

AC Consort 400 Herbicide

Axichem Pty Ltd

Chemwatch: 5315-90

Version No: 8.1

Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Chemwatch Hazard Alert Code: 3

Issue Date: 27/10/2023

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L.GHS.AUS.EN

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

Product Identifier

| | |
|-------------------------------|--|
| Product name | AC Consort 400 Herbicide |
| Chemical Name | Not Applicable |
| Synonyms | Not Available |
| Proper shipping name | ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains carfentrazone-ethyl) |
| Chemical formula | Not Applicable |
| Other means of identification | Not Available |

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

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| Relevant identified uses | For improvement in the control of marshmallow and certain other broadleaf weeds prior to establishment of broadacre crops, fallows or forest plantations. |
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Details of the manufacturer or supplier of the safety data sheet

| | |
|-------------------------|--|
| Registered company name | Axichem Pty Ltd |
| Address | 9 Palings Court Nerang QLD 4211 Australia |
| Telephone | 07 5596 1736 |
| Fax | Not Available |
| Website | www.axichem.com.au |
| Email | msds@axichem.com.au |

Emergency telephone number

| | |
|-----------------------------------|-------------------------------------|
| Association / Organisation | CHEMWATCH EMERGENCY RESPONSE (24/7) |
| Emergency telephone numbers | +61 1800 951 288 |
| Other emergency telephone numbers | +61 3 9573 3188 |

Once connected and if the message is not in your preferred language then please dial 01

SECTION 2 Hazards identification

Classification of the substance or mixture

COMBUSTIBLE LIQUID, regulated for storage purposes only

| | |
|-------------------------------|--|
| Poisons Schedule | S5 |
| Classification ^[1] | Flammable Liquids Category 4, Aspiration Hazard Category 1, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1A, Serious Eye Damage/Eye Irritation Category 2A, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Reproductive Toxicity Category 1A, Hazardous to the Aquatic Environment Long-Term Hazard Category 1 |
| Legend: | 1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI |

Label elements

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|---------------------|---|
| Hazard pictogram(s) |  |
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|-------------|--------|
| Signal word | Danger |
|-------------|--------|

Hazard statement(s)

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| H227 | Combustible liquid. |
| H304 | May be fatal if swallowed and enters airways. |
| H315 | Causes skin irritation. |
| H317 | May cause an allergic skin reaction. |
| H319 | Causes serious eye irritation. |
| H335 | May cause respiratory irritation. |
| H360Df | May damage the unborn child. Suspected of damaging fertility. |
| H410 | Very toxic to aquatic life with long lasting effects. |

Precautionary statement(s) Prevention

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| P201 | Obtain special instructions before use. |
| P210 | Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking. |
| P271 | Use only outdoors or in a well-ventilated area. |
| P280 | Wear protective gloves, protective clothing, eye protection and face protection. |
| P261 | Avoid breathing mist/vapours/spray. |
| P273 | Avoid release to the environment. |
| P264 | Wash all exposed external body areas thoroughly after handling. |
| P272 | Contaminated work clothing should not be allowed out of the workplace. |

Precautionary statement(s) Response

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|----------------|--|
| P301+P310 | IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider. |
| P331 | Do NOT induce vomiting. |
| P308+P313 | IF exposed or concerned: Get medical advice/ attention. |
| P370+P378 | In case of fire: Use alcohol resistant foam or normal protein foam to extinguish. |
| 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. |
| P312 | Call a POISON CENTER/doctor/physician/first aider/if you feel unwell. |
| 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. |
| P304+P340 | IF INHALED: Remove person to fresh air and keep comfortable for breathing. |

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

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| P501 | Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation. |
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SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

| CAS No | %[weight] | Name |
|---|-----------|-------------------------------|
| 128639-02-1 | 30-50 | <u>carfentrazone-ethyl</u> |
| Not Available | | 400 g/L) |
| Various | 30-50 | <u>liquid hydrocarbons</u> |
| 872-50-4 | 10-20 | <u>N-methyl-2-pyrrolidone</u> |
| 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

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| Eye Contact | If this product comes in contact with the eyes: <ul style="list-style-type: none"> Wash out immediately with fresh running water. Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. Seek medical attention without delay; if pain persists or recurs seek medical attention. Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. |
| Skin Contact | If skin contact occurs: <ul style="list-style-type: none"> Immediately remove all contaminated clothing, including footwear. Flush skin and hair with running water (and soap if available). Seek medical attention in event of irritation. |
| Inhalation | <ul style="list-style-type: none"> If fumes or combustion products are inhaled remove from contaminated area. Lay patient down. Keep warm and rested. 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 | <ul style="list-style-type: none"> If swallowed do NOT induce vomiting. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. Observe the patient carefully. Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. Seek medical advice. |

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

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

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| Fire Incompatibility | <ul style="list-style-type: none"> Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result |
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Advice for firefighters

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| Fire Fighting | <ul style="list-style-type: none"> Alert Fire Brigade and tell them location and nature of hazard. Wear breathing apparatus plus protective gloves. 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. |
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| | <ul style="list-style-type: none"> 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 | <ul style="list-style-type: none"> 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 acid smoke. Mists containing combustible materials may be explosive. <p>Combustion products include: carbon dioxide (CO₂) nitrogen oxides (NO_x) hydrogen chloride fluorides other pyrolysis products typical of burning organic material.</p> |
| HAZCHEM | •3Z |

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

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| Minor Spills | <p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> 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 | <p>Environmental hazard - contain spillage. Moderate hazard.</p> <ul style="list-style-type: none"> Clear area of personnel and move upwind. Alert Fire Brigade and tell them location and nature of hazard. Wear breathing apparatus plus protective gloves. Prevent, by any means available, spillage from entering drains or water course. 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

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|---------------|--|
| Safe handling | <ul style="list-style-type: none"> DO NOT allow clothing wet with material to stay in contact with skin Avoid all personal contact, including inhalation. Wear protective clothing when risk of exposure occurs. Use in a well-ventilated area. 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. |
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| | <ul style="list-style-type: none">▶ 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 | <ul style="list-style-type: none">▶ Store in original containers.▶ Keep containers securely sealed.▶ No smoking, naked lights or ignition sources.▶ Store in a cool, dry, well-ventilated area.▶ Store away from incompatible materials and foodstuff containers.▶ Protect containers against physical damage and check regularly for leaks.▶ Observe manufacturer's storage and handling recommendations contained within this SDS. |

Conditions for safe storage, including any incompatibilities

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| Suitable container | <ul style="list-style-type: none">▶ Metal can or drum▶ Packaging as recommended by manufacturer.▶ Check all containers are clearly labelled and free from leaks. |
| Storage incompatibility | <ul style="list-style-type: none">▶ Avoid reaction with oxidising agents |

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

| Source | Ingredient | Material name | TWA | STEL | Peak | Notes |
|------------------------------|------------------------|------------------------|--------------------|--------------------|---------------|---------------|
| Australia Exposure Standards | N-methyl-2-pyrrolidone | 1-Methyl-2-pyrrolidone | 25 ppm / 103 mg/m3 | 309 mg/m3 / 75 ppm | Not Available | Not Available |

Emergency Limits

| Ingredient | TEEL-1 | TEEL-2 | TEEL-3 |
|------------------------|--------|--------|---------|
| N-methyl-2-pyrrolidone | 30 ppm | 32 ppm | 190 ppm |

| Ingredient | Original IDLH | Revised IDLH |
|------------------------|---------------|---------------|
| carfentrazone-ethyl | Not Available | Not Available |
| liquid hydrocarbons | Not Available | Not Available |
| N-methyl-2-pyrrolidone | Not Available | Not Available |

Occupational Exposure Banding


| Ingredient | Occupational Exposure Band Rating | Occupational Exposure Band Limit |
|---------------------|-----------------------------------|----------------------------------|
| carfentrazone-ethyl | E | ≤ 0.1 ppm |

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| 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. |
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MATERIAL DATA

Exposure controls

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| Appropriate engineering controls | <p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in specific circumstances. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. 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</p> |
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| | 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. | |
| Individual protection measures, such as personal protective equipment |  | |
| Eye and face protection | <ul style="list-style-type: none">▶ Safety glasses with side shields.▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59]. | |
| Skin protection | See Hand protection below | |
| Hands/feet protection | <ul style="list-style-type: none">▶ Wear chemical protective gloves, e.g. PVC.▶ Wear safety footwear or safety gumboots, e.g. Rubber <p>NOTE:</p> <ul style="list-style-type: none">▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none">· frequency and duration of contact,· chemical resistance of glove material,· glove thickness and· dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none">· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.· Contaminated gloves should be replaced. <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> | |

| | |
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| | <ul style="list-style-type: none"> · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> |
| Body protection | See Other protection below |
| Other protection | <ul style="list-style-type: none"> ▸ Overalls. ▸ P.V.C apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit. |

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

The effect(s) of the following substance(s) are taken into account in the

computer-generated selection:

AC Consort 400 Herbicide

| Material | CPI |
|----------------|-----|
| BUTYL | A |
| PE/EVAL/PE | A |
| NATURAL RUBBER | B |
| PVA | B |

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

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

^ - Full-face

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

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

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| Appearance | Yellow to orange liquid; emulsifies with water. |
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|--|----------------|---|----------------|
| 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 Applicable | Decomposition temperature (°C) | Not Available |
| Melting point / freezing point (°C) | Not Available | Viscosity (cSt) | Not Available |
| Initial boiling point and boiling range (°C) | Not Available | Molecular weight (g/mol) | Not Applicable |
| Flash point (°C) | >61 | Taste | Not Available |
| Evaporation rate | Not Available | Explosive properties | Not Available |
| Flammability | Combustible. | 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 | Miscible | pH as a solution (1%) | Not Available |
| Vapour density (Air = 1) | Not Available | VOC g/L | Not Available |

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

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|--------------|--|
| Inhaled | <p>Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination. Inhalation of high vapour concentrations of N-methyl-2-pyrrolidone (NMP) may produce mucous membrane irritation, headache, giddiness, mental confusion and nausea. Fatalities were not recorded following inhalation of 180-200 mg/m³ for 2 hours by mice and following a 6 hour exposure to saturated vapours by rats.</p> <p>Laboratory animals exposed to concentrations of 50 ppm for 8 hours daily for 20 days or 370 ppm for 6 hours daily for 10 days showed no gross or histopathological abnormalities.</p> |
| Ingestion | <p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>Ingestion may result in nausea, pain, vomiting. Vomit entering the lungs by aspiration may cause potentially lethal chemical pneumonitis.</p> |
| Skin Contact | <p>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.</p> <p>The material may accentuate any pre-existing dermatitis condition.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material.</p> <p>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.</p> |

Eye

Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals.

Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.

Chronic

There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of:

- clear results in appropriate animal studies where effects 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.

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.

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.

Hydrocarbon solvents are liquid hydrocarbon fractions derived from petroleum processing streams, containing only carbon and hydrogen atoms, with carbon numbers ranging from approximately C5-C20 and boiling between approximately 35-370 deg C. Many of the hydrocarbon solvents have complex and variable compositions with constituents of 4 types, alkanes (normal paraffins, isoparaffins, and cycloparaffins) and aromatics (primarily alkylated one- and two-ring species). Despite the compositional complexity, most hydrocarbon solvent constituents have similar toxicological properties, and the overall toxicological hazards can be characterized in generic terms. Hydrocarbon solvents can cause chemical pneumonitis if aspirated into the lung, and those that are volatile can cause acute CNS effects and/or ocular and respiratory irritation at exposure levels exceeding occupational recommendations. Otherwise, there are few toxicologically important effects. The exceptions, n-hexane and naphthalene, have unique toxicological properties

Animal studies:

No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) was observed in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of a alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human.

The teratogenic potential, subchronic and long term inhalation toxicity of N-methyl-2-pyrrolidone (NMP) has been studied in rats. No evidence of nephrotoxicity was seen.

No carcinogenic effects were observed. Very high doses are embryotoxic to rats and mice. Reproductive effects have been reported in animals.

Azole fungicides are broad spectrum antifungal compounds used in agriculture and in human and veterinary medicine. The mechanism of antifungal action relies on inhibition of CYP51 (sterol 14-demethylase), resulting in inhibition of fungal cell growth. Known adverse health effects of azole fungicides are mainly linked to cytochrome P450 (CYP) inhibition. The relatively poor selectivity of the agricultural azoles for the fungal CYP51 over the human homolog raises the concern that exposure to azole fungicide residues might disrupt sterol biosynthesis and other endogenous downstream cytochrome P450 metabolic systems, such as human steroidogenesis and phase I metabolism of xenobiotics in the liver.

Agricultural azoles have the general ability to inhibit the P450 enzymes in steroidogenesis, albeit with different potencies. Since the introduction of large-scale use of azole antifungals increasing evidence of hepatotoxicity and associated hepatic tumors has been reported with liver tissue concentrations of ketoconazole and itraconazole being reported as 22- and 10-fold higher, respectively, than plasma levels indicating azole toxicity in the liver being more acute than in other tissues.

Additionally, azole fungicide-induced neurotoxicity has been reported. Several azole fungicides induced a nonspecific inhibition of voltage-gated calcium channels (VGCCs), each with varying potency. These findings demonstrate modulation of intracellular calcium ion via inhibition of VGCCs as a novel mode of action of azole fungicides. Calcium plays a pivotal role in neuronal survival and functioning.

Triazoles are important antifungal compounds, and all drugs in this class inhibit ergosterol synthesis by blocking the 14-alpha-demethylase enzyme, resulting in the accumulation of toxic methylsterols that may culminate in fungal death.

Triazole pesticides all contain a triazole ring with nitrogen atoms at the 1,2 and 4 positions. 1,2,4-Triazole (1,2,4-T) and its conjugates, triazole alanine (TA), triazole acetic acid (TAA), triazole pyruvic acid, and triazole lactic acid are the metabolic products of plant, fungal and animal bioconversion. These compounds all possess potentially significant toxicological properties. Following application of a triazole-derivative fungicide, biological and/or chemical processes may cause the triazole ring to be

released from the parent compound. In rats and livestock, 1,2,4-triazole is relatively stable and is the terminal form of the triazole ring. In plants, the 1,2,4-triazole molecule may become conjugated to serine. The resulting compound, triazole alanine, may be oxidised to form triazole acetic acid. Triazole alanine and triazole acetic acid are the primary terminal forms of the triazole ring in plants, though some 1,2,4-triazole may remain. The degree of formation of any given form of the triazole ring is highly dependent on the nature and properties of the parent compound. Although other triazole conjugates such as triazole lactic acid and triazole pyruvate have been observed in plant metabolism studies, TA and TAA are the predominant conjugates that need to be included in the dietary risk assessment.

Although for most pesticides, mammals convert only a small proportion to free triazole (less than 25%), two compounds (tetraconazole and flusilazole) demonstrate relatively high conversion (68-77%) in rat metabolism studies.

Available acute data indicate that 1,2,4-triazole is slightly toxic by the oral route (with oral LD50 values ranging from 666 mg/kg in rabbits to 3650 mg/kg in mice) and slightly to moderately toxic by the dermal route (dermal LD50s were less than 2000 mg/kg in rabbits, and 3000-4000 mg/kg in rats). Limited available information indicates that 1,2,4-triazole is slightly irritating or non-irritating to the skin, but severely irritating to the eye. Based on the limited acute toxicity data, as well as the available developmental toxicity data (see below), it appears that rabbits may be substantially more susceptible to 1,2,4-triazole than are rats or mice.

Studies indicate that 1,2,4-triazole affects the central and peripheral nervous systems, reproductive tissues of both sexes, and the hematological system. Developmental and reproductive effects have been noted for this compound. Based on the available metabolism data from rats and livestock, 1,2,4-triazole may form in humans following exposure to parent triazole compounds. Relative to triazole alanine, fewer studies are available depicting the toxicological effects of the other triazole conjugates. It is assumed that the triazole conjugates are all toxicologically equivalent to triazole alanine. The available studies found developmental skeletal effects, decreased body weight and body weight gain, and decreased leukocytes and triglycerides.

A number of target organs and critical effects have been identified. 1,2,4-triazole targets the nervous system, both central and peripheral, as brain lesions (most notably in the cerebellum) were seen in both rats and mice, and peripheral nerve degeneration was also seen in the subchronic neurotoxicity study in rats. In addition, brain weight decreases were seen in several studies, including in the offspring in the reproductive toxicity study. In the subchronic/neurotoxicity study, there is evidence that effects progress over time, with an increase in incidence of clinical signs (including tremors and muscle fasciculations) during weeks 8 and 13 that were not seen during earlier evaluations.

There is no evidence that exposure to triazole alanine results in neurotoxicity. No clinical signs of neurotoxicity, changes in brain weights, changes in brain gross or microscopic pathology, or any other neurotoxic effects were observed in the short-term rat studies, the subchronic rat and dog feeding studies, the rat developmental toxicity study, or the two-generation reproduction study.

Effects were also seen on reproductive organs in both sexes, most notably ovaries (in rats) and testes (in rats and mice), in both the reproductive toxicity and subchronic toxicity studies. Hematological changes, including slightly decreased hemoglobin and/or hematocrit, have also been seen in multiple studies and species (in rats at doses of 33 mg/kg/day and above, and in mice at doses of 487 mg/kg/day and above).

1,2,4-triazole also causes developmental toxicity in both rats and rabbits, including malformations, at doses similar to those inducing maternal toxicity (decreased body weight gain in rats and clinical signs and mortality in rabbits). Developmental toxicity was also seen in the reproductive toxicity study, with offspring showing adverse effects on multiple endpoints (including decreased brain and body weight) at doses lower than those at which effects were seen in parents. In addition, reproductive toxicity was seen in both sexes: at the highest dose (3000 ppm), only two F1 litters (one pup/litter) were produced, and neither survived to adulthood.

Triazole alanine showed increased incidences of skeletal findings in the offspring at the mid and high doses, while no treatment-related effects were seen in the dams up to the limit dose. The skeletal findings included unossified odontoid processes at 300 and 1000 mg/kg/day, with partially ossified transverse processes of the 7th cervical vertebra (bilateral), unossified 5th sternebra, and partially ossified 13th thoracic centrum observed only at 1000 mg/kg/day.

Available mutagenicity data are limited but negative. A large number of parent triazole-derivative pesticides have been classified as carcinogens (most also non-mutagenic), but the relevance of that finding to expected effects of free triazole may be limited.

The types of tumors associated with exposure to the parent chemicals are most commonly hepatocellular adenomas/carcinomas in mice. Other tumor types vary considerably (including liver tumors, thyroid tumors, ovarian tumors, testicular tumors, and bladder tumors). None of the tumor types are clearly associated with the proportion of free triazole formed in available rat metabolism studies. Evidence indicates that the parent triazole compounds appear to result in a tumor response subsequent to perturbation of liver metabolism, specifically xenobiotic and fatty acid metabolic pathways. In addition the thyroid response appears to be secondary to perturbation of thyroid homeostasis. Thus, the conazoles appear to drive a tumor response secondary to epigenetic effects and not from direct interaction with the DNA. An epigenetic mode of action would be consistent with a nonlinear process.

Azole antifungals cause hepatotoxicity by inducing the expression of liver cytochrome P450 enzymes (CYP1, CYP2, and CYP3 families), which in turn increases the abundance of reactive oxygen species in liver cells, resulting in lipid peroxidation and DNA damage. In addition, azole antifungals have the potential to inhibit liver P450 enzymes, interfering in the phase I metabolism of xenobiotics.

Disruption of the endocrine system, by azoles, can lead to impaired reproduction, alterations in sexual differentiation, impaired growth and development, and the formation of hormone-dependent cancers. Azole antifungals disrupt the endocrine system by inhibiting several highly substrate-selective cytochrome P450 enzymes involved in mammalian steroid hormone biosynthesis.

These include aromatase (CYP19) (which catalyzes the C-10 demethylation of androgens to estrogens), CYP11A (which converts cholesterol to pregnenolone), steroid 21-hydroxylase (CYP21), aldosterone synthase (CYP11B2), steroid 11 β -hydroxylase (CYP11B1), steroid 17 α -hydroxylase/17,20-lyase (CYP17), and lanosterol 14 α -demethylase (CYP51), disturbing the in vivo hormonal balance.

Azole fungicides show a broad antifungal activity and are used either to prevent fungal infections or to cure an infection.

Therefore, they are important tools in integrated agricultural production. According to their chemical structure, azole compounds are classified into triazoles and imidazoles; however, their antifungal activity is due to the same molecular mechanism. The difference between the imidazoles and the triazoles involves the mechanism of inhibition of the cytochrome P450 enzyme. The N3 of the imidazole compound binds to the heme iron atom of ferric cytochrome P450, whereas the N4 of the triazoles bind to the heme group. The triazoles have been shown to have a higher specificity for the cytochrome P450 than imidazoles, thereby

making them more potent than the imidazoles

The cell membrane assembly of fungi and yeast is disturbed by blocking the synthesis of the essential membrane component ergosterol. This fundamental biochemical mechanism is the basis for the use of azole fungicides in agriculture and in human and veterinary antimycotic therapies. The enzyme involved is sterol 14[alpha]-demethylase, which is found in several phyla. In mammals, it converts lanosterol into the meiosis-activating sterols (MAS) which regulate or modify cell division. These precursors of cholesterol have been discovered to moderate the development of male and female germ (sexual) cells. Several metabolites of lanosterol have been regarded only as precursors of cholesterol without any biological function in animals. This view dramatically changed recently with the observation that FF-MAS isolated from human follicle fluid and T-MAS isolated from bull testis as well as the MAS-412 and MAS-414 induced resumption of meiosis in cultivated mouse oocytes

Aromatase is another target enzyme of azole compounds. In steroidogenesis, it converts androgens into the corresponding oestrogens. The importance of androgens and oestrogens for the development of reproductive organs, for fertility, and in certain sex steroid-dependent diseases is well known. Therefore, azole compounds can be directed against aromatase to treat oestrogen-responsive diseases. Based on the inhibitory activity of azoles on key enzymes involved in sex steroid hormone synthesis, it is likely that effects on fertility, sexual behavior, and reproductive organ development will occur depending on dose level and duration of treatment of laboratory animals. Several azole compounds were shown to inhibit the aromatase and to disturb the balance of androgens and estrogens in vivo. In fact, the clinical use of azole compounds in estrogen-dependent diseases is based on this effect. Additionally, azole antifungals developed to inhibit the sterol 14[alpha]-demethylase of fungi and yeast in agriculture and medicine are also inhibiting aromatase. Therefore, these antifungals may unintentionally disturb the balance of androgens and estrogens. Until now, it is not clear whether this effect is compensated by an increased expression of aromatase or by other unknown mechanisms.

The broad use of biologically active compounds in human therapy as well as in nonhuman applications may involve some risks, as exemplified by emerging antibiotic resistance. In agriculture, fungi and yeast are well known to develop resistance to azoles, and some molecular mechanisms of resistance development have been described. The significance of the agricultural azole resistance for human clinical antimycotic therapies has been discussed in Europe, but is not clarified yet. The actual target enzyme of azole antifungals, the fungal sterol 14[alpha]-demethylase, is expressed in many species including humans, and it is highly conserved through evolution. Hence, it seems reasonable to assume that most of the azole antifungals used in agriculture and medicine as well as azoles used in management of breast cancer also act as inhibitors on human sterol 14[alpha]-demethylase to an unknown extent. The toxicologic profiles of individual azole fungicides provide evidence for endocrine effects. In fact, many of these fungicides have effects on prostate, testis, uterus, and ovaries as well as on fertility, development, and sexual behavior. The current database does not allow us to establish causal relationships of these effects with inhibition of sterol 14[alpha]-demethylase and/or aromatase, but the overall view strongly suggests a connection with disturbed steroidogenesis.

Zam et al; Environmental Health Perspectives-3/1/2003

Some azoles have been associated with prolongation of the QT interval on the electrocardiogram.

| AC Consort 400 Herbicide | TOXICITY | IRRITATION |
|--------------------------|---|---|
| | Not Available | Not Available |
| carfentrazone-ethyl | TOXICITY | IRRITATION |
| | dermal (rat) LD50: >4000 mg/kg ^[2] | Not Available |
| | Inhalation(Rat) LC50: 5.09 mg/L4h ^[2] | |
| liquid hydrocarbons | TOXICITY | IRRITATION |
| | Not Available | Not Available |
| N-methyl-2-pyrrolidone | TOXICITY | IRRITATION |
| | Dermal (rabbit) LD50: 8000 mg/kg ^[2] | Eye (rabbit): 100 mg - moderate *[Manufacturer] |
| | Inhalation(Rat) LC50: 3.1-8.8 mg/l4h ^[2] | |
| | TOXICITY | IRRITATION |
| | Oral (Rat) LD50: 3914 mg/kg ^[2] | |

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

CARFENTRAZONE-ETHYL

* Sigma Aldrich SDS Carfentrazone-ethyl acts as an inhibitor of protoporphyrinogen oxidase, which in mammals interferes with the heme biosynthetic pathway and results in increased porphyrin levels. Carfentrazone-ethyl has low acute oral, dermal, and inhalation toxicity. It is minimally irritating to the eyes, non-irritating to the skin, and is not a skin sensitizer. The mutagenic test battery demonstrated that carfentrazone-ethyl is not mutagenic. In carcinogenicity studies in mice and rats, there was no indication of increased incidence of neoplasms and spontaneous tumor formation at the doses tested.

For Protoporphyrinogen Oxidase (PPO) Inhibitors:

PPO inhibition at high doses resulted in a range of observations in mammalian toxicology studies. As oxidised porphyrin is a key component of mammalian haemoglobin, a common finding at comparatively high doses in toxicology studies was a slight reduction in haemoglobin levels and related blood parameters. Inhibition of porphyrin synthesis results in precursor porphyrins accumulating in the liver where they are excreted in the bile coupled with cholesterol. This process results in deposition of pigment in the liver and other tissues, as well as alterations in cholesterol levels due to increased production to compensate for that lost with the porphyrin excretion.

The developmental toxicity studies conducted on rats and rabbits indicate that the majority of the compounds did not show any reproductive, developmental, or teratogenic abnormalities, except at very high doses that elicit maternal toxicity. The developmental toxicity correlates with PPO herbicide accumulation

The PPO inhibitor herbicides are either not readily absorbed and/or are rapidly degraded by metabolism and/or excreted. The mammalian metabolites are similar to photochemical degradation products. In mammals, there are remarkable species differences in the levels of porphyrin accumulation resulting from exposure to PPO inhibitors. There is no bioaccumulation risk in animals. Metabolism of PPO inhibitors has been studied in a number of species, including rats, rabbits, goats, sheep, cattle, and chicken. In general, the metabolic degradation of these compounds by animals includes nitroreduction, deesterification, and conjugation to GSH, cysteine, and carbohydrates. Most of the metabolites are excreted in urine, with small amounts excreted in faeces and milk. In chickens, 95% of the metabolites are eliminated in excreta, with small amounts (0.09%) eliminated in the eggs

PPO inhibition in mammals may disrupt heme synthesis, which in turn causes anemia. In the submitted studies, decreased hematological parameters [decreased red blood cells (RBC), decreased hematocrit (Ht), decreased mean corpuscular haemoglobin concentration (MCHC), and mean corpuscular volume (MCV)] were observed at about the same dose level across species, with the exception of the dog, where effects were observed at a slightly higher dose. These effects occurred around the same dose level from short- through long-term exposures, without increasing in severity. Effects were also seen in the liver (increased weight, centrilobular fatty change, lymphoid infiltrate) in mice, the spleen (increased spleen weight and extramedullary hematopoiesis) in rats, and in both these organs (increased iron storage in the liver and extramedullary hematopoiesis in the spleen) in dogs. These effects also occurred around the same dose level from short- through long-term exposures, without increasing in severity. No dermal toxicity was seen at the limit dose in a 28-day dermal toxicity study in rats. Toxicology studies with PPO inhibitors have shown that certain chemicals cause embryo lethality, teratogenicity and growth retardation in rats but not in other mammals such as rabbits. In these studies it was shown that the effect of 30 mg/kg of S-52482, a phenylimide PPO inhibitor, on embryo development in rats was correlated with the accumulation of protoporphyrin IX (Proto IX) in the embryo with a concomitant loss of haeme. However, 3000 mg/kg of S-52482 caused no accumulation of Proto IX in rabbit embryos and there was no adverse effect on the embryos. The authors concluded that this difference was due to the relative sensitivity of PPO in rats versus rabbits. Thus, the effects of PPO-inhibiting herbicides on mammals is species-dependent. The mammalian toxicity of these herbicides appears to be minimal at the rates they are used.

Protoporphyrinogen oxidase (PPO, E.C.1.3.3.4) catalyzes the oxygen-dependent oxidation of protoporphyrinogen IX to protoporphyrin IX (Proto IX).

In the presence of light, accumulated protoporphyrin can generate highly reactive oxygen species and induce membrane lipid peroxidation. The peroxidation of the lipid can result in a chain reaction and cause fragmentation and destruction of the lipid. The consequence of lipid peroxidation for a cell is loss of the membrane function.

Protoporphyrin IX (Proto IX) is an important precursor to biologically essential prosthetic groups such as heme, cytochrome c, and chlorophylls. As a result, a number of organisms are able to synthesize this tetrapyrrole from basic precursors such as glycine and succinyl CoA, or glutamate. Despite the wide range of organisms that synthesize protoporphyrin IX the process is largely conserved from bacteria to mammals with a few distinct exceptions in higher plants.

The inhibition or functional loss of PPO is more than merely blocking the production of heme and chlorophyll. When the enzyme is inhibited, the substrate protoporphyrinogen-IX will accumulate in the cytoplasm and will be slowly oxidized by O₂ in the mitochondrion and chloroplast to produce protoporphyrin-IX. This spontaneous production can have dire consequences: In the presence of light, the photosensitive protoporphyrin-IX generates singlet oxygen that causes lipid peroxidation and cell death.

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Symptoms may be cutaneous or neurovisceral with similar inciting factors as acute intermittent porphyria (AIP), but the cutaneous symptoms are more difficult to treat and persist longer. Like hereditary coproporphyria, may be associated with acute episodes (as seen in acute intermittent porphyria) and with photocutaneous manifestations (as seen in porphyria cutanea tarda).

Protoporphyrin can lead to reactive singlet oxygen formation in the presence of light, and photodermatitis within variegate porphyria (VP) patients is thought to be caused by photooxidation of protoporphyrinogen and increased production of reactive oxygen species within skin fibroblasts. The symptom of VP and its highly variable penetrance of infected individuals make the study of the nature of PPO causing the disease of great interest. Besides, protoporphyrin-IX is an extremely effective photosensitizer, but it is not useful before activation. PPO inhibitors could activate the photosensitizer protoporphyrin-IX and cause its accumulation within tumor cells. Hence, an important medical application of PPO inhibitors is associated with photodynamic therapy (PDT), which has been used in the detection and treatment of cancer.

Currently PDT is performed by administering photosensitizers to patients and attempting to establish high concentrations in the tumors. These tumors are then exposed to irradiation with light with the appropriate wavelength to activate the photosensitizers and destroy the cells. Proto IX is an extremely effective photosensitizer, but it cannot be used since it does not accumulate within tumors after parenteral administration. PPO inhibitors could cause the accumulation of Proto IX within tumor cells. The levels reached after treatment with certain PPO analogs was tenfold higher than the critical levels needed for effective PDT. The use of PPO inhibitors for PDT is being further explored.

LIQUID HYDROCARBONS

For olefins:

Studies have shown that normal alpha olefins have little or no toxic effect on animals except in very severe inhalation conditions and that they may produce minimal skin and eye irritation, but are not skin sensitizers. Laboratory exposures to very high airborne concentrations of C₆-C₁₆ normal alpha olefin vapors or mists produced central nervous system effects including anesthesia. If C₂₀+ products are heated, fumes may produce nausea and irritation of the upper respiratory tract. Although not all products have been tested in genetic toxicity assays, the available data indicate normal alpha olefins are not mutagenic.

Acute toxicity: The weight of evidence indicates alpha and internal olefins with carbon numbers between C₆ and C₅₄ have a similar and low level of mammalian toxicity, and the toxicity profile is not affected by changes in the location of the double bond or the addition of branching to the structure. These materials are not eye irritants or skin sensitizers. Prolonged exposure of the skin for many hours may cause skin irritation.

Olefins (alkenes) ranging in carbon number from C6 to C24 alpha (linear) and internal (linear and branched), and C24-54 alpha (linear and branched) demonstrate low acute toxicity by the oral, inhalation and dermal routes of exposure: Rat oral LD50 >5 g/kg; rat 4-hr inhalation LC50 range = 110 mg/L (32,000 ppm) to 6.4 mg/L (693 ppm) for C6 to C16; and rat/rabbit dermal LD50 > highest doses tested (1.43-10 g/kg).

Repeated dose toxicity: Studies, using the inhalation (C6 alpha), dermal (C12-16 alpha), or oral (C6 alpha and internal linear/branched; C8 and C14 alpha; and C16/18, C18 and C20-24 internal linear/branched) routes of exposure, have shown comparable levels of low toxicity in rats. In females, alterations in body and organ weights, changes in certain clinical chemistry/haematology values, and liver effects were noted (NOELs of ≥ 100 mg/kg oral or ≥ 3.44 mg/kg [1000 ppm] inhalation). In males, alterations in organ weights, changes in certain clinical chemistry/hematology values, liver effects, and kidney damage were noted (LOELs ≥ 100 mg/kg oral only). The male rat kidney damage suggests alpha_{2u}-globulin nephropathy, a male rat specific effect that is not considered relevant to human health. The noted liver effects were seen in oral studies with C14 alpha olefins (minimal-to-mild hepatocyte cytoplasmic vacuolation with increased liver weight in males and females) and with C20-24 internal olefins (minimal centrilobular hepatocyte hypertrophy with increased liver weight in females only). No effects were present in the study with C20-24 internal olefins following a 4-week recovery period, indicating reversibility of the observed effects. These liver effects seen only with the larger molecules may be indirect effects of an intensified liver burden, rather than a direct toxic effect of the olefin. Based on evidence from neurotoxicity screens included in repeated dose studies with C6 and C14 alpha olefins and with C6, C16/18 and C20-24 internal linear/branched olefins, the category members are not neurotoxic.

Reproductive/developmental toxicity: Based on evidence from reproductive/developmental toxicity screens in rats with C6 and C14 alpha olefins and C6 and C18 linear/branched internal olefins, along with the findings of no biologically significant effects on male or female reproductive organs in repeated dose toxicity studies, olefins are not expected to cause reproductive or developmental toxicity.

Genotoxicity: Based on the weight of evidence from studies with alpha and internal olefins, category members are not genotoxic.

Carcinogenicity: No carcinogenicity tests have been conducted on C6-54 alpha or internal olefins; however, there are no structural alerts indicating a potential for carcinogenicity in humans. No significant acute toxicological data identified in literature search.

Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.

for N-methyl-2-pyrrolidone (NMP):

Acute toxicity: In rats, NMP is absorbed rapidly after inhalation, oral, and dermal administration, distributed throughout the organism, and eliminated mainly by hydroxylation to polar compounds, which are excreted via urine. About 80% of the administered dose is excreted as NMP and NMP metabolites within 24 h. A probably dose-dependent yellow coloration of the urine in rodents is observed. The major metabolite is 5-hydroxy-N-methyl-2-pyrrolidone.

Studies in humans show comparable results. Dermal penetration through human skin has been shown to be very rapid. NMP is rapidly biotransformed by hydroxylation to 5-hydroxy-N-methyl-2-pyrrolidone, which is further oxidized to N-methylsuccinimide; this intermediate is further hydroxylated to 2-hydroxy-N-methylsuccinimide. These metabolites are all colourless. The excreted amounts of NMP metabolites in the urine after inhalation or oral intake represented about 100% and 65% of the administered doses, respectively.

NMP has a low potential for skin irritation and a moderate potential for eye irritation in rabbits. Repeated daily doses of 450 mg/kg body weight administered to the skin caused painful and severe haemorrhage and eschar formation in rabbits. These adverse effects have not been seen in workers occupationally exposed to pure NMP, but they have been observed after dermal exposure to NMP used in cleaning processes. No sensitisation potential has been observed.

In acute toxicity studies in rodents, NMP showed low toxicity. Uptake of oral, dermal, or inhaled acutely toxic doses causes functional disturbances and depressions in the central nervous system. Local irritation effects were observed in the respiratory tract when NMP was inhaled and in the pyloric and gastrointestinal tracts after oral administration. In humans, there was no irritative effect in the respiratory system after an 8-h exposure to 50 mg/m³.

Repeat dose toxicity: There is no clear toxicity profile of NMP after multiple administration. In a 28-day dietary study in rats, a compound-related decrease in body weight gain was observed in males at 1234 mg/kg body weight and in females at 2268 mg/kg body weight. Testicular degeneration and atrophy in males and thymic atrophy in females were observed at these dose levels. The no-observed-adverse-effect level (NOAEL) was 429 mg/kg body weight in males and 1548 mg/kg body weight in females. In a 28-day intubation study in rats, a dose-dependent increase in relative liver and kidney weights and a decrease in lymphocyte count in both sexes were observed at 1028 mg/kg body weight. The NOAEL in this study was 514 mg/kg body weight. In another rat study, daily dietary intake for 90 days caused decreased body weights at doses of 433 and 565 mg/kg body weight in males and females, respectively. There were also neurobehavioural effects at these dose levels. The NOAELs in males and females were 169 and 217 mg/kg body weight, respectively.

The toxicity profile after exposure to airborne NMP depends strongly on the ratio of vapour to aerosol and on the area of exposure (i.e., head-only or whole-body exposure). Because of higher skin absorption for the aerosol, uptake is higher in animals exposed to aerosol than in those exposed to vapour at similar concentrations. Studies in female rats exposed head only to 1000 mg/m³ showed only minor nasal irritation, but massive mortality and severe effects on major organs were observed when the females were whole-body exposed to the same concentration of coarse droplets at high relative humidity. Several studies in rats following repeated exposure to NMP at concentrations between 100 and 1000 mg/m³ have shown systemic toxicity effects at the lower dose levels. In most of the studies, the effects were not observed after a 4-week observation period.

In rats, exposure to 3000 mg NMP/m³ (head only) for 6 h/day, 5 days/week, for 13 weeks caused a decrease in body weight

N-METHYL-2-PYRROLIDONE

gain, an increase in erythrocytes, haemoglobin, haematocrit, and mean corpuscular volume, decreased absolute testis weight, and cell loss in the germinal epithelium of the testes. The NOAEL was 500 mg/m3.

There are no data in humans after repeated-dose exposure.

Carcinogenicity: NMP did not show any clear evidence for carcinogenicity in rats exposed to concentrations up to 400 mg/m3 in a long-term inhalation study.

Genotoxicity: The mutagenic potential of NMP is weak. Only a slight increase in the number of revertants was observed when tested in a *Salmonella* assay with base-pair substitution strains. NMP has been shown to induce aneuploidy in yeast *Saccharomyces cerevisiae* cells. No investigations regarding mutagenicity in humans were available.

Reproductive toxicity: In a two-generation reproduction study in rats, whole-body exposure of both males and females to 478 mg/m3 of NMP vapour for 6 h/day, 7 days/week, for a minimum of 100 days (pre-mating, mating, gestation, and lactation periods) resulted in a 7% decrease in fetal weight in the F1 offspring. A 4-11% transient, non-dose-dependent decrease was observed in the average pup weight at all exposure levels tested (41, 206, and 478 mg/m3).

Developmental toxicity: When NMP was administered dermally, developmental toxicity was registered in rats at 750 mg/kg body weight. The observed effects were increased preimplantation losses, decreased fetal weights, and delayed ossification. The NOAEL for both developmental effects and maternal toxicity (decreased body weight gain) was 237 mg/kg body weight.

Inhalation studies in rats (whole-body exposure) demonstrated developmental toxicity as increased preimplantation loss without significant effect on implantation rate or number of live fetuses at 680 mg/m3 and behavioural developmental toxicity at 622 mg/m3. In an inhalation study (whole-body exposure), the NOAEL for maternal effects was 100 mg/m3, and the NOAEL for developmental effects was 360 mg/m3.

A tolerable inhalation concentration, 0.3 mg/m3, based on mortality and organ damage, is expected to be protective against any possible reproductive toxicity. Similarly, an oral tolerable intake of 0.6 mg/kg body weight per day, based on a 90-day study, is expected to provide adequate protection against possible reproductive effects. Because of non-existent data on the exposure of the general population and very limited information on occupational exposure, no meaningful risk characterisation can be performed.

A substance (or part of a group of chemical substances) of very high concern (SVHC) - or product containing an SVHC: It is proposed that use within the European Union be subject to authorisation under the REACH Regulation. Indeed, listing of a substance as an SVHC by the European Chemicals Agency (ECHA) is the first step in the procedure for authorisation or restriction of use of a chemical.

The criteria are given in article 57 of the REACH Regulation. A substance may be proposed as an SVHC if it meets one or more of the following criteria:

- it is carcinogenic *;
- it is mutagenic *;
- it is toxic for reproduction *;
- it is persistent, bioaccumulative and toxic (PBT substances);
- it is very persistent and very bioaccumulative (vPvB substances);
- there is "scientific evidence of probable serious effects to human health or the environment which give rise to an equivalent level of concern"; such substances are identified on a case-by-case basis.

* Collectively described as CMR substances

The "equivalent concern" criterion is significant because it is this classification which allows substances which are, for example, neurotoxic, endocrine-disrupting or otherwise present an unanticipated environmental health risk to be regulated under REACH. Simply because a substance meets one or more of the criteria does not necessarily mean that it will be proposed as an SVHC.

Many such substances are already subject to restrictions on their use within the European Union, such as those in Annex XVII of the REACH Regulation. SVHCs are substances for which the current restrictions on use (where these exist) might be insufficient.

There are three priority groups for assessment:

- PBT substances and vPvB substances;
- substances which are widely dispersed during use;
- substances which are used in large quantities.

| | | | |
|-----------------------------------|---|--------------------------|---|
| 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

| AC Consort 400 Herbicide | Endpoint | Test Duration (hr) | Species | Value | Source |
|--------------------------|---------------|--------------------|---------------|---------------|---------------|
| | Not Available | Not Available | Not Available | Not Available | Not Available |

Continued...

| carfentrazone-ethyl | Endpoint | Test Duration (hr) | Species | Value | Source |
|------------------------|---------------|--------------------|-------------------------------|---------------|---------------|
| | EC50 | 48h | Crustacea | >9.8mg/l | Not Available |
| | EC50 | 72h | Algae or other aquatic plants | 0.006mg/l | Not Available |
| | EC50(ECx) | 72h | Algae or other aquatic plants | 0.006mg/l | Not Available |
| | LC50 | 96h | Fish | 1.6mg/l | Not Available |
| liquid hydrocarbons | Endpoint | Test Duration (hr) | Species | Value | Source |
| | Not Available | Not Available | Not Available | Not Available | Not Available |
| N-methyl-2-pyrrolidone | Endpoint | Test Duration (hr) | Species | Value | Source |
| | EC50 | 48h | Crustacea | ca.4897mg/l | 1 |
| | EC50 | 72h | Algae or other aquatic plants | >500mg/l | 1 |
| | NOEC(ECx) | 504h | Crustacea | 12.5mg/l | 2 |
| | LC50 | 96h | Fish | 464mg/l | 1 |

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

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

Persistence and degradability

| Ingredient | Persistence: Water/Soil | Persistence: Air |
|------------------------|-------------------------|------------------|
| carfentrazone-ethyl | HIGH | HIGH |
| N-methyl-2-pyrrolidone | LOW | LOW |

Bioaccumulative potential

| Ingredient | Bioaccumulation |
|------------------------|--------------------------|
| carfentrazone-ethyl | MEDIUM (LogKOW = 4.2583) |
| N-methyl-2-pyrrolidone | LOW (BCF = 0.16) |

Mobility in soil

| Ingredient | Mobility |
|------------------------|-------------------|
| carfentrazone-ethyl | LOW (KOC = 6858) |
| N-methyl-2-pyrrolidone | LOW (KOC = 20.94) |



SECTION 13 Disposal considerations

Waste treatment methods

| | |
|------------------------------|--|
| Product / Packaging disposal | <ul style="list-style-type: none">Recycle wherever possible or consult manufacturer for recycling options.Consult State Land Waste Authority for disposal.Bury or incinerate residue at an approved site.Recycle containers if possible, or dispose of in an authorised landfill. |
|------------------------------|--|

SECTION 14 Transport information

Labels Required

| | |
|------------------|---|
| |  |
| Marine Pollutant |  |
| HAZCHEM | •3Z |

Land transport (ADG)

| | | |
|------------------------------------|--|----------------------|
| 14.1. UN number or ID number | 3082 | |
| 14.2. UN proper shipping name | ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains carfentrazone-ethyl) | |
| 14.3. Transport hazard class(es) | Class | 9 |
| | Subsidiary Hazard | Not Applicable |
| 14.4. Packing group | III | |
| 14.5. Environmental hazard | Environmentally hazardous | |
| 14.6. Special precautions for user | Special provisions | 274 331 335 375 AU01 |
| | Limited quantity | 5 L |

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082

are not subject to this Code when transported by road or rail in;

(a) packagings;

(b) IBCs; or

(c) any other receptacle not exceeding 500 kg(L).

- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

Air transport (ICAO-IATA / DGR)

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

Sea transport (IMDG-Code / GGVSee)

| | | |
|-------------------------------|--|--|
| 14.1. UN number | 3082 | |
| 14.2. UN proper shipping name | ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains carfentrazone-ethyl) | |

| | | |
|------------------------------------|------------------------|----------------|
| 14.3. Transport hazard class(es) | IMDG Class | 9 |
| | IMDG Subsidiary Hazard | Not Applicable |
| 14.4. Packing group | III | |
| 14.5. Environmental hazard | Marine Pollutant | |
| 14.6. Special precautions for user | EMS Number | F-A , S-F |
| | Special provisions | 274 335 969 |
| | Limited Quantities | 5 L |

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

Not Applicable

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

| Product name | Group |
|------------------------|---------------|
| carfentrazone-ethyl | Not Available |
| liquid hydrocarbons | Not Available |
| N-methyl-2-pyrrolidone | Not Available |

14.7.3. Transport in bulk in accordance with the IGC Code

| Product name | Ship Type |
|------------------------|---------------|
| carfentrazone-ethyl | Not Available |
| liquid hydrocarbons | Not Available |
| N-methyl-2-pyrrolidone | Not Available |

SECTION 15 Regulatory information

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

carfentrazone-ethyl is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

liquid hydrocarbons is found on the following regulatory lists

Not Applicable

N-methyl-2-pyrrolidone is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

Additional Regulatory Information

Not Applicable

National Inventory Status

| National Inventory | Status |
|---|--|
| Australia - AIIC / Australia Non-Industrial Use | No (carfentrazone-ethyl) |
| Canada - DSL | No (carfentrazone-ethyl) |
| Canada - NDSL | No (carfentrazone-ethyl; N-methyl-2-pyrrolidone) |
| China - IECSC | Yes |
| Europe - EINEC / ELINCS / NLP | No (carfentrazone-ethyl) |
| Japan - ENCS | No (carfentrazone-ethyl) |
| Korea - KECI | Yes |

| National Inventory | Status |
|---------------------|--|
| New Zealand - NZIoC | Yes |
| Philippines - PICCS | No (carfentrazone-ethyl) |
| USA - TSCA | No (carfentrazone-ethyl) |
| Taiwan - TCSI | Yes |
| Mexico - INSQ | No (carfentrazone-ethyl) |
| Vietnam - NCI | Yes |
| Russia - FBEPH | No (carfentrazone-ethyl) |
| Legend: | <p>Yes = All CAS declared ingredients are on the inventory</p> <p>No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.</p> |

SECTION 16 Other information

| | |
|---------------|------------|
| Revision Date | 27/10/2023 |
| Initial Date | 08/08/2018 |

SDS Version Summary

| Version | Date of Update | Sections Updated |
|---------|----------------|---|
| 7.1 | 10/03/2023 | Classification change due to full database hazard calculation/update. |
| 8.1 | 27/10/2023 | UN Number update |

Other information

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

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

Definitions and abbreviations

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

- AIIC: Australian Inventory of Industrial Chemicals
- DSL: Domestic Substances List
- NDSL: Non-Domestic Substances List
- IECSC: Inventory of Existing Chemical Substance in China
- EINECS: European INventory of Existing Commercial chemical Substances
- ELINCS: European List of Notified Chemical Substances
- NLP: No-Longer Polymers
- ENCS: Existing and New Chemical Substances Inventory
- KECI: Korea Existing Chemicals Inventory
- NZIoC: New Zealand Inventory of Chemicals
- PICCS: Philippine Inventory of Chemicals and Chemical Substances
- TSCA: Toxic Substances Control Act

AC Consort 400 Herbicide

- TCSI: Taiwan Chemical Substance Inventory
- INSQ: Inventario Nacional de Sustancias Químicas
- NCI: National Chemical Inventory
- FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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