CURACEF DUO Suspension for Injection for Cattle
Virbac (Australia) Pty Limited

Chemwatch Hazard Alert Code: **2**

Chemwatch: **87-5592**
Version No: **4.1.16.10**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **11/01/2019**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	CURACEF DUO Suspension for Injection for Cattle
Chemical Name	Not Applicable
Synonyms	APVMA No: 83071
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains ketoprofen)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	For the treatment of respiratory infections in cattle associated with signs of inflammation or pyrexia.

Details of the supplier of the safety data sheet

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

Emergency telephone number

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

SECTION 2 Hazards identification

Classification of the substance or mixture

HAZARDOUS CHEMICAL. DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

ChemWatch Hazard Ratings

	Min	Max
Flammability	1	
Toxicity	2	
Body Contact	2	
Reactivity	1	
Chronic	2	

0 = Minimum
1 = Low
2 = Moderate
3 = High
4 = Extreme

Poisons Schedule	S4
Classification [1]	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2A, Sensitisation (Skin) Category 1, Sensitisation (Respiratory) Category 1, Reproductive Toxicity Category 2, Hazardous to the Aquatic Environment Acute Hazard Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 2, Acute Toxicity (Oral) Category 4
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)

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H315	Causes skin irritation.
H319	Causes serious eye irritation.
H317	May cause an allergic skin reaction.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H361	Suspected of damaging fertility or the unborn child.
H411	Toxic to aquatic life with long lasting effects.
H302	Harmful if swallowed.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P261	Avoid breathing mist/vapours/spray.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P284	[In case of inadequate ventilation] wear respiratory protection.
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.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P330	Rinse mouth.

Precautionary statement(s) Storage

P405	Store locked up.

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
22071-15-4	10-20	ketoprofen
103980-44-5	1-10	ceftiofur hydrochloride
Not Available	>60	Ingredients determined not to be hazardous
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available*	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Wash out immediately with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary.

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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. ▸ 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

Treat symptomatically.
for non-steroidal anti-inflammatories (NSAIDs)

- Symptoms following acute NSAIDs overdoses are usually limited to lethargy, drowsiness, nausea, vomiting, and epigastric pain, which are generally reversible with supportive care. Gastrointestinal bleeding can occur. Hypertension, acute renal failure, respiratory depression, and coma may occur, but are rare. Anaphylactoid reactions have been reported with therapeutic ingestion of NSAIDs, and may occur following an overdose.
- Patients should be managed by symptomatic and supportive care following a NSAIDs overdose.
- There are no specific antidotes.
- Emesis and/or activated charcoal (60 to 100 grams in adults, 1 to 2 g/kg in children), and/or osmotic cathartic may be indicated in patients seen within 4 hours of ingestion with symptoms or following a large overdose (5 to 10 times the usual dose).
- Forced diuresis, alkalinisation of urine, hemodialysis, or haemoperfusion may not be useful due to high protein binding.
- For gastrointestinal haemorrhage, monitor stool guaiac and administer antacids or sucralfate.
- For mild/moderate allergic reactions, administer antihistamines with or without inhaled beta agonists,corticosteroids, or epinephrine.
- For severe allergic reactions, administer oxygen, antihistamines,epinephrine, or corticosteroids. Nephritis or nephrotic syndrome, thrombocytopenia, or haemolytic anemia may respond to glucocorticoid administration.
- For severe acidosis, administer sodium bicarbonate.
- Administer as required: plasma volume expanders for severe hypotension; diazepam or other benzodiazepine for convulsions; vitamin K1 for hypoprothrombinaemia; and/or dopamine plus dobutamine intravenously to prevent or reverse early indications of renal failure.

Serious gastrointestinal toxicity, such as bleeding, ulceration, and perforation, can occur at any time, with or without warning symptoms, in patients treated chronically with NSAID therapy. Although minor upper gastrointestinal problems, such as dyspepsia, are common, usually developing early in therapy, physicians should remain alert for ulceration and bleeding in patients treated chronically with NSAIDs even in the absence of previous GI tract symptoms. In patients observed in clinical trials of several months to two years duration, symptomatic upper GI ulcers, gross bleeding or perforation appear to occur in approximately 1% of patients treated for 3 to 6 months, and in about 2% to 4% of patients treated for one year. Physicians should inform patients about the signs and/or symptoms of serious GI toxicity and what steps to take if they occur.
Studies to date have not identified any subset of patients not at risk of developing peptic ulceration and bleeding. Except for a prior history of serious GI events and other risk factors known to be associated with peptic ulcer disease, such as alcoholism, smoking, etc., no risk factors (e.g., age, sex) have been associated with increased risk. Elderly or debilitated patients seem to tolerate ulceration or bleeding less well than other individuals, and most spontaneous reports of fatal GI events are in this population. Studies to date are inconclusive concerning the relative risk of various NSAIDs in causing such reactions. High doses of any NSAID probably carry a greater risk of these reactions, although controlled clinical trials showing this do not exist in most cases. In considering the use of relatively large doses (within the recommended dosage range), sufficient benefit should be anticipated to offset the potential increased risk of GI toxicity.

SECTION 5 Firefighting measures

Extinguishing media

- Foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.
- Water spray or fog - Large fires only.

Special hazards arising from the substrate or mixture

Fire Incompatibility	▸ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ Avoid spraying water onto liquid pools. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	▸ Combustible. ▸ Slight fire hazard when exposed to heat or flame. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke. ▸ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO_2) acrolein 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.
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SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. 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. **CARE**: Absorbent materials wetted with occluded oil must be moistened with water as they may auto-oxidize, become self heating and ignite. Some oils slowly oxidise when spread in a film and oil on cloths, mops, absorbents may autoxidise and generate heat, smoulder, ignite and burn. In the workplace oily rags should be collected and immersed in water. 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. ▸ 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	▸ Glass container is suitable for laboratory quantities ▸ **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. **HAZARD**: ▸ Although anti-oxidants may be present, in the original formulation, these may deplete over time as they come into contact with air.

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	▸ 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. ▸ Avoid reaction with oxidising agents · 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

Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
CURACEF DUO Suspension for Injection for Cattle	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
ketoprofen	Not Available	Not Available
ceftiofur hydrochloride	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
ketoprofen	E	≤ 0.01 mg/m³
ceftiofur hydrochloride	E	≤ 0.01 mg/m³
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

Exposure controls

Appropriate engineering controls	**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 Enclosed local exhaust ventilation is required at points of dust, fume or vapour generation. HEPA terminated local exhaust ventilation should be considered at point of generation of dust, fumes or vapours. Barrier protection or laminar flow cabinets should be considered for laboratory scale handling. A fume hood or vented balance enclosure is recommended for weighing/ transferring quantities exceeding 500 mg. When handling quantities up to 500 gram in either a standard laboratory with general dilution ventilation (e.g. 6-12 air changes per hour) is preferred. Quantities up to 1 kilogram may require a designated laboratory using fume hood, biological safety cabinet, or approved vented enclosures. Quantities exceeding 1 kilogram should be handled in a designated laboratory or containment laboratory using appropriate barrier/ containment technology. Manufacturing and pilot plant operations require barrier/ containment and direct coupling technologies.

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Barrier/ containment technology and direct coupling (totally enclosed processes that create a barrier between the equipment and the room) typically use double or split butterfly valves and hybrid unidirectional airflow/ local exhaust ventilation solutions (e.g. powder containment booths). Glove bags, isolator glove box systems are optional. HEPA filtration of exhaust from dry product handling areas is required.

Fume-hoods and other open-face containment devices are acceptable when face velocities of at least 1 m/s (200 feet/minute) are achieved. Partitions, barriers, and other partial containment technologies are required to prevent migration of the material to uncontrolled areas. For non-routine emergencies maximum local and general exhaust are necessary. 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, 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 (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, 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.)

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 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 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.

The need for respiratory protection should also be assessed where incidental or accidental exposure is anticipated: Dependent on levels of contamination, PAPR, full face air purifying devices with P2 or P3 filters or air supplied respirators should be evaluated.

The following protective devices are recommended where exposures exceed the recommended exposure control guidelines by factors of:

10; high efficiency particulate (HEPA) filters or cartridges

10-25; loose-fitting (Tyvek or helmet type) HEPA powered-air purifying respirator.

25-50; a full face-piece negative pressure respirator with HEPA filters

50-100; tight-fitting, full face-piece HEPA PAPR

100-1000; a hood-shroud HEPA PAPR or full face-piece supplied air respirator operated in pressure demand or other positive pressure mode.

Personal protection

Eye and face protection

When handling very small quantities of the material eye protection may not be required.
For laboratory, larger scale or bulk handling or where regular exposure in an occupational setting occurs:

- Chemical goggles.
- Face shield. Full face shield may be required for supplementary but never for primary protection of eyes.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection

See Hand protection below

Hands/feet protection

NOTE:

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

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
· frequency and duration of contact,
· chemical resistance of glove material,
· 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

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	EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. ▸ Rubber gloves (nitrile or low-protein, powder-free latex, latex/ nitrile). Employees allergic to latex gloves should use nitrile gloves in preference. ▸ Double gloving should be considered. ▸ PVC gloves. ▸ Change gloves frequently and when contaminated, punctured or torn. ▸ Wash hands immediately after removing gloves. ▸ Protective shoe covers. [AS/NZS 2210] ▸ Head covering. ▸ Neoprene rubber gloves
Body protection	See Other protection below
Other protection	▸ For quantities up to 500 grams a laboratory coat may be suitable. ▸ For quantities up to 1 kilogram a disposable laboratory coat or coverall of low permeability is recommended. Coveralls should be buttoned at collar and cuffs. ▸ For quantities over 1 kilogram and manufacturing operations, wear disposable coverall of low permeability and disposable shoe covers. ▸ For manufacturing operations, air-supplied full body suits may be required for the provision of advanced respiratory protection. ▸ Eye wash unit. ▸ Ensure there is ready access to an emergency shower. ▸ For Emergencies: Vinyl suit

Respiratory protection

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

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	A-AUS / Class1 P2	-
up to 50	1000	-	A-AUS / Class 1 P2
up to 50	5000	Airline *	-
up to 100	5000	-	A-2 P2
up to 100	10000	-	A-3 P2
100+			Airline**

* - Continuous Flow ** - Continuous-flow or positive pressure demand
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	Off white to pink oily liquid; does not mix with water.

Physical state	Liquid	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available

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Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Not Available	**pH as a solution (%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting. Inhalation hazard is increased at higher temperatures. Not normally a hazard due to non-volatile nature of product Inhalation of oil droplets/ aerosols may cause discomfort and may produce chemical pneumonitis. 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. Fatty acid esters are relatively non-toxic in rats. Large doses of 20-60 gm/kg are lethal in rats. The most serious side-effect of the cephalosporins is acute and potentially fatal renal failure due to proximal tubular necrosis. Neurological disturbances may occasionally occur. A generalised sensitivity reaction may occur producing pruritis, urticaria, skin rashes, fever and chills, reactions resembling serum sickness, eosinophilia, joint pain, oedema and erythema. Rarely, there may be mild neutropenia, elevated serum aspartate aminotransferase (SGOT) values and increased prothrombin times. Common adverse drug reactions (ADRs) (1% of patients) associated with the cephalosporin therapy include: diarrhea, nausea, rash, electrolyte disturbances, and/or pain and inflammation at injection site. Infrequent ADRs (0.1-1% of patients) include: vomiting, headache, dizziness, oral and vaginal candidiasis, pseudomembranous colitis, superinfection, eosinophilia, thrombocytopenia, an autoimmune type of haemolytic anaemia, and/or fever. Other effects of therapy may include taste distortion (dysgeusia), distortions of smell (dysosmia), hypotension and headache. Nausea and vomiting may result with concomitant intake of alcohol. Anaphylaxis may produce generalised breathing difficulties and respiratory distress, and may be fatal. The commonly quoted figure of 10% of patients with allergic hypersensitivity to penicillins and/or carbapenems also having cross-reactivity with cephalosporins originated from a 1975 study looking at the original cephalosporins, and subsequent "safety first" policy meant this was widely quoted and assumed to apply to all members of the group. Hence it was commonly stated that they are contraindicated in patients with a history of severe, immediate allergic reactions (urticaria, anaphylaxis, interstitial nephritis, etc) to penicillins, carbapenems or cephalosporins. This however should be viewed in the light of recent epidemiological work suggesting that for many 2nd generation (or later) cephalosporins that the cross-reactivity rate with penicillin is much lower, having no significantly increased risk of reactivity in the studies examined. Several cephalosporins are associated with hypoprothrombinaemia and a disulfiram-like reaction. This is thought to be due to the N-methylthiotetrazole (NMTT) side chain of these cephalosporins, which blocks the enzyme vitamin K epoxide reductase. JECFA established an acceptable daily intake (ADI) of 0-25 mg/kg bw for polyglyceryl esters of fatty acids having an average chain length of up to 3 glycerol units and an ADI of 0-7.5 mg/kg bw for polyglyceryl esters of interesterified ricinoleic acid. In the EU, the esters are listed as food additives at concentrations between 5000 and 10,000 mg/kg in certain foods, and up to 7% free glycerol/polyglycerol is allowed (i.e., 700 mg/kg). Non-steroidal anti-inflammatory drugs (NSAID) can cause serious gastrointestinal (GI) adverse events including inflammation, bleeding, ulceration, and perforation of the stomach, small intestine, or large intestine, which can be fatal. These serious adverse events can occur at any time, with or without warning symptoms, in patients treated with NSAIDs. Only one in five patients, who develop a serious upper GI adverse event on NSAID therapy, is symptomatic. Upper GI ulcers, gross bleeding, or perforation caused by NSAIDs occur in approximately 1% of patients treated for 3-6 months, and in about 2-4% of patients treated for one year. These trends continue with longer duration of use, increasing the likelihood of developing a serious GI event at some time during the course of therapy. Anaphylactoid (allergic) reactions may occur. This typically occurs in asthmatic patients who experience rhinitis with or without nasal polyps, or who exhibit severe, potentially fatal bronchospasm after taking aspirin or other NSAIDs. NSAIDs, can cause serious skin adverse events such as exfoliative dermatitis, Stevens-Johnson Syndrome (SJS), and toxic epidermal necrolysis (TEN), which can be fatal Non-steroidal anti-inflammatory drug (NSAID) overdose may produce nausea, vomiting, indigestion and epigastric pain. Central nervous system effects may include drowsiness, dizziness, mental confusion, disorientation, lethargy, paraesthesia, numbness, intense headache, blurred vision, tinnitus, decreased auditory acuity, ataxia, muscle twitching, convulsions, stupor and coma. Other reported effects include sweating, oliguria or anuria, tachycardia and hypo- or hypertension. Renal damage may also occur.

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Skin Contact	The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either ▸ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or ▸ produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals. Repeated or prolonged eye contact may cause inflammation (similar to windburn) characterised by a temporary redness of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects. Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects. On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Adverse effects associated with cephalosporin therapy include Stevens-Johnson syndrome, erythema multiforme, toxic epidermal necrolysis, renal dysfunction, toxic nephropathy, aplastic anaemia, haemolytic anaemia, haemorrhage, hepatic dysfunction including cholestasis and pancytopenia. In some tests, the class of beta-lactam antibiotics is clastogenic at high concentrations; no evidence exists, however, that this is a significant finding in humans. Although there have been no reports, of a general nature, that cephalosporins produce adverse effects in the foetus, their safe use in pregnancy has not been established. Cephalosporins are distributed in mothers milk. Prolonged or repeated use of antibiotics, at therapeutic doses, may produce bacterial resistance for some types of bacteria. Prolonged use may result in the overgrowth of non-susceptible organisms (i.e. super-infection). Rarely, antibiotic-associated pseudomembranous colitis, caused by toxin-producing clostridia resistant to cephalosporins, has occurred. Clinical use of cephalosporins may also produce adverse haematological effects; most of these reactions are rare, mild and transient. Transient leukopenia, neutropenia, agranulocytosis and thrombocytopenia have been reported in patients undergoing cephalosporin therapies. Mild and transient hepatic effects have also been associated with cephalosporin therapies. Many cephalosporins have produced a fall in prothrombin time. Those at risk include individuals with renal or hepatic impairment, or poor nutritional state, as well as those undergoing a protracted course of antimicrobial therapy (Vitamin K administration may be indicated). Several cephalosporins have been implicated in triggering seizures, particularly in individuals with renal impairment. Several cephalosporins have been implicated in triggering seizures, particularly in patients with renal impairment Glyceryl triesters (triglycerides), following ingestion, are metabolised to monoglycerides, free fatty acids and glycerol, all of which are absorbed in the intestinal mucosa and undergo further metabolism. Medium chain triglycerides (C8-C10) appear to have relatively rapid metabolism and elimination from blood and tissues compared to long chain triglycerides (C16-C18). Little or no acute, subchronic or chronic oral toxicity was seen in animal studies unless levels approached a significant percentage of calorific intake. Subcutaneous injections of tricaprylin in rats over a five-week period caused granulomatous reaction characterised by oil deposits surrounded by macrophages. Diets containing substantial levels of tributyrin produced gastric lesions in rats fed for 3-35 weeks; the irritative effect of the substance was thought to be the cause of tissue damage. Dermal application was not associated with significant irritation in rabbit skin; ocular exposures were, at most, mildly irritating to rabbit eyes. No evidence of sensitisation or photosensitisation was seen in a guinea pig maximisation test. Most of the genotoxicity test systems were negative. Tricaprylin, trioctanoin and triolein have been used, historically, as vehicles in carcinogenicity testing of other chemicals. In one study, subcutaneous injection of tricaprylin, in newborn mice, produced more tumours in lymphoid tissue than were seen in untreated animals whereas, in another study, subcutaneous or intraperitoneal injection in 4- to 6-week old female mice produced no tumours. Trioctanoin injected subcutaneously in hamster produced no tumours; when injected intraperitoneally in pregnant rats there was an increase in mammary tumours among the off-spring but similar studies in pregnant hamsters and rabbits showed no tumours in the off-spring. The National Toxicological Program conducted a 2-year study in rats given tricaprylin by gavage. The treatment was associated with a statistically significant dose-related increase in pancreatic acinar cell hyperplasia and adenoma but there were no acinar carcinomas. Tricaprylin is not teratogenic to mice or rats but some reproductive effects were seen in rabbits. A low level of foetal eye abnormalities and a small percentage of abnormal sperm were reported in mice injected with trioctanoin. Trioctanoin was also used as a vehicle control in a sperm abnormality test. Ten male control mice received an intraperitoneal injection of 0.25 ml trioctanoin 0.05 g/kg of benz[a]pyrene (known reproductive toxicant and mutagen) daily for 5 days and sperm from caudae epididymides analysed. Based on these studies there is no sufficient evidence to classify the trioctanoin as reproductive toxicant. In the human body, high levels of triglycerides in the bloodstream have been linked to atherosclerosis, heart disease and stroke. However, the relative negative impact of raised levels of triglycerides compared to that of LDL:HDL ratios is as yet unknown. The risk can be partly accounted for by a strong inverse relationship between triglyceride level and HDL-cholesterol level. But the risk is also due to high triglyceride levels increasing the quantity of small, dense LDL particles Prolonged or repeated use of antibiotics, at therapeutic doses, may produce bacterial resistance for some types of bacteria. Prolonged use may result in the overgrowth of non-susceptible organisms (i.e. super- infection). *Clostridium difficile* associated diarrhea (CDAD) has been reported with use of nearly all antibacterial agent and may range in severity from mild

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diarrhea to fatal colitis. Treatment with antibacterial agents alters the normal flora of the colon leading to overgrowth of *C. difficile*.
C. difficile produces toxins A and B which contribute to the development of CDAD. Hypertoxin producing strains of *C. difficile* cause increased morbidity and mortality, as these infections can be refractory to antimicrobial therapy and may require colectomy. CDAD must be considered in all patients who present with diarrhea following antibiotic use or exposure.
NSAIDs may cause an increased risk of serious cardiovascular thrombotic events, myocardial infarction, and stroke, which can be fatal.
NSAIDs cause an increased risk of serious gastrointestinal adverse events including bleeding, ulceration, and perforation of the stomach or intestines, which can be fatal. Both cyclooxygenase-1 and cyclooxygenase-2 (COX-1 and COX-2) inhibit the production of prostaglandins in the stomach and intestines responsible for maintaining the mucous lining of the gastrointestinal tract.
These events can occur at any time during use and without warning symptoms.
All NSAIDs increase plasma renin activity and aldosterone levels, and increase sodium and potassium retention. Vasopressin activity is also enhanced. Together these may lead to:

- oedema (swelling due to fluid retention)
- hyperkalaemia (high potassium levels)
- hypernatraemia (high sodium levels)
- hypertension

Elevations of serum creatinine and more serious renal damage such as acute renal failure, chronic nephritis and nephrotic syndrome, are also possible. These conditions also often begin with edema and hyperkalemia.
Many NSAIDs cause lithium retention by reducing its excretion by the kidneys; users have an elevated risk of lithium toxicity.
Prolonged treatment with non-steroidal anti-inflammatory drugs (NSAIDs) has been associated with gastrointestinal irritation, erosion, ulceration, perforation, frank or occult bleeding, diarrhoea, constipation, and blood in the vomit or stool. Kidney damage may result in haematuria (blood in the urine), pyuria (white blood cells in the urine), proteinuria (protein in the urine), urinary casts (cylindrical aggregations of particles that form in the distal nephron, dislodge, and pass into the urine), nocturia (excessive night time urination) , polyuria (production of large volumes of pale urea), dysuria (painful or difficult urination), oliguria (production of abnormally small volumes of urea), or anuria (inability to urinate), renal insufficiency (insufficient excretion of wastes by the kidney), nephrosis and nephrotic syndrome (conditions characterized by oedema and large amounts of protein in the urine and usually increased blood cholesterol), and glomerular and interstitial nephritis. Liver effects, although rare, include jaundice, hepatocellular injury, possible fatal hepatitis, and abnormal liver function tests.
Aspirin and other non-steroidal anti-inflammatory drugs, causes foetotoxicity, minor skeletal malformations, e.g., supernumerary ribs, and delayed ossification in rodent reproduction trials, but no major teratogenicity. Similarly, NSAIDs prolong gestation and interfere with parturition and with normal development of young before weaning.
Therapeutic use of NSAIDs during the second half of pregnancy is associated with adverse effects in the foetus such as premature closure of the ductus arteriosus, which may lead to persistent pulmonary hypertension in the newborn.
In rat studies with NSAIDs, as with other drugs known to inhibit prostaglandin synthesis, an increased incidence of dystocia, delayed parturition, and decreased pup survival occurred.
Because of the known effects of NSAIDs on the foetal cardiovascular system (closure of ductus arteriosus), use during pregnancy (particularly late pregnancy) should be avoided..
Animal studies have shown that NSAIDs administered during late pregnancy can cause prolonged gestation, difficult labour, delayed birth, and decreased pup survival rates

Clinical trials of several COX-2 selective and nonselective NSAIDS of up to three years duration have shown an increased risk of serious cardiovascular (CV) thrombotic events, myocardial infarction, and stroke, which can be fatal. All NSAIDs, both COX-2 selective and nonselective, may have a similar risk.
NSAIDs, can lead to onset of new hypertension or worsening of preexisting hypertension, either of which may contribute to the increased incidence of CV events.
Long-term administration of NSAIDs has resulted in renal papillary necrosis and other renal injury. Acute interstitial nephritis with haematuria, proteinuria, and occasionally nephritic syndrome have been reported.
Anaphylactoid reactions may occur in patients with known prior exposure to other NSAIDs.
NSAIDs have produced ocular changes in animals and there have been reports of adverse eye findings in patients.
Anaemia is sometimes seen in patients receiving NSAIDs.. This may be due to fluid retention, occult or gross GI blood loss, or an incompletely described effect upon erythropoiesis.

NSAIDs inhibit enzymes collectively described as "COXs". In the course of the early search for a specific inhibitor of the negative effects of prostaglandins which spared the positive effects, it was discovered that prostaglandins could indeed be separated into two general classes which could loosely be regarded as "good prostaglandins" and "bad prostaglandins", according to the structure of a particular enzyme involved in their biosynthesis, cyclooxygenase (COX).
Prostaglandins whose synthesis involves the cyclooxygenase-I enzyme, or COX-1, are responsible for maintenance and protection of the gastrointestinal tract, while prostaglandins whose synthesis involves the cyclooxygenase-II enzyme, or COX-2, are responsible for inflammation and pain.

The existing non-steroidal anti-inflammatory drugs (NSAIDs) differ in their relative specificities for COX-2 and COX-1
There has been much concern about the possibility of increased risk for heart attack and stroke in users of NSAID drugs, particularly COX-2 selective NSAIDs. The cardiovascular risks associated with NSAIDs are controversial, with apparently contradictory data produced from different clinical trials and in published meta-analyses. Cardiovascular risk of COX-2 specific inhibitors is not surprising since prostaglandins are involved in regulation of blood pressure by the kidneys. COX-inhibitors produce blood dyscrasias (abnormal conditions of the blood), and interfere with platelet function.
Phototoxic or photoallergic skin reactions may also occur. Anaphylactoid reactions characterised by maculopapular rash, urticaria, pruritus, bronchospasm, and syncope have been described. Other effects include oedema, metabolic acidosis, hyperkalaemia, azotemia, cystitis and urinary tract infections, visual and hearing disturbances, conjunctivitis, corneal deposits, retinal degeneration, ear pain and occasionally, deafness. Idiosyncratic responses include asthma, allergic interstitial nephritis, hypersensitivity hepatitis, aplastic anaemia and exfoliative dermatitis.

Non-steroidal anti-inflammatory drugs with an inhibitory effect on prostaglandin synthesis, when given during the latter stages of pregnancy, cause premature closure of the foetal ductus arteriosus (1). When given at term they prolong labour and delay parturition. Evidence (1) from animal experimental studies, clinical investigations in humans, and epidemiological studies supports the hypothesis that NSAIDs are chemopreventative agents against colon cancer. This is corroborated by knowledge of the underlying pathophysiological mechanisms and the effects of arachidonic metabolites, i.e prostaglandins, on the carcinogenic process and the influence of cyclooxygenase (COX) inhibitors such as NSAIDs on these metabolites. 1. Berkel et al; Epidemiol Rev., Vol 18, No. 2, 1996

Because of the known effects of NSAIDs drugs on the foetal cardiovascular system (closure of ductus arteriosus), use during pregnancy (particularly late pregnancy) should be avoided. In rat studies with NSAIDs, as with other drugs known to inhibit prostaglandin synthesis, an increased incidence of dystocia, delayed parturition, and decreased pup survival occurred
Aspirin and NSAIDs may cause anaphylactic or anaphylactoid reactions. Constitutively-expressed cyclooxygenase (COX-1) inhibition is likely to be responsible for the cross-reactions and side effects associated with these drugs, as well as the anaphylactoid reactions sometimes seen in aspirin-sensitive respiratory disease. Though anaphylactic and anaphylactoid reactions may be clinically indistinguishable, they involve different mechanisms. Anaphylactic reactions are due to immediate hypersensitivity involving cross-linking of drug-specific IgE. Regardless of COX selectivity pattern, NSAIDs may function as haptens capable of inducing allergic sensitization. Unlike anaphylaxis, anaphylactoid reactions are most likely related to inhibition of COX-1 by NSAIDS. Thus, an anaphylactoid reaction caused by a particular COX-1 inhibiting NSAID will occur with a chemically unrelated NSAID which also inhibits COX-1 enzymes. Selective COX-2 inhibitors appear to be safe in patients with a history of NSAID-related anaphylactoid reactions but can function as haptens, with resulting sensitisation and anaphylaxis upon next exposure. Eva A

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Berkes Clinical Reviews in Allergy and Immunology 24, pp 137-147 2003.

COX-2 inhibitors reduce inflammation (and pain) while minimising gastrointestinal adverse drug reactions (e.g. stomach ulcers) that are common with non-selective NSAIDs. COX-1 is involved in synthesis of prostaglandins and thromboxane, but COX-2 is only involved in the synthesis of prostaglandin. Therefore, inhibition of COX-2 inhibits only prostaglandin synthesis without affecting thromboxane and thus has no effect on platelet aggregation or blood clotting.

Chronic abuse of analgesics has been associated with nephropathy. Patients invariably have a history of regular ingestion of substantial or excessive doses over a period of years. In mild cases the condition is reversible. The initial renal lesion is papillary necrosis proceeding to secondary atrophic changes in the renal cortex body. An abnormally high incidence of transitional cell carcinoma of the renal pelvis and bladders has been reported in patients with analgesic nephropathy.
Synthetic 1,2-diglycerides of short chain (C6, C8, C10) fatty acids are activators of protein kinase C (PKC). PKC is a serine-threonine kinase which also requires calcium ion for its activation. Activated PKC phosphorylates proteins of the cellular signal cascade, which eventually induce expression of growth regulatory genes. This, in turn, may promote the growth of tumours. Structural analogues of the 1,2-diglycerides, such as the phorbol esters, have been shown to strongly promote such an event.
In biochemical signaling, diacylglycerol (DAG) functions as a second messenger signaling lipid, and is a product of the hydrolysis of the phospholipid PIP2 (phosphatidylinositolbisphosphate) by the enzyme phospholipase C (PLC) (a membrane-bound enzyme) that, through the same reaction, produces inositol trisphosphate (IP3). Although inositol trisphosphate (IP3) diffuses into the cytosol, DAG remains within the plasma membrane due to its hydrophobic properties. IP3 stimulates the release of calcium ions from the smooth endoplasmic reticulum, whereas DAG is a physiological activator of protein kinase C (PKC). The production of DAG in the membrane facilitates translocation of PKC from the cytosol to the plasma membrane.
Glyceryl dilaurate, glyceryl diarachidate, glyceryl dibehenate, glyceryl dierucate, glyceryl dihydroxystearate, glyceryl diisopalmitate, glyceryl diisostearate, glyceryl dilinoleate, glyceryl dimyristate, glyceryl dioleate, glyceryl diricinoleate, glyceryl dipalmitate, glyceryl dipalmitoleate, glyceryl distearate, glyceryl palmitate lactate, glyceryl stearate citrate, glyceryl stearate lactate, and glyceryl stearate succinate are diacylglycerols (also known as DAGs, diglycerides or glyceryl diesters) that function as skin conditioning agents-emollients in cosmetics. Only glyceryl dilaurate (up to 5%), glyceryl diisostearate (up to 43%), glyceryl dioleate (up to 2%), glyceryl distearate (up to 7%), and glyceryl stearate lactate (up to 5%) are reported to be in current use. Production proceeds from fully refined vegetable oils, which are further processed using hydrogenation and fractionation techniques, and the end products are produced by reacting selected mixtures of the partly hydrogenated, partly fractionated oils and fats with vegetable-derived glycerine to yield partial glycerides. In the final stage of the production process, the products are purified by deodorization, which effectively removes pesticide residues and lower boiling residues such as residues of halogenated solvents and aromatic solvents. Diglycerides have been approved by the Food and Drug Administration (FDA) for use as indirect food additives. Nominally, these ingredients are 1,3-diglycerides, but are easily isomerised to the 1,2-diglycerides form. The 1,3-diglyceride isomer is not a significant toxicant in acute, short-term, subchronic, or chronic animal tests. Glyceryl dilaurate was a mild primary irritant in albino rabbits, but not a skin sensitiser in guinea pig maximization tests. Diacylglycerol oil was not genotoxic in the Ames test, in mammalian Chinese hamster lung cells, or in a rodent bone marrow micronucleus assay. An eye shadow containing 1.5% glyceryl dilaurate did not induce skin irritation in a single insult patch test, but mild skin irritation reactions to a foundation containing the same concentration were observed. A trade mixture containing an unspecified concentration of glyceryl dibehenate did not induce irritation or significant cutaneous intolerance in a 48-h occlusive patch test. In maximization tests, neither an eye shadow nor a foundation containing 1.5% glyceryl dilaurate was a skin sensitiser. Sensitisation was not induced in subjects patch tested with 50% *w/w* glyceryl dioleate in a repeated insult, occlusive patch test. Glyceryl palmitate lactate (50% *w/v*) did not induce skin irritation or sensitization in subjects patch tested in a repeat-insult patch test. Phototoxicity or photoallergenicity was not induced in healthy volunteers tested with a lipstick containing 1.0% Glyceryl rosinate. Two diacylglycerols, 1-oleoyl-2-acetoyl-sn-glycerol and 1,2-dipalmitoyl-sn-glycerol, did not alter cell proliferation (as determined by DNA synthesis) in normal human dermal fibroblasts in vitro at doses up to 10 μg/ml. In the absence of initiation, Glyceryl distearate induced a moderate hyperplastic response in randomly bred mice of a tumor-resistant strain, and with 9,10-dimethyl-1,2-benzanthracene (DMBA) initiation, an increase in the total cell count was observed. In a glyceryl monoester study, a single application of DMBA to the skin followed by 5% glyceryl stearate twice weekly produced no tumors, but slight epidermal hyperplasia at the site of application. Glyceryl dioleate induced transformation in 3-methylcholanthrene-initiated BALB/3T3 A31-1-1 cloned cells in vitro. A tumour-promoting dosing regimen that consisted of multiple applications of 10 μmol of a 1,2-diacylglycerol (sn-1,2-didecanoylglycerol) to female mice twice daily for 1 week caused more than a 60% decrease in protein kinase C (PKC) activity and marked epidermal hyperplasia. Applications of 10 umol sn-1,2-didecanoylglycerol twice weekly for 1 week caused a decrease in cytosolic PKC activity, an increase in particulate PKC activity, and no epidermal hyperplasia. In studies of the tumour-promoting activity of 1,2-diacylglycerols, dose and the exposure regimen by which the dose is delivered play a role in tumor promotion. The 1,2-diacylglycerol–induced activation of PKC may also relate to the saturation of the fatty acid in the 1 or 2 position; 1,2-Diacylglycerols with two saturated fatty acids are less effective. Also, the activity of 1,2-diacylglycerols may be reduced when the fatty acid moiety in the structure is a long-chain fatty acid. A histological evaluation was performed on human skin from female volunteers (18 to 56 years old) who had applied a prototype lotion or placebo formulation, both containing 0.5% Glyceryl Dilaurate, consecutively for 16 weeks or 21 weeks. Skin irritation was not observed in any of the subjects tested. Biopsies (2 mm) taken from both legs of five subjects indicated no recognizable abnormalities of the skin; the epidermis was normal in thickness, and there was no evidence of scaling, inflammation, or neoplasms in any of the tissues that were evaluated. The available safety test data indicate that diglycerides in the 1,3-diester form do not present any significant acute toxicity risk, nor are these ingredients irritating, sensitizing, or photosensitising. Whereas no data are available regarding reproductive or developmental toxicity, there is no reason to suspect any such toxicity because the dermal absorption of these chemicals is negligible. 1,3-Diglycerides contain 1,2-diglycerides, raising the concern that 1,2-diglycerides could potentially induce hyperplasia. Data regarding the induction of PKC and the tumour promotion potential of 1,2-diacylglycerols increases the level of concern. Most of the diglycerides considered above, however, have fatty acid chains longer than 14 carbons and none have mixed saturated/unsaturated fatty acid moieties. In a 21-week use study of a prototype lotion containing 0.5% glyceryl dilaurate (a 14-carbon chain fatty acid) indicated no evidence of scaling, inflammation, or neoplasms in biopsy specimens. Also, DNA synthesis assays on glyceryl dilaurate and glyceryl distearate indicated that neither chemical altered cell proliferation (as determined by DNA synthesis) in normal human dermal fibroblasts in vitro at doses up to 10 ug/ml. However the concentration of these ingredients can vary (up to 43% for glyceryl diisostearate in lipstick), the frequency of application can be several times daily, and the proportion of diglycerides that are inactive 1,3 isomers versus potentially biologically active 1,2 isomers is unknown; as a precaution it is believed that each use should be examined to ensure the absence of epidermal hyperplasia during product development and testing. In the absence of inhalation toxicity data on the glyceryl diesters it is thought that these ingredients can be used safely in aerosolised products because they are not respirable. Although there are gaps in knowledge about product use, the overall information available on the types of products in which these ingredients are used and at what concentration indicate a pattern of use. Within this overall pattern of use, the CIR Expert Panel considers all ingredients in this group to be safe.
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Exposure to small quantities may induce hypersensitivity reactions characterised by acute bronchospasm, hives (urticaria), deep dermal wheals (angioneurotic oedema), running nose (rhinitis) and blurred vision. Anaphylactic shock and skin rash (non-thrombocytopenic purpura) may occur. An individual may be predisposed to such antibody mediated reaction if other chemical agents have caused prior sensitisation (cross-sensitivity).

CURACEF DUO Suspension for Injection for Cattle

TOXICITY	IRRITATION
Inhalation (Rat) LC50: 8.3 mg/l*(ceftiofur HCl)[2]	Not Available
Oral (Rat) LD50: 7760* mg/kg*(ceftiofurHCl)[2]	

ketoprofen

TOXICITY	IRRITATION
Oral(Rat) LD50; 62.4 mg/kg[2]	Not Available

Continued...

ceftiofur hydrochloride	**TOXICITY**	**IRRITATION**
	Oral(Rat) LD50; >7760 mg/kg[2]	Eye(rabbit) : minimally irritating*
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*	

KETOPROFEN	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production. Oral (woman) TDLo: 80 mg/kg/10d-I Zonal hepatitis (hepatocellular necrosis), jaundice, peritonitis, body temperature increase, nausea, vomiting, changes in bladder tubules, degenerative brain changes, headache, ulceration/ bleeding from the small intestine, changes in spleen weight, gastrointestinal changes, reproductive system tumours, pigmented/ nucleated red blood cells, ulceration/ bleeding from the duodenum, specific developmental abnormalities (cardiovascular system) recorded.
CEFTIOFUR HYDROCHLORIDE	ADI: 0.03 mg/kg/day NOEL: 30 mg/kg/day Target Organs: skin, respiratory tract, immune system, gastrointestinal tract, blood * Oral (dog) NOEL 30 mg/kg/d-90D for effects blood forming organs Reproductive toxicity Oral (rat) NOEL 1000 mg/kg/day for foetotoxicity * Developmental toxicity Oral (rat) NOEL 3200 mg/kg/d for embryo/ foetal developmental effects 8 Mutagenicity: Salmonella/ E. coli - Ames: negative * Micronucleus: negative V79 cells mammalian cell mutation assay: negative DNA (unscheduled synthesis): negative CHO cellss (in absence of S9 metabolic activation: positive (chromosome aberration assay Teratogenicity May cause hypersensitivity reactions * Zhejiang Hisun Pharmaceuticl Company SDS 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. Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.

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

CURACEF DUO Suspension for Injection for Cattle	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
ketoprofen	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	NOEC(ECx)	6h	Fish	0.025mg/L	4
ceftiofur hydrochloride	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
Legend:	*Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*				

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

Persistence and degradability

Continued...

Ingredient	Persistence: Water/Soil	Persistence: Air
ketoprofen	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
ketoprofen	LOW (LogKOW = 3.0001)

Mobility in soil

Ingredient	Mobility
ketoprofen	LOW (KOC = 287.8)

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)

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

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

Continued...

Air transport (ICAO-IATA / DGR)

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

Sea transport (IMDG-Code / GGVSee)

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

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

Not Applicable

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

Product name	Group
ketoprofen	Not Available
ceftiofur hydrochloride	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
ketoprofen	Not Available
ceftiofur hydrochloride	Not Available

SECTION 15 Regulatory information

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

ketoprofen is found on the following regulatory lists

Australia Chemicals with non-industrial uses removed from the Australian Inventory of Chemical Substances (old Inventory)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 3
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
FEI Equine Prohibited Substances List - Controlled Medication
FEI Equine Prohibited Substances List (EPSL)

ceftiofur hydrochloride is found on the following regulatory lists

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

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (ceftiofur hydrochloride)
Canada - DSL	No (ketoprofen; ceftiofur hydrochloride)
Canada - NDSL	No (ceftiofur hydrochloride)

Continued...

National Inventory	Status
China - IECSC	No (ceftiofur hydrochloride)
Europe - EINEC / ELINCS / NLP	No (ceftiofur hydrochloride)
Japan - ENCS	No (ketoprofen; ceftiofur hydrochloride)
Korea - KECI	No (ceftiofur hydrochloride)
New Zealand - NZIoC	Yes
Philippines - PICCS	No (ceftiofur hydrochloride)
USA - TSCA	No (ceftiofur hydrochloride)
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	No (ketoprofen; ceftiofur hydrochloride)
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	11/01/2019
Initial Date	11/15/2017

SDS Version Summary

Version	Date of Update	Sections Updated
3.1.1.1	11/22/2017	First Aid (skin), Ingredients, Toxicity and Irritation (Toxicity Figure), Toxicity and Irritation (Other), Use
4.1.1.1	11/01/2019	One-off system update. NOTE: This may or may not change the GHS classification
4.1.2.1	04/26/2021	Regulation Change
4.1.3.1	05/03/2021	Regulation Change
4.1.4.1	05/06/2021	Regulation Change
4.1.5.1	05/10/2021	Regulation Change
4.1.5.2	05/30/2021	Template Change
4.1.5.3	06/04/2021	Template Change
4.1.5.4	06/05/2021	Template Change
4.1.6.4	06/07/2021	Regulation Change
4.1.6.5	06/09/2021	Template Change
4.1.6.6	06/11/2021	Template Change
4.1.6.7	06/15/2021	Template Change
4.1.7.7	06/17/2021	Regulation Change
4.1.8.7	06/21/2021	Regulation Change
4.1.8.8	07/05/2021	Template Change
4.1.9.8	07/14/2021	Regulation Change
4.1.10.8	07/19/2021	Regulation Change
4.1.10.9	08/01/2021	Template Change
4.1.11.9	08/02/2021	Regulation Change
4.1.12.9	08/05/2021	Regulation Change
4.1.13.9	08/09/2021	Regulation Change
4.1.14.9	08/23/2021	Regulation Change
4.1.15.9	08/26/2021	Regulation Change
4.1.15.10	08/29/2021	Template Change
4.1.16.10	08/30/2021	Regulation Change

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

Continued...

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

end of SDS