning and memory. These are the exact types of changes normally seen at the onset of Alzheimer's, but seen after six weeks, even though the rats were still young. There is a growing concern that the risk of type 2 diabetes increases with trans fat consumption.[52] However, consensus has not been reached. For example, one study found that risk is higher for those in the highest quartile of trans fat consumption.Another study has found no diabetes risk once other factors such as total fat intake and BMI were accounted for. Research indicates that trans fat may increase weight gain and abdominal fat, despite a similar caloric intake. A 6-year experiment revealed that monkeys fed a trans fat diet gained 7.2% of their body weight, as compared to 1.8% for monkeys on a mono-unsaturated fat diet. Although obesity is frequently linked to trans fat in the popular media, this is generally in the context of eating too many calories;

Continued...

	there is not a strong scientific consensus connecting trans fat and obesity, although the 6-year experiment did find such a link, concluding that "under controlled feeding conditions, long-term TFA consumption was an independent factor in weight gain. TFAs enhanced intra-abdominal deposition of fat, even in the absence of caloric excess, and were associated with insulin resistance, with evidence that there is impaired post-insulin receptor binding signal transduction. Liver Dysfunction: Trans fats are metabolised differently by the liver than other fats and interfere with delta 6 desaturase. Delta 6 desaturase is an enzyme involved in converting essential fatty acids to arachidonic acid and prostaglandins, both of which are important to the functioning of cells. Infertility in women: One 2007 study found, "Each 2% increase in the intake of energy from trans unsaturated fats, as opposed to that from carbohydrates, was associated with a 73% greater risk of ovulatory infertility...". Major depressive disorder: Spanish researchers analysed the diets of 12,059 people over six years and found those who ate the most trans fats had a 48 per cent higher risk of depression than those who did not eat trans fats. One mechanism may be trans-fats' substitution for docosahexaenoic acid (DHA) levels in the orbitofrontal cortex (OFC). Very high intake of trans-fatty acids (43% of total fat) in mice from 2 to 16 months of age was associated with lowered DHA levels in the brain (p=0.001) When the brains of 15 major depressive subjects who had committed suicide were examined post-mortem and compared against 27 age-matched controls, the suicidal brains were found to have 16% less (male average) to 32% (female average) less DHA in the OFC. The OFC is known to control reward, reward expectation and empathy, which are all negatively impacted in depressive mood disorders, as well as regulating the limbic system>
ALCOHOLS C12-15 ETHOXYLATED	for Tergitol 25-L-9: Neodol 25-9 Neodol 25-7 *Shell Canada ** Huntsman (for Teric 12A9) Humans have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents and other cleaning products. Exposure to these chemicals can occur through swallowing, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that relatively high volumes would have to occur to produce any toxic response. No death due to poisoning with alcohol ethoxylates has ever been reported. Studies show that alcohol ethoxylates have low toxicity through swallowing and skin contact. Animal studies show these chemicals may produce gastrointestinal irritation, stomach ulcers, hair standing up, diarrhea and lethargy. Slight to severe irritation occurred when undiluted alcohol ethyoxylates were applied to the skin and eyes of animals. These chemicals show no indication of genetic toxicity or potential to cause mutations and cancers. Toxicity is thought to be substantially lower than that of nonylphenol ethoxylates. Some of the oxidation products of this group of substances may have sensitizing properties. As they cause less irritation, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their tendency to auto-oxidise also increases their irritation. Due to their irritating effect it is difficult to diagnose allergic contact dermatitis (ACD) by patch testing. Both laboratory and animal testing has shown that there is no evidence for alcohol ethoxylates (AEs) causing genetic damage, mutations or cancer. No adverse reproductive or developmental effects were observed. Tri-ethylene glycol ethers undergo enzymatic oxidation to toxic alkoxy acids. They may irritate the skin and the eyes. At high oral doses, they may cause depressed reflexes, flaccid muscle tone, breathing difficulty and coma. Death may result in experimental animal. However, repeated exposure may cause dose dependent damage to the kidneys as well as reproductive and developmental defects. The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
PROPYLENE GLYCOL	The acute oral toxicity of propylene glycol is very low; large amounts are needed to cause perceptible health damage in humans. Serious toxicity generally occurs only at blood concentrations over 1 g/L, which requires extremely high intake over a relatively short period of time; this is nearly impossible with consuming foods or supplements which contain 1g/kg of PG at most. Poisonings are usually due to injection through a vein or accidental swallowing of large amounts by children. The potential for long-term oral toxicity is also low. Prolonged contact with propylene glycol is essentially non-irritating to the skin. Undiluted propylene glycol is minimally irritating to the eye, and can produce a slight, temporary inflammation of the conjunctiva. Exposure to mists may cause irritation of both the eye and the upper airway. Inhalation of propylene glycol vapours may 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 metabolized in humans to pyruvic acid, acetic acid, lactic acid and propionaldehyde; the last of which is potentially hazardous. Propylene glycol show s no evidence of causing cancer or genetic toxicity. Research has suggested that individuals who cannot tolerate propylene glycol probably experience a special form of irritation, but they only rarely develop allergic contact dermatitis. Other investigators believe that the incidence of allergic contact dermatitis in people exposed 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 inflammation of the nose and hives, in children. Another study suggested that the concentration of PGEs (propylene glycol and glycol ethers) in indoor 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 bladder inflammation and vulvodynia (chronic pain of the vulva) may be especially sensitive to propylene glycol. Women suffering with yeast infections may notice that some over the counter creams cause intense burning. Post-menopausal women who require the use of an oestrogen cream may notice that creams made with propylene glycol often cause extremely uncomfortable burning along the vulva and around the anus. Some electronic cigarette users who inhale propylene glycol vapour may experience dryness of the throat or shortness of breath. Adverse responses to administration of drugs which use propylene glycol as an incipient have been seen in a number of people especially at high doses. These include low blood pressure, slow heart rate, ECG abnormalities, heartbeat irregularities, lactic acidosis, breakdown of red cells and cardiac arrest.
HEXAHYDRO-1,3,5-TRIS(HYDROXYETHYL)TRIAZINE	for 78% aqueous solution Sensitisation possible by skin contact * * Aerosol OECD 403 - Thor Chemical SDS for Emulcid 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. A ban on the use of s-triazine-based biocides in metal working fluids (MWFs) has been proposed or is in place in certain jurisdictions. The most widely used antimicrobial compounds function by releasing formaldehyde once inside the microbe cell. Some, especially triazines, release detectable levels of formaldehyde into the air space above MWFs especially when pH has dropped. This is often due to excess growth of micro-organisms that can generate organic acid as a by-product of growth. Yeasts, in particular, generate acid rapidly and can decompose in the presence of triazine to release formaldehyde. Microbes may develop tolerance or resistance to certain biocides or may be inherently less sensitive.This has been observed for the triazines typically used for bacterial control in MWFs One hypothesis, linking the use of s-triazine biocides in MWFs to the proliferation of mycobacterial species in these fluids, and hence the development of hypersensitivity pneumonitis (HP), has been proposed. It has also been suggested that exposure to aerosols containing endotoxins (powerful immune system potentiators derived from cell membranes of Gram-negative bacteria), along with mycobacterial cell wall fragments and biocides, may cause some workers to eventually manifest HP symptoms. Formaldehyde generators (releasers) are often used as preservatives. The maximum authorised concentration of free formaldehyde is 0.2% and must be labelled with the warning sign "contains formaldehyde" where the concentration exceeds 0.05%.The use of formaldehyde-releasing preservatives ensures that the level of free formaldehyde in the products is always low but sufficient to inhibit microbial growth - it disrupts metabolism to cause death of the organism. However there is a concern that formaldehyde generators can produce amines capable of causing cancers (nitrosamines) when used in formulations containing amines.

Continued...

CANOLA OIL & HEXAHYDRO-1,3,5-TRIS(HYDROXYETHYL)TRIAZINE	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production. No significant acute toxicological data identified in literature search.
CANOLA OIL & PROPYLENE GLYCOL	The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.
CASTOR OIL, HYDROGENATED, ETHOXYLATED & ALCOHOLS C12-15 ETHOXYLATED	Polyethers (such as ethoxylated surfactants and polyethylene glycols) are highly susceptible to being oxidized in the air. They then form complex mixtures of oxidation products. Animal testing reveals that whole the pure, non-oxidised surfactant is non-sensitizing, many of the oxidation products are sensitisers. The oxidization products also cause irritation.

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

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

SECTION 12 Ecological information

Toxicity

OzCrop Fipronil 200 SC Insecticide

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

canola oil

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

fipronil

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	0.005-0.023mg/L	4
EC50	72h	Algae or other aquatic plants	0.27-1.12mg/l	4
EC50	48h	Crustacea	<0.001mg/L	4
NOEC(ECx)	4h	Crustacea	<0.001mg/L	4
EC50	96h	Algae or other aquatic plants	0.631mg/L	4

castor oil, hydrogenated, ethoxylated

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	>7.33mg/l	2
NOEC(ECx)	504h	Crustacea	<0.001mg/l	2
EC50	72h	Algae or other aquatic plants	6.61mg/l	2
EC50	48h	Crustacea	>25mg/l	2

alcohols C12-15 ethoxylated

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	>=0.423<=8.211mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	0.013mg/l	2
EC50	72h	Algae or other aquatic plants	0.031mg/l	2
EC50	96h	Algae or other aquatic plants	0.7mg/l	4
EC50	48h	Crustacea	0.143mg/l	2

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

hexahydro-1,3,5-tris(hydroxyethyl)triazine

Endpoint	Test Duration (hr)	Species	Value	Source
EC10(ECx)	72h	Algae or other aquatic plants	0.92mg/l	2
EC50	72h	Algae or other aquatic plants	3.5mg/l	2
EC50	48h	Crustacea	11.9mg/l	2
LC50	96h	Fish	16.07mg/l	2

Continued...

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***

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

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
fipronil	HIGH	HIGH
propylene glycol	LOW	LOW
hexahydro-1,3,5-tris(hydroxyethyl)triazine	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
fipronil	MEDIUM (LogKOW = 4.0887)
propylene glycol	LOW (BCF = 1)
hexahydro-1,3,5-tris(hydroxyethyl)triazine	LOW (LogKOW = -4.6674)

Mobility in soil

Ingredient	Mobility
fipronil	LOW (Log KOC = 30930)
propylene glycol	HIGH (Log KOC = 1)
hexahydro-1,3,5-tris(hydroxyethyl)triazine	LOW (Log KOC = 10)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	•3Z

Land transport (ADG)

14.1. **UN number or ID number**	3082
	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains fipronil)

Continued...

14.2. **UN proper shipping name**	
14.3. **Transport hazard class(es)**	Class: 9 Subsidiary Hazard: Not Applicable
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: 274 331 335 375 AU01 Limited quantity: 5 L

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

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	3082
14.2. **UN proper shipping name**	Environmentally hazardous substance, liquid, n.o.s. (contains fipronil)
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 9 ICAO / IATA Subsidiary Hazard: Not Applicable ERG Code: 9L
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: A97 A158 A197 A215 Cargo Only Packing Instructions: 964 Cargo Only Maximum Qty / Pack: 450 L Passenger and Cargo Packing Instructions: 964 Passenger and Cargo Maximum Qty / Pack: 450 L Passenger and Cargo Limited Quantity Packing Instructions: Y964 Passenger and Cargo Limited Maximum Qty / Pack: 30 kg G

Sea transport (IMDG-Code / GGVSee)

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

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
canola oil	Not Available
fipronil	Not Available
castor oil, hydrogenated, ethoxylated	Not Available
alcohols C12-15 ethoxylated	Not Available
propylene glycol	Not Available
hexahydro-1,3,5-tris(hydroxyethyl)triazine	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
canola oil	Not Available
fipronil	Not Available

Continued...

Product name	Ship Type
castor oil, hydrogenated, ethoxylated	Not Available
alcohols C12-15 ethoxylated	Not Available
propylene glycol	Not Available
hexahydro-1,3,5-tris(hydroxyethyl)triazine	Not Available

SECTION 15 Regulatory information

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

canola oil is found on the following regulatory lists

Not Applicable

fipronil is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

castor oil, hydrogenated, ethoxylated is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

alcohols C12-15 ethoxylated is found on the following regulatory lists

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

propylene glycol is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

hexahydro-1,3,5-tris(hydroxyethyl)triazine is found on the following regulatory lists

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

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (canola oil; fipronil)
Canada - DSL	No (fipronil)
Canada - NDSL	No (fipronil; castor oil, hydrogenated, ethoxylated; alcohols C12-15 ethoxylated; propylene glycol; hexahydro-1,3,5-tris(hydroxyethyl)triazine)
China - IECSC	No (fipronil)
Europe - EINEC / ELINCS / NLP	No (canola oil)
Japan - ENCS	No (canola oil)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (canola oil; fipronil)
USA - TSCA	No (fipronil)
Taiwan - TCSI	Yes
Mexico - INSQ	No (canola oil)
Vietnam - NCI	Yes
Russia - FBEPH	No (canola oil)
Legend:	***Yes = All CAS declared ingredients are on the inventory*** ***No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.***

SECTION 16 Other information

Revision Date	26/04/2024
Initial Date	24/04/2024

SDS Version Summary

Version	Date of Update	Sections Updated
3.1	25/04/2024	Toxicological information - Acute Health (inhaled), Toxicological information - Acute Health (skin), Toxicological information - Acute Health (swallowed), Physical and chemical properties - Appearance, Toxicological information - Chronic Health, Hazards identification - Classification, Ecological Information - Environmental, Exposure controls / personal protection - Exposure Standard, Firefighting measures - Fire Fighter (fire/explosion hazard), First Aid measures - First Aid (skin), Handling and storage - Handling Procedure, Composition / information on ingredients - Ingredients, Accidental release measures - Spills (major), Handling and storage - Storage (storage incompatibility), Handling and storage - Storage (storage requirement), Toxicological information - Toxicity and Irritation (Other), Identification of the substance / mixture and of the company / undertaking - Use, Name

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4.1	26/04/2024	Composition / information on ingredients - Ingredients

Other information

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

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

Definitions and abbreviations

- PC—TWA: Permissible Concentration-Time Weighted Average
- PC—STEL: Permissible Concentration-Short Term Exposure Limit
- IARC: International Agency for Research on Cancer
- ACGIH: American Conference of Governmental Industrial Hygienists
- STEL: Short Term Exposure Limit
- TEEL: Temporary Emergency Exposure Limit。
- IDLH: Immediately Dangerous to Life or Health Concentrations
- ES: Exposure Standard
- OSF: Odour Safety Factor
- NOAEL: No Observed Adverse Effect Level
- LOAEL: Lowest Observed Adverse Effect Level
- TLV: Threshold Limit Value
- LOD: Limit Of Detection
- OTV: Odour Threshold Value
- BCF: BioConcentration Factors
- BEI: Biological Exposure Index
- 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
- 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

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end of SDS