

Cydectin Pour-On for Cattle and Red Deer

Virbac (Australia) Pty Limited

Chemwatch: **6095669** Version No: **4.1.1.1**

Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 3

Issue Date: **10/16/2017** Print Date: **10/18/2017** L.GHS.AUS.EN

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

Product Identifier

| Product name | Cydectin Pour-On for Cattle and Red Deer | |
|-------------------------------|--|--|
| Synonyms | APVMA No: 45970 | |
| Other means of identification | Not Available | |
| | | |

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

Details of the supplier of the safety data sheet

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

Emergency telephone number

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

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

| Poisons Schedule | \$5 | |
|-------------------------------|--|--|
| Classification ^[1] | kin Corrosion/Irritation Category 2, Serious Eye Damage Category 1, Specific target organ toxicity - single exposure Category 3 (narcotic effects), spiration Hazard Category 1, Acute Aquatic Hazard Category 3 (Chronic Aquatic Hazard Category 3) | |
| Legend: | 1. Classified by Chemwatch; 2. Classification drawn from HSIS; 3. Classification drawn from EC Directive 1272/2008 - Annex VI | |

Label elements

Hazard pictogram(s)







SIGNAL WORD DANGER

Hazard statement(s)

| H315 | Causes skin irritation. |
|--------|---|
| H318 | Causes serious eye damage. |
| H336 | May cause drowsiness or dizziness. |
| H304 | May be fatal if swallowed and enters airways. |
| H412 | Harmful to aquatic life with long lasting effects. |
| AUH066 | Repeated exposure may cause skin dryness and cracking |

Precautionary statement(s) Prevention

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

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Precautionary statement(s) Response

| P301+P310 | IF SWALLOWED: Immediately call a POISON CENTER or doctor/physician. | |
|----------------|--|--|
| P305+P351+P338 | IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing. | |
| P331 | Do NOT induce vomiting. | |
| P362 | Take off contaminated clothing and wash before reuse. | |
| P302+P352 | IF ON SKIN: Wash with plenty of soap and water. | |
| P304+P340 | IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing. | |
| P332+P313 | If skin irritation occurs: Get medical advice/attention. | |

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

Dispose of contents/container in accordance with local regulations.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

| CAS No | %[weight] | Name |
|---------------|-----------|--|
| 113507-06-5 | 0.525 | <u>moxidectin</u> |
| | | (5.25g/L) |
| 64742-95-6. | 10-20 | naphtha petroleum, light aromatic solvent |
| 74775-06-7 | 1-10 | polypropylene glycol monomyristyl ether propionate |
| 9003-27-4 | 1-10 | isobutylene homopolymer |
| Not Available | >60 | Ingredients determined not to be hazardous |

SECTION 4 FIRST AID MEASURES

Description of first aid measures

| Description of mist did measi | * |
|-------------------------------|---|
| 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 furnes 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. |
| Ingestion | For advice, contact a Poisons Information Centre or a doctor at once. Urgent hospital treatment is likely to be needed. If swallowed do NOT induce vomiting. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. Observe the patient carefully. Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. Transport to hospital or doctor without delay. |

Indication of any immediate medical attention and special treatment needed

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.

Treat symptomatically.

For abamectin (avermectins):

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

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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

| opoolar nazarao arioing nom | | | |
|-----------------------------|---|--|--|
| 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 (CO2) , acrolein | | |
| | other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes. 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 | Not Applicable | | |

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

| Minor Spills | Slippery when spilt. Remove all ignition sources. 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 | 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.

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SECTION 7 HANDLING AND STORAGE

Safe handling

Precautions for safe handling

- ▶ 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.

- Store in original containers.
- Keep containers securely sealed.
- Other information
- No smoking, naked lights or ignition sources.
- Store in a cool, drv. well-ventilated area.
- Store away from incompatible materials and foodstuff containers.
- Protect containers against physical damage and check regularly for leaks.
- ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container

- Metal can or drum
- Packaging as recommended by manufacturer
- Check all containers are clearly labelled and free from leaks.
- Storage incompatibility
- Avoid reaction with oxidising agents
- Avoid strong acids, bases.

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Not Available

EMERGENCY LIMITS

| Ingredient | Material name | TEEL-1 | TEEL-2 | TEEL-3 |
|--|---------------|---------------|---------------|---------------|
| Cydectin Pour-On for Cattle and Red Deer | Not Available | Not Available | Not Available | Not Available |
| Ingredient | Original IDLH | | Revised IDLH | |
| moxidectin | Not Available | | Not Available | |
| naphtha petroleum, light aromatic solvent | Not Available | | Not Available | |
| polypropylene glycol monomyristyl ether propionate | Not Available | | Not Available | |
| isobutylene homopolymer | Not Available | | Not Available | |
| Ingredients determined not to be hazardous | Not Available | | Not Available | |

MATERIAL DATA

NOTE M: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.005% w/w benzo[a]pyrene (EINECS No 200-028-5). This note applies only to certain complex oil-derived substances in Annex IV.

European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

NOTE P: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene (EINECS No 200-753-7). Note E shall also apply when the substance is classified as a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI.

European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

Exposure controls

Care: Atmospheres in bulk storages and even apparently empty tanks may be hazardous by oxygen depletion. Atmosphere must be checked before entry.

Appropriate engineering controls

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

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

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match the particular process and chemical or contaminant in use.

Employers may need to use multiple types of controls to prevent employee overexposure.

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.

An approved self contained breathing apparatus (SCBA) may be required in some situations.

Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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

Within each range the appropriate value depends on:

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

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

Personal protection











Eye and face protection

- Safety glasses with side shields.
- Chemical goggles
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly, [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection

Hands/feet protection

See Hand protection below

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

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 moisturizer 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.

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

recommended.

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| Body protection | See Other protection below |
|------------------|---|
| Other protection | Overalls. P.V.C. apron. Barrier cream. Skin cleansing cream. Eye wash unit. |
| Thermal hazards | Not Available |

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.

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

| Appearance | 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 |
| 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 (g/L) | Immiscible | pH as a solution (1%) | Not Available |
| Vapour density (Air = 1) | Not Available | VOC g/L | Not Available |

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

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Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo

Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.

Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals. following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.

The acute toxicity of inhaled alkylbenzenes is best described by central nervous system depression. As a rule, these compounds may also act as general anaesthetics.

Systemic poisoning produced by general anaesthesia is characterised by lightheadedness, nervousness, apprehension, euphoria, confusion, dizziness, drowsiness, tinnitus, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness and respiratory depression and arrest. Cardiac arrest may result from cardiovascular collapse. Bradycardia, and hypotension may also be produced. Inhaled alkylbenzene vapours cause death in animals at air levels that are relatively similar (typically LC50s are in the range 5000 -8000 ppm for 4 to 8 hour exposures). It is likely that acute inhalation exposure to alkylbenzenes resembles that to general anaesthetics.

Alkylbenzenes are not generally toxic other than at high levels of exposure. This may be because their metabolites have a low order of toxicity and are easily excreted. There is little or no evidence to suggest that metabolic pathways can become saturated leading to spillover to alternate pathways. Nor is there evidence that toxic reactive intermediates, which may produce subsequent toxic or mutagenic effects, are formed

Inhaled

High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations.

Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal. A significant number of individuals exposed to mixed trimethylbenzenes complained of nervousness, tension, anxiety and asthmatic bronchitis. Peripheral blood showed a tendency to hypochromic anaemia and a deviation from normal in coagulability of the blood. Hydrocarbon concentrations ranged from 10 to 60 ppm. Contamination of the mixture with benzene may have been responsible for the blood dyscrasias.

High concentrations of mesitylene vapour (5000 to 9000 ppm) caused central nervous system depression in mice. Similar exposures of pseudocumene also produced evidence of CNS involvement.

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

Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result. Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish

coloured skin (cvanosis). Accidental ingestion of the material may be damaging to the health of the individual.

Ingestion

Fatty acid esters are relatively non-toxic in rats. Large doses of 20-60 gm/kg are lethal in rats.

Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosal ulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea and vomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injury may produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Light aromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration into the lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage.

Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.

The material may accentuate any pre-existing dermatitis condition

Skin Contact

Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.

One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants

Open cuts, abraded or irritated skin should not be exposed to this material

The liquid may be miscible with fats or oils and may degrease the skin, producing a skin reaction described as non-allergic contact dermatitis. The material is unlikely to produce an irritant dermatitis as described in EC Directives

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.

When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.

Eye

Some nonionic surfactants may produce a localised anaesthetic effect on the comea; this may effectively eliminate the warning discomfort produced by other substances and lead to corneal injury. Irritant effects range from minimal to severe dependent on the nature of the surfactant, its concentration and the duration of contact. Pain and corneal damage represent the most severe manifestation of irritation.

Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may also result. The aromatic fraction may produce irritation and lachrymation.

Chronic

Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical

There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects

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have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.

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.

Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses. Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.

| Cydectin Pour-On for Cattle and Red Deer | TOXICITY | IRRITATION | |
|---|--|---------------------------------|--|
| | Not Available | Not Available | |
| | TOXICITY | IRRITATION | |
| moxidectin | Dermal (rabbit) LD50: >2000 mg/kg ^[2] | Eye (rabbit): slight irritant * | |
| | Oral (rat) LD50: 106 mg/kg ^[2] | Skin (rabbit): non-irritant * | |
| | TOXICITY | IRRITATION | |
| naphtha petroleum, light | Dermal (rabbit) LD50: >1900 mg/kg ^[1] | Not Available | |
| aromatic solvent | Inhalation (rat) LC50: >7331.62506 mg/l/8h*[2] | | |
| | Oral (rat) LD50: >4500 mg/kg ^[1] | | |
| | TOXICITY | IRRITATION | |
| polypropylene glycol | Oral (rat) LD50: >5000 mg/kg ^[2] | Eye (rabbit): non irritating | |
| onomyristyl ether propionate | | Skin (human): non irritating | |
| | | Skin (rabbit): mild | |
| | TOXICITY | IRRITATION | |
| isobutylene homopolymer | dermal (rat) LD50: >2000 mg/kg ^[1] | Not Available | |
| | Oral (rat) LD50: >2000 mg/kg ^[1] | | |

Leaend:

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

Technical avermectin exhibits high mammalian acute toxicity. It is not considered to be mutagenic and does not sensitise skin. It is not readily absorbed by mammals and the majority of the residue is excreted in the faeces within 2 days. The 24-month rat chronic feeding/ oncogenicity study and 94-week mouse chronic toxicity oncogenicity study were negative for oncogenic potential. The results of a series of developmental toxicity studies (rat, rabbit, mouse) have been evaluated and showed that avermectin B1 produces developmental toxicity (cleft palate) in the CF1 mouse. Toxicology data were also evaluated for the delta-8,9-isomer of avermectin B1 which is a plant photodegradate that can range between 5 and 20 percent of the residue on/in cottonseed. This isomer possesses avermectin-like toxicological activity. It was concluded that the delta 8,9-isomer also produces developmental toxicity (cleft palate) in mice, but not in rats. In addition to avermectin and its delta 8,9-isomer, toxicology data were also evaluated for the "polar degradates" of avermectin, which constitute a large percentage (up to 70%) of the total residue on cottonseed. Review of the toxicology data indicated that these polar degradates do not possess avermectin-like toxicological activity and for this reason need not be included in the tolerance expression for residues in/on cottonseed. Abamectin (a mixture of avermectin isomers) is a reproductive toxin in laboratory animals at doses which are acutely toxic to the mother. In development toxicity studies with abamectin, cleft palates were seen in mice and rabbits and clubbing of the forepaws was seen in rabbits. The no-observed-adverseeffect-level (NOAEL) for maternal and developmental toxicity in rabbits was 1 mg/kg/day. In CF-1 mice, a strain recognised to be particularly sensitive to

MOXIDECTIN

content than human breast milk and abamectin concentrates in fat; (b) on a weight basis, the neonatal rat consumes significantly greater quantities of milk

appropriate model for assessing human risk in humans because (a) rat milk has a greater fat

avermectins, the NOAEL for maternal toxicity was 0.05 mg/kg/day and the NOAEL for malformations was 0.2 mg/kg/day. Studies show that the sensitivity of a subpopulation of CF-1 mice to avermectins is due to the absence of a transmembrane P-glycoprotein, a significant component of the blood-brain interface that normally acts as a non-selective protective barrier in a wide range of species including humans. CF-1 mice are therefore an unlikely candidate for assessing human risk. No evidence of developmental toxicity was seen in oral studies in rats in the absence of maternal toxicity (NOAEL = 1.6 mg/kg/day). In a rat multigenerational reproduction study, pup toxicity and deaths were seen at 0.4 mg/kg/day (NOAEL = 0.12 mg/kg/day). Neonatal rats are not an

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than the newborn human and(c) the blood brain barrier in rodents is formed post-natally (as evidenced by low P-glycoprotein levels) while in humans this membrane is formed pre-natally.

Ivermectin, a close structural analogue, has been used extensively in the treatment of human onchocerciasis at an oral therapeutic dose of 0.2 mg/kg, without serious drug-related effects. Despite its wide usage in animals and humans, ivermectin does dot appear to produce birth defects. Abamectin is non-mutagenic in the Ames test and the micronucleus test.

Dietary carcinogenicity studies in mice and rats showed negative results. In a 14-week oral study in monkeys no effects were seen at 0.2, 0.5 or 1.0 mg/kg/day; emesis was seen at 2.0 mg/kg/day; delayed pupillary obstruction at 6 and 8 mg/kg/day and mydriasis at 12 mg/kg/day.

In chronic oral toxicity, abamectin produced decreased body weight gain in mice (no-observed-adverse-effect-level (NOAEL) = 1.5 mg/kg/day); tremors in rats (NOAEL = 1.5 mg/kg/day), weight loss, tremors, mydriasis, liver and gall bladder changes and death in dogs (NOAEL = 0.25 mg/kg/day); and emesis, mydriasis and sedation in monkeys (NOAL = 1 mg/kg/day).

May produce developmental toxicity in rat offspring at maternally toxic doses. This does not occur in rabbits. * * Cyanamid The ADI for Moxidectin is set at 0.01mg/kg/day. The corresponding NOEL is set at 1mg/kg/day. ADI means Acceptable Daily Intake and NOEL means No-observable-effect-level. In rats given oral doses of moxidectin, decreased activity, prostration, tremors, chromodacryorrhea, decreased respiration, diarrhoea, hypersensitivity to touch and sound, and epistaxis occurred. Congestion of the liver, kidneys and lungs were observed in animals that died, but animals which were sacrificed at the end of the 14-day observation period showed no abnormalities. No overt signs of toxicity were noted in rabbits treated dermally with moxidectin.

Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure. Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal. Following oral administration of the chemical to rats, 62.6% of the dose was recovered as urinary metabolites indicating substantial absorption . 1,2,4-Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. In the blood stream, approximately 85% of the chemical is bound to red blood cells. Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion. After a single oral dose to rats of 1200 mg/kg, urinary metabolites consisted of approximately 43.2% glycine, 6.6% glucuronic, and 12.9% sulfuric acid conjugates. The two principle metabolites excreted by rabbits after oral administration of 438 mg/kg/day for 5 days were 2,4-dimethylbenzoic acid and 3,4-dimethylhippuric acid . The major routes of excretion of 1,2,4-trimethyl- benzene are exhalation of parent compound and elimination of urinary metabolites. Half-times for urinary metabolites were reported as 9.5 hours for glycine, 22.9 hours for glucuronide, and 37.6 hours for sulfuric acid conjugates.

Acute Toxicity Direct contact with liquid 1,2,4-trimethylbenzene is irritating to the skin and breathing the vapor is irritating to the respiratory tract causing pneumonitis. Breathing high concentrations of the chemical vapor causes headache, fatigue, and drowsiness. In humans liquid 1,2,4-trimethylbenzene is irritating to the skin and inhalation of vapor causes chemical pneumonitis. High concentrations of vapor (5000-9000 ppm) cause headache, fatigue, and drowsiness. The concentration of 5000 ppm is roughly equivalent to a total of 221 mg/kg assuming a 30 minute exposure period (see end note 1). 2. Animals - Mice exposed to 8130-9140 ppm 1,2,4-trimethylbenzene (no duration given) had loss of righting response and loss of reflexes Direct dermal contact with the chemical (no species given) causes vasodilation, erythema, and irritation (U.S. EPA). Seven of 10 rats died after an oral dose of 2.5 mL of a mixture of trimethylbenzenes in olive oil (average dose approximately 4.4 q/kg). Rats and mice were exposed by inhalation to a coal tar distillate containing about 70% 1,3,5- and 1,2,4-trimethylbenzene; no pathological changes were noted in either species after exposure to 1800-2000 ppm for up to 48 continuous hours, or in rats after 14 exposures of 8 hours each at the same exposure levels . No effects were reported for rats exposed to a mixture of trimethyl- benzenes at 1700 ppm for 10 to 21 days

Neurotoxicity 1.2.4-Trimethylbenzene depresses the central nervous system. Exposure to solvent mixtures containing the chemical causes headache. fatigue, nervousness, and drowsiness. Occupationally, workers exposed to a solvent containing 50% 1.2.4-trimethylbenzene had nervousness, headaches, drowsiness, and vertigo (U.S. EPA). Headache, fatigue, and drowsiness were reported for workers exposed (no dose given) to paint thinner containing 80% 1.2.4- and 1.3.5-trimethylbenzenes

Results of the developmental toxicity study indicate that the C9 fraction caused adverse neurological effects at the highest dose (1500 ppm) tested. Subchronic/Chronic Toxicity Long-term exposure to solvents containing 1,2,4-trimethylbenzene may cause nervousness, tension, and bronchitis. Painters that worked for several years with a solvent containing 50% 1,2,4- and 30% 1,3,5-trimethylbenzene showed nervousness, tension and anxiety, asthmatic bronchitis, anemia, and alterations in blood clotting; haematological effects may have been due to trace amounts of benzene Rats given 1,2,4-trimethylbenzene orally at doses of 0.5 or 2.0 g/kg/day, 5 days/week for 4 weeks. All rats exposed to the high dose died and 1 rat in the low dose died (no times given); no other effects were reported. Rats exposed by inhalation to 1700 ppm of a trimethylbenzene isomeric mixture for 4 months had decreased weight gain, lymphopenia and neutrophilia.

Genotoxicity: Results of mutagenicity testing, indicate that the C9 fraction does not induce gene mutations in prokaryotes (Salmonella tymphimurium/mammalian microsome assay); or in mammalian cells in culture (in Chinese hamster ovary cells with and without activation). The C9 fraction does not does not induce chromosome mutations in Chinese hamster ovary cells with and without activation; does not induce chromosome aberrations in the bone marrow of Sprague-Dawley rats exposed by inhalation (6 hours/day for 5 days); and does not induce sister chromatid exchange in Chinese hamster ovary cells with and without activation.

Developmental/Reproductive Toxicity: A three-generation reproductive study on the C9 fraction was conducted CD rats (30/sex/group) were exposed by inhalation to the C9 fraction at concentrations of 0, 100, 500, or 1500 ppm (0, 100, 500, or 1500 mg/kg/day) for 6 hours/day, 5 days/week. There was evidence of parental and reproductive toxicity at all dose levels. Indicators of parental toxicity included reduced body weights, increased salivation, hunched posture, aggressive behavior, and death. Indicators of adverse reproductive system effects included reduced litter size and reduced pup body weight. The LOEL was 100 ppm; a no-observed-effect level was not established. Developmental toxicity, including possible develop- mental neurotoxicity, was evident in rats in a 3-generation reproductive study

No effects on fecundity or fertility occurred in rats treated dermally with up to 0.3 mL/rat/day of a mixture of trimethyl- benzenes, 4-6 hours/day, 5 days/week over one generation

For C9 aromatics (typically trimethylbenzenes - TMBs)

Acute Toxicity

Acute toxicity studies (oral, dermal and inhalation routes of exposure) have been conducted in rats using various solvent products containing predominantly mixed C9 aromatic hydrocarbons (CAS RN 64742-95-6). Inhalation LC50's range from 6,000 to 10,000 mg/m 3 for C9 aromatic naphtha and 18,000 to 24,000 mg/m3 for 1,2,4 and 1,3,5-TMB, respectively. A rat oral LD50 reported for 1,2,4-TMB is 5 grams/kg bw and a rat dermal LD50 for the C9 aromatic naphtha is >4 ml/kg bw. These data indicate that C9 aromatic solvents show that LD50/LC50 values are greater than limit doses for acute toxicity studies established under OECD test guidelines.

Irritation and Sensitization

Several irritation studies, including skin, eye, and lung/respiratory system, have been conducted on members of the category. The results indicate that C9 aromatic hydrocarbon solvents are mildly to moderately irritating to the skin, minimally irritating to the eye, and have the potential to irritate the respiratory tract and cause depression of respiratory rates in mice. Respiratory irritation is a key endpoint in the current occupational exposure limits established for C9 aromatic hydrocarbon solvents and trimethylbenzenes. No evidence of skin sensitization was identified. Repeated Dose Toxicity

Inhalation: The results from a subchronic (3 month) neurotoxicity study and a one-year chronic study (6 hr/day, 5 days/week) indicate that effects from inhalation exposure to C9 Aromatic Hydrocarbon Solvents on systemic toxicity are slight. A battery of neurotoxicity and neurobehavioral endpoints were evaluated in the 3-month inhalation study on C9 aromatic naphtha tested at concentrations of 0, 101, 452, or 1320 ppm (0, 500, 2,220, or 6,500 mg/m3). In this study, other than a transient weight reduction in the high exposure group (not statistically significant at termination of exposures), no effects were reported on neuropathology or neuro/behavioral parameters. The NOAEL for systemic and/or neurotoxicity was 6,500 mg/m3, the highest concentration tested. In an inhalation study of a commercial blend, rats were exposed to C9 aromatic naphtha concentrations of 0, 96, 198, or 373 ppm (0, 470, 970, 1830) mg/m3) for 6 hr/day, 5 days/week, for 12 months. Liver and kidney weights were increased in the high exposure group but no accompanying histopathology was observed in these organs.

The NOAEL was considered to be the high exposure level of 373 ppm, or 1830 mg/m3. In two subchronic rat inhalation studies, both of three months duration, rats were exposed to the individual TMB isomers (1,2,4-and 1,3,5-) to nominal concentrations of 0, 25, 100, or 250 ppm (0, 123, 492, or 1230 mg/m3). Respiratory irritation was observed at 492 (100 ppm) and 1230 mg/m3 (250 ppm) and no systemic toxicity was observed in either study. For both pure isomers, the NOELs are 25 ppm or 123 mg/m3 for respiratory irritation and 250 ppm or 1230 mg/m3 for systemic effects. Oral: The C9 aromatic naphtha has not been tested via the oral route of exposure. Individual TMB isomers have been evaluated in a series of repeated-dose

oral studies ranging from 14 days to 3 months over a wide range of doses. The effects observed in these studies included increased liver and kidney

NAPHTHA PETROLEUM. LIGHT AROMATIC SOLVENT

Continued...

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weights, changes in blood chemistry, increased salivation, and decreased weight gain at higher doses. Organ weight changes appeared to be adaptive as they were not accompanied by histopathological effects. Blood changes appeared sporadic and without pattern. One study reported hyaline droplet nephropathy in male rats at the highest dose (1000 mg/kg bw-day), an effect that is often associated with alpha-2mu-globulin-induced nephropathy and not considered relevant to humans. The doses at which effects were detected were 100 mg/kg-bw day or above (an exception was the pilot 14 day oral study LOAEL 150 mg/kg bw-day - but the follow up three month study had a LOAEL of 600 mg/kg/bw-day with a NOAEL of 200 mg/kg bw-day). Since effects generally were not severe and could be considered adaptive or spurious, oral exposure does not appear to pose a high toxicity hazard for pure trimethylbenzene isomers.

Mutagenicity

In vitro genotoxicity testing of a variety of C9 aromatics has been conducted in both bacterial and mammalian cells. In vitro point mutation tests were conducted with Salmonella typhimurium and Escherichia coli bacterial strains, as well as with cultured mammalian cells such as the Chinese hamster cell ovary cells (HGPRT assay) with and without metabolic activation. In addition, several types of in vitro chromosomal aberration tests have been performed (chromosome aberration frequency in Chinese hamster ovary and lung cells, sister chromatid exchange in CHO cells). Results were negative both with and without metabolic activation for all category members. For the supporting chemical 1,2,3-TMB, a single in vitro chromosome aberration test was weakly positive. In in vivo bone marrow cytogenetics test, rats were exposed to C9 aromatic naphtha at concentrations of 0, 153, 471, or 1540 ppm (0, 750, 2,310, or 7,560 mg/m3) 6 hr/day, for 5 days. No evidence of in vivo somatic cell genotoxicity was detected. Based on the cumulative results of these assays, genetic toxicity is unlikely for substances in the C9 Aromatic Hydrocarbon Solvents Category Reproductive and Developmental Toxicity

Results from the three-generation reproduction inhalation study in rats indicate limited effects from C9 aromatic naphtha. In each of three generations (F0, F1 and F2), rats were exposed to High Flash Aromatic Naphtha (CAS RN 64742-95-6) via whole body inhalation at target concentrations of 0, 100, 500, or 1500 ppm (actual mean concentrations throughout the full study period were 0, 103, 495, or 1480 ppm, equivalent to 0, 505, 2430, or 7265 mg/m3, respectively). In each generation, both sexes were exposed for 10 weeks prior to and two weeks during mating for 6 hrs/day, 5 days/wks. Female rats in the F0, F1, and F2 generation were then exposed during gestation days 0-20 and lactation days 5-21 for 6 hrs/day, 7 days/wk. The age at exposure initiation differed among generations; F0 rats were exposed starting at 9 weeks of age, F1 exposure began at 5-7 weeks, and F2 exposure began at postnatal day (PND) 22. In the F0 and F1 parental generations, 30 rats/sex/group were exposed and mated. However, in the F2 generation, 40/sex/group were initially exposed due to concerns for toxicity, and 30/sex/group were randomly selected for mating, except that all survivors were used at 1480 ppm. F3 litters were not exposed directly and were sacrificed on lactation day 21. Systemic Effects on Parental Generations:

The F0 males showed statistically and biologically significantly decreased mean body weight by ~15% at 1480 ppm when compared with controls. Seven females died or were sacrificed in extremis at 1480 ppm. The F0 female rats in the 495 ppm exposed group had a 13% decrease in body weight gain when adjusted for initial body weight when compared to controls. The F1 parents at 1480 ppm had statistically significantly decreased mean body weights (by ~13% (females) and 22% (males)), and locomotor activity. F1 parents at 1480 ppm had increased ataxia and mortality (six females). Most F2 parents (70/80) exposed to 1480 ppm died within the first week. The remaining animals survived throughout the rest of the exposure period. At week 4 and continuing through the study, F2 parents at 1480 ppm had statistically significant mean body weights much lower than controls (~33% for males; ~28% for females); body weights at 495 ppm were also reduced significantly (by 13% in males and 15% in females). The male rats in the 495 ppm exposed group had a 12% decrease in body weight gain when adjusted for initial body weight when compared to controls. Based on reduced body weight observed, the overall systemic toxicity LOAEC is 495 ppm (2430 mg/m3).

Reproductive Toxicity-Effects on Parental Generations: There were no pathological changes noted in the reproductive organs of any animal of the F0, F1, or F2 generation. No effects were reported on sperm morphology, gestational period, number of implantation sites, or post-implantation loss in any generation. Also, there were no statistically or biologically significant differences in any of the reproductive parameters, including: number of mated females, copulatory index, copulatory interval, number of females delivering a litter, number of females delivering a live litter, or male fertility in the F0 or in the F2 generation. Male fertility was statistically significantly reduced at 1480 ppm in the F1 rats. However, male fertility was not affected in the F0 or in the F2 generations; therefore, the biological significance of this change is unknown and may or may not be attributed to the test substance. No reproductive effects were observed in the F0 or F1 dams exposed to 1480 ppm (7265 mg/m3). Due to excessive mortality at the highest concentration (1480 ppm, only six dams available) in the F2 generation,, a complete evaluation is precluded. However, no clear signs of reproductive toxicity were observed in the F2 generation. Therefore, the reproductive NOAEC is considered 495 ppm (2430 mg/m3), which excludes analysis of the highest concentration due to

Developmental Toxicity - Effects on Pups: Because of significant maternal toxicity (including mortality) in dams in all generations at the highest concentration (1480 ppm), effects in offspring at 1480 ppm are not reported here. No significant effects were observed in the F1 and F2 generation offspring at 103 or 495 ppm. However, in F3 offspring, body weights and body weight gain were reduced by ~ 10-11% compared with controls at 495 ppm for approximately a week (PND 14 through 21). Maternal body weight was also depressed by ~ 12% throughout the gestational period compared with controls. The overall developmental LOAEC from this study is 495 ppm (2430 mg/m3) based on the body weights reductions observed in the F3 offspring. Conclusion: No effects on reproductive parameters were observed at any exposure concentration, although a confident assessment of the group exposed at the highest concentration was not possible. A potential developmental effect (reduction in mean pup weight and weight gain) was observed at a concentration that was also associated with maternal toxicity. Inhalation (rat) TCLo: 1320 ppm/6h/90D-I * [Devoe]

Alcohol ethoxylates are according to CESIO (2000) classified as Irritant or Harmful depending on the number of EO-units:

EO < 5 gives Irritant (Xi) with R38 (Irritating to skin) and R41 (Risk of serious damage to eyes)

EO > 5-15 gives Harmful (Xn) with R22 (Harmful if swallowed) - R38/41

EO > 15-20 gives Harmful (Xn) with R22-41

>20 EO is not classified (CESIO 2000)

Oxo-AE, C13 EO10 and C13 EO15, are Irritating (Xi) with R36/38 (Irritating to eyes and skin) .

AE are not included in Annex 1 of the list of dangerous substances of the Council Directive 67/548/EEC

In general, alcohol ethoxylates (AE) are readily absorbed through the skin of guinea pigs and rats and through the gastrointestinal mucosa of rats. AE are quickly eliminated from the body through the urine, faeces, and expired air (CO2). Orally dosed AE was absorbed rapidly and extensively in rats, and more than 75% of the dose was absorbed. When applied to the skin of humans, the doses were absorbed slowly and incompletely (50% absorbed in 72 hours). Half of the absorbed surfactant was excreted promptly in the urine and smaller amounts of AE appeared in the faeces and expired air (CO2)). The metabolism of C12 AE yields PEG, carboxylic acids, and CO2 as metabolites. The LD50 values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.

POLYPROPYLENE GLYCOL MONOMYRISTYL ETHER **PROPIONATE**

The ability of nonionic surfactants to cause a swelling of the stratum comeum of guinea pig skin has been studied. The swelling mechanism of the skin involves a combination of ionic binding of the hydrophilic group as well as hydrophobic interactions of the alkyl chain with the substrate. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants. A substantial amount of toxicological data and information in vivo and in vitro demonstrates that there is no evidence for alcohol ethoxylates (AEs) being genotoxic, mutagenic or carcinogenic. No adverse reproductive or developmental effects were observed. The majority of available toxicity studies revealed NOAELs in excess of 100 mg/kg bw/d but the lowest NOAEL for an individual AE was established to be 50 mg/kg bw/day. This value was subsequently considered as a conservative, representative value in the risk assessment of AE. The effects were restricted to changes in organ weights with no histopathological organ changes with the exception of liver hypertrophy (indicative of an adaptive response to metabolism rather than a toxic effect). It is noteworthy that there was practically no difference in the NOAEL in oral studies of 90-day or 2 years of duration in rats. A comparison of the aggregate consumer exposure and the systemic NOAEL (taking into account an oral absorption value of 75%) results in a Margin of Exposure of 5,800. Taking into account the conservatism in the exposure assessment and the assigned systemic NOAEL, this margin of exposure is considered more than adequate to account for the inherent uncertainty and variability of the hazard database and inter and intra-species extrapolations.

AEs are not contact sensitisers. Neat AE are irritating to eyes and skin. The irritation potential of aqueous solutions of AEs depends on concentrations. Local dermal effects due to direct or indirect skin contact in certain use scenarios where the products are diluted are not of concern as AEs are not

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| | expected to be irritating to the skin at in-use concentrations. Potential irritation of the respiratory tract is not a concern given the very low levels of airborne AE generated as a consequence of spray cleaner aerosols or laundry powder detergent dust. In summary, the human health risk assessment has demonstrated that the use of AE in household laundry and cleaning detergents is safe and does not cause concern with regard to consumer use. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. | | |
|-----------------------------------|---|--------------------------|---|
| ISOBUTYLENE HOMOPOLYMER | No significant acute toxicological data identified in literature search. | | |
| Acute Toxicity | 0 | Carcinogenicity | 0 |
| Skin Irritation/Corrosion | ✓ | Reproductivity | 0 |
| Serious Eye Damage/Irritation | ✓ | STOT - Single Exposure | ✓ |
| Respiratory or Skin sensitisation | 0 | STOT - Repeated Exposure | 0 |
| Mutagenicity | 0 | Asniration Hazard | ٠ |

Legend:

X − Data available but does not fill the criteria for classification
 v − Data available to make classification

Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

| 0.1.4.6.0.0.0.6.0.6.0.0.44. | ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURC |
|---|------------------|--------------------|-------------------------------|------------------|------------------|
| Cydectin Pour-On for Cattle and Red Deer | Not Available | Not Available | Not Available | Not Available | Not Available |
| | ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURC |
| moxidectin | Not Available | Not Available | Not Available | Not Available | Not Available |
| | ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURC |
| | EC50 | 48 | Crustacea | =6.14mg/L | 1 |
| naphtha petroleum, light aromatic solvent | EC50 | 72 | Algae or other aquatic plants | 3.29mg/L | 1 |
| | EC10 | 72 | Algae or other aquatic plants | 1.13mg/L | 1 |
| | NOEC | 72 | Algae or other aquatic plants | =1mg/L | 1 |
| | ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURC |
| polypropylene glycol nonomyristyl ether propionate | Not Available | Not Available | Not Available | Not Available | Not Available |
| isobutylene homopolymer | ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURC |
| | LC50 | 96 | Fish | >5600mg/L | 4 |

(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

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

Persistence and degradability

| Ingredient | Persistence: Water/Soil | Persistence: Air |
|-------------------------|-------------------------|------------------|
| isobutylene homopolymer | LOW | LOW |

Bioaccumulative potential

| Ingredient | Bioaccumulation |
|-------------------------|-----------------------|
| isobutylene homopolymer | LOW (LogKOW = 2.2256) |

Mobility in soil

| Ingredient | Mobility |
|-------------------------|-------------------|
| isobutylene homopolymer | LOW (KOC = 35.04) |

SECTION 13 DISPOSAL CONSIDERATIONS

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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)

Product / Packaging disposal

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 | NO |
|------------------|----------------|
| HAZCHEM | Not Applicable |

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

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

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

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

MOXIDECTIN(113507-06-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists

NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT(64742-95-6.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

POLYPROPYLENE GLYCOL MONOMYRISTYL ETHER PROPIONATE(74775-06-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

ISOBUTYLENE HOMOPOLYMER(9003-27-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

| National Inventory | Status |
|-------------------------------|---|
| Australia - AICS | N (moxidectin) |
| Canada - DSL | N (moxidectin) |
| Canada - NDSL | N (moxidectin; isobutylene homopolymer; naphtha petroleum, light aromatic solvent; polypropylene glycol monomyristyl ether propionate) |
| China - IECSC | N (moxidectin; polypropylene glycol monomyristyl ether propionate) |
| Europe - EINEC / ELINCS / NLP | N (moxidectin) |
| Japan - ENCS | N (moxidectin; polypropylene glycol monomyristyl ether propionate) |
| Korea - KECI | N (moxidectin; polypropylene glycol monomyristyl ether propionate) |
| New Zealand - NZIoC | Y |
| Philippines - PICCS | N (moxidectin) |
| USA - TSCA | N (moxidectin; polypropylene glycol monomyristyl ether propionate) |
| Legend: | Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets) |

SECTION 16 OTHER INFORMATION

Other information

Ingredients with multiple cas numbers

| Name | CAS No |
|---|--------------------------|
| naphtha petroleum, light aromatic solvent | 64742-95-6., 25550-14-5. |
| isobutylene homopolymer | 9003-27-4, 9003-29-6 |

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

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

Definitions and abbreviations

PC-TWA: Permissible Concentration-Time Weighted Average

PC-STEL: Permissible Concentration-Short Term Exposure Limit

IARC: International Agency for Research on Cancer

ACGIH: American Conference of Governmental Industrial Hygienists

STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit。

IDLH: Immediately Dangerous to Life or Health Concentrations

OSF: Odour Safety Factor

NOAEL: No Observed Adverse Effect Level LOAEL: Lowest Observed Adverse Effect Level

TLV: Threshold Limit Value LOD: Limit Of Detection

OTV: Odour Threshold Value BCF: BioConcentration Factors BEI: Biological Exposure Index

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