

# AC Consort 400 Herbicide

AXICHEM Pty Ltd

Chemwatch Hazard Alert Code: 3

Chemwatch: 5315-90

Issue Date: 09/08/2018

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Safety Data Sheet according to WHS and ADG requirements

L.GHS.AUS.EN

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

### Product Identifier

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

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

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 supplier of the safety data sheet

Registered company name	AXICHEM Pty Ltd
Address	18 Conquest Way Wangara WA 6065 Australia
Telephone	+61 8 9302 4666
Fax	Not Available
Website	www.axichem.com.au
Email	msds@axichem.com.au

### Emergency telephone number

Association / Organisation	Chemwatch
Emergency telephone numbers	1800 039 008 (All Hours)
Other emergency telephone numbers	Not Available

## CHEMWATCH EMERGENCY RESPONSE

Primary Number	Alternative Number 1	Alternative Number 2
1800 039 008	1800 039 008	+612 9186 1132

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 Liquid Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Reproductive Toxicity Category 1B, Aspiration Hazard Category 1, Acute Aquatic Hazard Category 1, Chronic Aquatic Hazard Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HSIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

### Label elements

Continued...

AC Consort 400 Herbicide

<b>Hazard pictogram(s)</b>	
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<b>SIGNAL WORD</b>	<b>DANGER</b>
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**Hazard statement(s)**

<b>H227</b>	Combustible liquid.
<b>H315</b>	Causes skin irritation.
<b>H319</b>	Causes serious eye irritation.
<b>H360</b>	May damage fertility or the unborn child.
<b>H304</b>	May be fatal if swallowed and enters airways.
<b>H410</b>	Very toxic to aquatic life with long lasting effects.

**Precautionary statement(s) Prevention**

<b>P201</b>	Obtain special instructions before use.
<b>P210</b>	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
<b>P281</b>	Use personal protective equipment as required.
<b>P273</b>	Avoid release to the environment.
<b>P280</b>	Wear protective gloves/protective clothing/eye protection/face protection.

**Precautionary statement(s) Response**

<b>P301+P310</b>	IF SWALLOWED: Immediately call a POISON CENTER or doctor/physician.
<b>P308+P313</b>	IF exposed or concerned: Get medical advice/attention.
<b>P331</b>	Do NOT induce vomiting.
<b>P362</b>	Take off contaminated clothing and wash before reuse.
<b>P370+P378</b>	In case of fire: Use water spray/fog for extinction.
<b>P305+P351+P338</b>	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
<b>P337+P313</b>	If eye irritation persists: Get medical advice/attention.
<b>P391</b>	Collect spillage.
<b>P302+P352</b>	IF ON SKIN: Wash with plenty of soap and water.
<b>P332+P313</b>	If skin irritation occurs: Get medical advice/attention.

**Precautionary statement(s) Storage**

<b>P403+P235</b>	Store in a well-ventilated place. Keep cool.
<b>P405</b>	Store locked up.

**Precautionary statement(s) Disposal**

<b>P501</b>	Dispose of contents/container in accordance with local regulations.
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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> 400 g/L)
Various	30-50	<u>liquid hydrocarbons</u>
872-50-4	10-20	<u>N-methyl-2-pyrrolidone</u>

## SECTION 4 FIRST AID MEASURES

### Description of first aid measures

<b>Eye Contact</b>	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> <li>▶ Wash out immediately with fresh running water.</li> <li>▶ 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.</li> <li>▶ Seek medical attention without delay; if pain persists or recurs seek medical attention.</li> <li>▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> </ul>
<b>Skin Contact</b>	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> <li>▶ Immediately remove all contaminated clothing, including footwear.</li> <li>▶ Flush skin and hair with running water (and soap if available).</li> <li>▶ Seek medical attention in event of irritation.</li> </ul>
<b>Inhalation</b>	<ul style="list-style-type: none"> <li>▶ If fumes or combustion products are inhaled remove from contaminated area.</li> <li>▶ Lay patient down. Keep warm and rested.</li> <li>▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.</li> <li>▶ 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.</li> <li>▶ Transport to hospital, or doctor.</li> </ul>
<b>Ingestion</b>	<ul style="list-style-type: none"> <li>▶ <b>If swallowed do NOT induce vomiting.</b></li> <li>▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.</li> <li>▶ Observe the patient carefully.</li> <li>▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.</li> <li>▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.</li> <li>▶ Seek medical advice.</li> </ul>

### 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

<b>Fire Incompatibility</b>	▶ 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

<b>Fire Fighting</b>	<ul style="list-style-type: none"> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ Use water delivered as a fine spray to control fire and cool adjacent area.</li> <li>▶ Avoid spraying water onto liquid pools.</li> <li>▶ <b>Do not</b> approach containers suspected to be hot.</li> <li>▶ Cool fire exposed containers with water spray from a protected location.</li> <li>▶ If safe to do so, remove containers from path of fire.</li> </ul>
<b>Fire/Explosion Hazard</b>	<ul style="list-style-type: none"> <li>▶ Combustible.</li> <li>▶ Slight fire hazard when exposed to heat or flame.</li> <li>▶ Heating may cause expansion or decomposition leading to violent rupture of containers.</li> <li>▶ On combustion, may emit toxic fumes of carbon monoxide (CO).</li> <li>▶ May emit acrid smoke.</li> <li>▶ Mists containing combustible materials may be explosive.</li> </ul> <p>Combustion products include:</p> <ul style="list-style-type: none"> <li>, carbon dioxide (CO<sub>2</sub>)</li> <li>, nitrogen oxides (NO<sub>x</sub>)</li> <li>,</li> </ul>

	hydrogen chloride , fluorides , other pyrolysis products typical of burning organic material.
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

Minor Spills	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> <li>▶ Remove all ignition sources.</li> <li>▶ Clean up all spills immediately.</li> <li>▶ Avoid breathing vapours and contact with skin and eyes.</li> <li>▶ Control personal contact with the substance, by using protective equipment.</li> <li>▶ Contain and absorb spill with sand, earth, inert material or vermiculite.</li> <li>▶ Wipe up.</li> <li>▶ Place in a suitable, labelled container for waste disposal.</li> </ul>
Major Spills	<p>Environmental hazard - contain spillage. Moderate hazard.</p> <ul style="list-style-type: none"> <li>▶ Clear area of personnel and move upwind.</li> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ No smoking, naked lights or ignition sources.</li> <li>▶ Increase ventilation.</li> <li>▶ Stop leak if safe to do so.</li> <li>▶ Contain spill with sand, earth or vermiculite.</li> <li>▶ Collect recoverable product into labelled containers for recycling.</li> <li>▶ Absorb remaining product with sand, earth or vermiculite.</li> <li>▶ Collect solid residues and seal in labelled drums for disposal.</li> <li>▶ Wash area and prevent runoff into drains.</li> <li>▶ If contamination of drains or waterways occurs, advise emergency services.</li> </ul>

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

## SECTION 7 HANDLING AND STORAGE

### Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> <li>▶ <b>DO NOT allow clothing wet with material to stay in contact with skin</b></li> <li>▶ Avoid all personal contact, including inhalation.</li> <li>▶ Wear protective clothing when risk of exposure occurs.</li> <li>▶ Use in a well-ventilated area.</li> <li>▶ Prevent concentration in hollows and sumps.</li> <li>▶ <b>DO NOT enter confined spaces until atmosphere has been checked.</b></li> <li>▶ Avoid smoking, naked lights or ignition sources.</li> <li>▶ Avoid contact with incompatible materials.</li> <li>▶ When handling, <b>DO NOT eat, drink or smoke.</b></li> <li>▶ Keep containers securely sealed when not in use.</li> <li>▶ Avoid physical damage to containers.</li> <li>▶ Always wash hands with soap and water after handling.</li> <li>▶ Work clothes should be laundered separately.</li> <li>▶ Use good occupational work practice.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.</li> </ul>
Other information	<ul style="list-style-type: none"> <li>▶ Store in original containers.</li> <li>▶ Keep containers securely sealed.</li> <li>▶ No smoking, naked lights or ignition sources.</li> <li>▶ Store in a cool, dry, well-ventilated area.</li> <li>▶ Store away from incompatible materials and foodstuff containers.</li> </ul>

- ▶ 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

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

## 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/m <sup>3</sup>	309 mg/m <sup>3</sup> / 75 ppm	Not Available	Not Available

#### EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
N-methyl-2-pyrrolidone	Methyl 2-pyrrolidinone, 1-; (N-Methylpyrrolidone)	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

#### MATERIAL DATA

### Exposure controls

<b>Appropriate engineering controls</b>	<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.</p> <p>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 the contaminant.</p>														
	<table border="1"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td> <td>0.25-0.5 m/s (50-100 f/min)</td> </tr> <tr> <td>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)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> </tbody> </table>	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.)	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
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	<p>2: Contaminants of low toxicity or of nuisance value only.</p> <p>3: Intermittent, low production.</p> <p>4: Large hood or large air mass in motion</p>	<p>2: Contaminants of high toxicity</p> <p>3: High production, heavy use</p> <p>4: Small hood-local control only</p>
<p>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.</p>		
<p><b>Personal protection</b></p>		
<p><b>Eye and face protection</b></p>	<ul style="list-style-type: none"> <li>▶ Safety glasses with side shields.</li> <li>▶ Chemical goggles.</li> <li>▶ 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]</li> </ul>	
<p><b>Skin protection</b></p>	<p>See Hand protection below</p>	
<p><b>Hands/feet protection</b></p>	<ul style="list-style-type: none"> <li>▶ Wear chemical protective gloves, e.g. PVC.</li> <li>▶ Wear safety footwear or safety gumboots, e.g. Rubber</li> </ul> <p><b>NOTE:</b></p> <ul style="list-style-type: none"> <li>▶ 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.</li> <li>▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.</li> </ul> <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"> <li>- frequency and duration of contact,</li> <li>- chemical resistance of glove material,</li> <li>- glove thickness and</li> <li>- dexterity</li> </ul> <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"> <li>- 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.</li> <li>- 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.</li> <li>- Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.</li> <li>- Contaminated gloves should be replaced.</li> </ul> <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> <li>- Excellent when breakthrough time &gt; 480 min</li> <li>- Good when breakthrough time &gt; 20 min</li> <li>- Fair when breakthrough time &lt; 20 min</li> <li>- Poor when glove material degrades</li> </ul> <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"> <li>- Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is</li> </ul>	

	needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. <ul style="list-style-type: none"> <li>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</li> </ul> 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.
<b>Body protection</b>	See Other protection below
<b>Other protection</b>	<ul style="list-style-type: none"> <li>Overalls.</li> <li>P.V.C. apron.</li> <li>Barrier cream.</li> <li>Skin cleansing cream.</li> <li>Eye wash unit.</li> </ul>

## 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(SO<sub>2</sub>), G = Agricultural chemicals, K = Ammonia(NH<sub>3</sub>), 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

<b>Appearance</b>	Yellow to orange liquid; emulsifies with water.		
<b>Physical state</b>	Liquid	<b>Relative density (Water = 1)</b>	Not Available
<b>Odour</b>	Not Available	<b>Partition coefficient n-octanol / water</b>	Not Available
<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	Not Available
<b>pH (as supplied)</b>	Not Applicable	<b>Decomposition temperature</b>	Not Available
<b>Melting point / freezing point (°C)</b>	Not Available	<b>Viscosity (cSt)</b>	Not Available
<b>Initial boiling point and boiling range (°C)</b>	Not Available	<b>Molecular weight (g/mol)</b>	Not Applicable
<b>Flash point (°C)</b>	>61	<b>Taste</b>	Not Available

<b>Evaporation rate</b>	Not Available	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	Combustible.	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	Not Available	<b>Surface Tension (dyn/cm or mN/m)</b>	Not Available
<b>Lower Explosive Limit (%)</b>	Not Available	<b>Volatile Component (%vol)</b>	Not Available
<b>Vapour pressure (kPa)</b>	Not Available	<b>Gas group</b>	Not Available
<b>Solubility in water (g/L)</b>	Miscible	<b>pH as a solution (1%)</b>	Not Available
<b>Vapour density (Air = 1)</b>	Not Available	<b>VOC g/L</b>	Not Available

## SECTION 10 STABILITY AND REACTIVITY

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

## SECTION 11 TOXICOLOGICAL INFORMATION

### Information on toxicological effects

<b>Inhaled</b>	<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</p> <p>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/m<sup>3</sup> 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>
<b>Ingestion</b>	<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>
<b>Skin Contact</b>	<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. 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>
<b>Eye</b>	<p>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.</p> <p>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.</p>
<b>Chronic</b>	<p>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:</p> <ul style="list-style-type: none"> <li>- 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.</li> </ul> <p>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.</p>

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.

**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 an 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.

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 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 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 (Byskov et al. 1995).

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.

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

<b>AC Consort 400 Herbicide</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
<b>carfentrazone-ethyl</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >4000 mg/kg <sup>[2]</sup>	Not Available

	Inhalation (rat) LC50: 5.09 mg/l/4h* <sup>[2]</sup>	
	Oral (rat) LD50: >5000 mg/kg <sup>[2]</sup>	
liquid hydrocarbons	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
N-methyl-2-pyrrolidone	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >5000 mg/kg <sup>[1]</sup>	Eye (rabbit): 100 mg - moderate
	Inhalation (rat) LC50: 8290.5297 mg/l/4H <sup>[2]</sup>	
	Oral (rat) LD50: 3914 mg/kg <sup>[2]</sup>	
<b>Legend:</b>	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	

<b>CARFENTRAZONE-ETHYL</b>	<p>* 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 sensitiser. 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. .</p>
<b>LIQUID HYDROCARBONS</b>	<p>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.</p> <p><b>Acute toxicity:</b> 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.</p> <p>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 &gt;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 &gt; highest doses tested (1.43-10 g/kg).</p> <p><b>Repeated dose toxicity:</b> 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 &gt;= 100 mg/kg oral or &gt;= 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 &gt; =100 mg/kg oral only). The male rat kidney damage suggests alpha2u,- 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.</p> <p><b>Reproductive/ developmental toxicity:</b> 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.</p> <p><b>Genotoxicity:</b> Based on the weight of evidence from studies with alpha and internal olefins, category members are not genotoxic.</p> <p><b>Carcinogenicity:</b> 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.</p> <p>No significant acute toxicological data identified in literature search.</p>
<b>N-METHYL-2-PYRROLIDONE</b>	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating</p>

substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

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/m<sup>3</sup>.

**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/m<sup>3</sup> 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/m<sup>3</sup> 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/m<sup>3</sup> (head only) for 6 h/day, 5 days/week, for 13 weeks caused a decrease in body weight 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/m<sup>3</sup>.

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/m<sup>3</sup> 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/m<sup>3</sup> 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/m<sup>3</sup>).

**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/m<sup>3</sup> and behavioural developmental toxicity at 622 mg/m<sup>3</sup>. In an inhalation study (whole-body exposure), the NOAEL for maternal effects was 100 mg/m<sup>3</sup>, and the NOAEL for developmental effects was 360 mg/m<sup>3</sup>.

A tolerable inhalation concentration, 0.3 mg/m<sup>3</sup>, 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

Acute Toxicity	☐	Carcinogenicity	☐
Skin Irritation/Corrosion	✓	Reproductivity	✓
Serious Eye Damage/Irritation	✓	STOT - Single Exposure	☐

AC Consort 400 Herbicide

Respiratory or Skin sensitisation	⊘	STOT - Repeated Exposure	⊘
Mutagenicity	⊘	Aspiration Hazard	✓

Legend: ✗ – Data available but does not fill the criteria for classification  
✓ – Data available to make classification  
⊘ – Data Not 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
carfentrazone-ethyl	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1.14mg/L	4
	EC50	48	Crustacea	>9.8mg/L	4
	EC50	72	Algae or other aquatic plants	0.0127mg/L	4
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
	LC50	96	Fish	464mg/L	1
	EC50	48	Crustacea	ca.4897mg/L	1
	EC50	72	Algae or other aquatic plants	>500mg/L	1
	EC90	72	Algae or other aquatic plants	>500mg/L	1
	NOEC	504	Crustacea	12.5mg/L	2

**Legend:** *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*

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

<b>Product / Packaging disposal</b>	<ul style="list-style-type: none"> <li>▶ Recycle wherever possible or consult manufacturer for recycling options.</li> <li>▶ Consult State Land Waste Authority for disposal.</li> <li>▶ Bury or incinerate residue at an approved site.</li> <li>▶ Recycle containers if possible, or dispose of in an authorised landfill.</li> </ul>
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## SECTION 14 TRANSPORT INFORMATION

### Labels Required

	
<b>Marine Pollutant</b>	
<b>HAZCHEM</b>	•3Z

### Land transport (ADG)

<b>UN number</b>	3082	
<b>UN proper shipping name</b>	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains carfentrazone-ethyl)	
<b>Transport hazard class(es)</b>	Class	9
	Subrisk	Not Applicable
<b>Packing group</b>	III	
<b>Environmental hazard</b>	Environmentally hazardous	
<b>Special precautions for user</b>	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)

<b>UN number</b>	3082	
<b>UN proper shipping name</b>	Environmentally hazardous substance, liquid, n.o.s. * (contains carfentrazone-ethyl)	
<b>Transport hazard class(es)</b>	ICAO/IATA Class	9
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	9L
<b>Packing group</b>	III	
<b>Environmental hazard</b>	Environmentally hazardous	
<b>Special precautions for user</b>	Special provisions	A97 A158 A197
	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)

AC Consort 400 Herbicide

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

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

Not Applicable

**SECTION 15 REGULATORY INFORMATION**

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

**CARFENTRAZONE-ETHYL(128639-02-1) 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) - Appendix B (Part 3)
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**LIQUID HYDROCARBONS(VARIOUS) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Not Applicable

**N-METHYL-2-PYRROLIDONE(872-50-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Inventory of Chemical Substances (AICS)	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

**National Inventory Status**

National Inventory	Status
Australia - AICS	N (carfentrazone-ethyl)
Canada - DSL	N (carfentrazone-ethyl)
Canada - NDSL	N (carfentrazone-ethyl; N-methyl-2-pyrrolidone)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	N (carfentrazone-ethyl)
Japan - ENCS	N (carfentrazone-ethyl)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	N (carfentrazone-ethyl)
USA - TSCA	N (carfentrazone-ethyl)
<b>Legend:</b>	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**

<b>Revision Date</b>	09/08/2018
<b>Initial Date</b>	08/08/2018

**Other information**

### Ingredients with multiple cas numbers

Name	CAS No
N-methyl-2-pyrrolidone	872-50-4, 26138-58-9

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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