

AC Oxxel

Axichem Pty Ltd

Chemwatch: 5152-94

Version No: 9.1

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

Chemwatch Hazard Alert Code: 3

Issue Date: 10/03/2023

Print Date: 14/02/2024

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

Product Identifier

Product name	AC Oxxel
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains oxyfluorfen)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Herbicide for selective weed control of certain broadleaf and grass weeds.
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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

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

Label elements

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

H304	May be fatal if swallowed and enters airways.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H336	May cause drowsiness or dizziness.
H351	Suspected of causing cancer.
H360	May damage fertility or the unborn child.
H373	May cause damage to organs through prolonged or repeated exposure.
H410	Very toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

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.
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.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.
P302+P352	IF ON SKIN: Wash with plenty of water.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

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

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
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CAS No	%[weight]	Name
Various	50-60	<u>liquid hydrocarbons</u>
42874-03-3	20-25	<u>oxyfluorfen</u>
Not Available		(240 g/L)
872-50-4	<10	<u>N-methyl-2-pyrrolidone</u>
Not Available	<20	Ingredients determined not to be hazardous

Legend: 1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <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	<p>If skin contact occurs:</p> <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, aerosols or combustion products are inhaled remove from contaminated area. Other measures are usually unnecessary.
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

For acute or short term repeated exposures to petroleum distillates or related hydrocarbons:

- Primary threat to life, from pure petroleum distillate ingestion and/or inhalation, is respiratory failure.
- Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO₂ 50 mm Hg) should be intubated.
- Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.
- Lavage is indicated in patients who require decontamination; ensure use of cuffed endotracheal tube in adult patients. [Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

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

Fire Fighting	Alert Fire Brigade and tell them location and nature of hazard.
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	<ul style="list-style-type: none"> ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ 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. ▶ Equipment should be thoroughly decontaminated after use.
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 acrid smoke. ▶ Mists containing combustible materials may be explosive. <p>Combustion products include: carbon dioxide (CO₂) hydrogen chloride phosgene hydrogen fluoride nitrogen oxides (NO_x) other pyrolysis products typical of burning organic material.</p>
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SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> ▶ 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

Safe handling	<ul style="list-style-type: none"> ▶ 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.
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	<ul style="list-style-type: none">▶ When handling, DO NOT eat, drink or smoke.▶ Keep containers securely sealed when not in use.▶ Avoid physical damage to containers.▶ Always wash hands with soap and water after handling.▶ Work clothes should be laundered separately.▶ Use good occupational work practice.▶ Observe manufacturer's storage and handling recommendations contained within this SDS.▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	<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

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	Avoid contamination with strong oxidising agents as ignition may result

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
liquid hydrocarbons	Not Available	Not Available
oxyfluorfen	Not Available	Not Available
N-methyl-2-pyrrolidone	Not Available	Not Available


Occupational Exposure Banding

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

MATERIAL DATA

Exposure controls

Appropriate engineering controls	<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> <ul style="list-style-type: none">Process controls which involve changing the way a job activity or process is done to reduce the risk.Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. <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</p>
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	<p>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> <tr> <th>Type of Contaminant:</th><th>Air Speed:</th></tr> <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> </table> <p>Within each range the appropriate value depends on:</p> <table> <tr> <th>Lower end of the range</th><th>Upper end of the range</th></tr> <tr> <td>1: Room air currents minimal or favourable to capture</td><td>1: Disturbing room air currents</td></tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td><td>2: Contaminants of high toxicity</td></tr> <tr> <td>3: Intermittent, low production.</td><td>3: High production, heavy use</td></tr> <tr> <td>4: Large hood or large air mass in motion</td><td>4: Small hood-local control only</td></tr> </table> <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>	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	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
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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>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> <ul style="list-style-type: none"> · Excellent when breakthrough time > 480 min 																				

	<ul style="list-style-type: none"> · 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 Oxxel

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)

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Amber liquid with an aromatic odour; emulsifies in water.		
Physical state	Liquid	Relative density (Water = 1)	1.08
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available

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Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	100	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	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

Inhaled	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
Ingestion	<p>Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.</p> <p>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).</p> <p>Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosal ulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea and vomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injury may produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Light aromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration into the lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage.</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.</p> <p>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>
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 a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	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.

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.

AC Oxxel	TOXICITY	IRRITATION
	Not Available	Not Available
liquid hydrocarbons	TOXICITY	IRRITATION
	Not Available	Not Available
oxyfluorfen	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >10000 mg/kg ^[2]	Eye (rabbit): mild to moderate *
	Oral (Dog) LD50; >5000 mg/kg ^[2]	Skin (rabbit): mild *
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]	
	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	

LIQUID HYDROCARBONS	TOXICITY	IRRITATION
	Not Available	Not Available
liquid hydrocarbons	TOXICITY	IRRITATION
	Not Available	Not Available
oxyfluorfen	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >10000 mg/kg ^[2]	Eye (rabbit): mild to moderate *
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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 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.

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.

OXYFLUORFEN

ADI 0.003 mg/kg * Toxicity class WHO Table 5, EPA IV * NOEL In chronic dietary trials, NOEL for rats 40, dogs 100, mice 2 mg/kg diet *

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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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. Variegate porphyria (VP) is also an autosomal dominant disorder caused by the deficiency of protoporphyrinogen oxidase. 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 photodermatitis 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.

551phenth

For chlorophenoxy pesticides:

551chlph

WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.

Side-reactions during manufacture of the parent compound may result in the production of trace amounts of polyhalogenated aromatic hydrocarbon(s). Halogenated phenols, and especially their alkali salts, can condense above 300 deg. C. to form polyphenoxyphenols or, in a very specific reaction, to form dibenzo-p-dioxins.

Polyhalogenated aromatic hydrocarbons (PHAHs) comprise two major groups. The first group represented by the halogenated derivatives of dibenzodioxins (the chlorinated form is PCDD), dibenzofurans (PCDF) and biphenyls (PCB) exert their toxic effect (as hepatocarcinogens, reproductive toxicants, immunotoxicants and procarcinogens) by interaction with a cytosolic protein known as the Ah receptor. In guinea pigs the Ah receptor is active in a mechanism which "pumps" PHAH into the cell whilst in humans the reverse appears to be true. This, in part, may account for species differences often cited in the literature. This receptor exhibits an affinity for the planar members of this group and carries these to the cellular nucleus where they bind, reversibly, to specific genomes on DNA. This results in the regulation of the production of certain proteins which elicit the toxic response. The potency of the effect is dependent on the strength of the original interaction with the Ah receptor and is influenced by the degree of substitution by the halogen and the position of such substitutions on the parent compound.

The most potent molecule is 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) while the coplanar PCBs (including mono-ortho coplanars) possess approximately 1% of this potency. Nevertheless, all are said to exhibit "dioxin-like" behaviour and in environmental and health assessments it has been the practice to assign each a TCDD-equivalence value.

The most subtle and important biological effects of the PHAHs are the effects on endocrine hormones and vitamin homeostasis. TCDD mimics the effect of thyroxine (a key metamorphosis signal during maturation) and may disrupt patterns of embryonic development at critical stages. Individuals from exposed wildlife populations have been observed to have altered sexual development, sexual dysfunction as adults and immune system suppression. Immunotoxic effects of the PHAHs (including the brominated congener, PBB) have been the subject of several studies. No clear pattern emerges in human studies however with T-cell numbers and function (a blood marker for immunological response) increasing in some and decreasing in others.

Developmental toxicity (e.g. cleft palate, hydronephrosis) occurs in relatively few species; functional alterations following TCDD exposure leads to deficits in cognitive functions in monkeys and to adverse effects in the male reproductive system of rats.

Three incidences have occurred which have introduced abnormally high levels of dioxin or dioxin-like congeners to humans. The explosion at a trichlorophenol-manufacturing plant in Seveso, Italy distributed TCDD across a large area of the country-side, whilst rice-oil contaminated with heat-transfer PCBs (and dioxin-like contaminants) has been consumed by two groups, on separate occasions (one in Yusho, Japan and another in Yu-cheng, Taiwan). The only symptom which can unequivocally be related to all these exposures is the development of chloracne, a disfiguring skin condition, following each incident. Contaminated oil poisonings also produced eye-discharge, swelling of eyelids and visual disturbances. The Babies born up to 3 years after maternal exposure (so-called "Yusho-babies") were characteristically brown skinned, coloured gums and nails and (frequently) produced eye-discharges. Delays in intellectual development have been noted. It has been estimated that Yu-cheng patients consumed an average level of 0.06 mg/kg body weight/day total PCB and 0.0002 mg/kg/day of PCDF before the onset of symptoms after 3 months. When the oil was withdrawn after 6 months they had consumed 1 gm total PCB containing 3.8 mg PCDF. Taiwanese patients consumed 10 times as much contaminated oil as the Japanese patients (because of later withdrawal); however since PCB/PCDF concentration in the Japanese oil was 10 times that consumed in Taiwan, patients from both countries consumed about the same amount of PCBs/PCDFs. Preliminary data from the Yusho cohort suggests a six-fold excess of liver cancer mortality in males and a three-fold excess in women.

Recent findings from Seveso indicate that the biological effects of low level exposure (BELLEs), experienced by a cohort located at a great distance from the plant, may be hormetic, i.e. may be protective AGAINST the development of cancer. The PHAHs do not appear to be genotoxic - they do not alter the integrity of DNA. This contrasts with the effects of the many polycyclic aromatic

hydrocarbons (PAHs) (or more properly, their reactive metabolites). TCDD induces carcinogenic effects in the laboratory in all species, strains and sexes tested. These effects are dose-related and occur in many organs. Exposures as low as 0.001 ug/kg body weight/day produce carcinoma. Several studies implicate PCBs in the development of liver cancer in workers as well as multi-site cancers in animals. The second major group of PHAH consists of the non-planar PCB congeners which possess two or more ortho-substituted halogens. These have been shown to produce neurotoxic effects which are thought to reduce the concentration of the brain neurotransmitter, dopamine, by inhibiting certain enzyme-mediated processes. The specific effect elicited by both classes of PHAH seems to depend on the as much on the developmental status of the organism at the time of the exposure as on the level of exposure over a lifetime.

NOTE: Some jurisdictions require that health surveillance be conducted on workers occupationally exposed to polycyclic aromatic hydrocarbons. Such surveillance should emphasise

- demography, occupational and medical history
- health advice, including recognition of photosensitivity and skin changes
- physical examination if indicated
- records of personal exposure including photosensitivity

The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

For oxyfluorfen:

- **Acute toxicity:** Oxyfluorfen is practically nontoxic by ingestion, with reported oral LD50 values of 5000 mg/kg in both rats and dogs, and 2700 to 5000 mg/kg in mice. The dermal LD50 is greater than 5000 mg/kg in both rats and rabbits, also indicating slight toxicity by this route. It causes no skin irritation in rabbits, no skin sensitization in guinea pigs, and moderate eye irritation in rabbits. However, Goal and other formulations may show severe skin and eye irritant properties, and may be skin sensitizers. The 4-hour inhalation LC50 for the technical product is not available, but that for Goal 1.6E is greater than 22.64 mg/L, indicating practically no toxicity via this route.
- **Chronic toxicity:** Effects on the liver have been observed in long-term feeding studies with rats, mice, and dogs.
- **Reproductive effects:** In a developmental study with rats given doses of 10, 100, or 1000 mg/kg/day by gavage, decreased implantation, increased resorption, and lower foetal survival was seen at the 1000 mg/kg level. Toxic effects on the mothers were also seen at this dose. At 5 mg/kg/day, there was decreased survival of fetuses and decreased maternal and foetal weights. It does not appear likely that oxyfluorfen will cause reproductive effects in humans at likely levels of exposure.
- **Teratogenic effects:** In a developmental study with rabbits, 30 mg/kg/day, the highest dose tested, produced an increase in fused sternal bones in the fetuses as well as toxic effects on the mothers. These data suggest oxyfluorfen may have teratogenic effects, but only at very high doses.
- **Mutagenic effects:** Mutagenicity tests on rats, mice and on bacterial cell cultures have produced mixed results. However, unscheduled DNA synthesis assays have been negative. Due to the conflicting results, it is not possible to determine the mutagenic potential of oxyfluorfen.
- **Carcinogenic effects:** In a 20-month study with mice fed 0.3, 3, or 30 mg/kg/day, doses at and above 3 mg/kg/day produced non-significant increases in both benign and malignant liver tumors in male mice. No increased tumor formation was seen in female mice at any dose. No carcinogenic effects were observed in a 23-year study with rats fed doses 2 mg/kg/day, nor in dogs at doses of 3 mg/kg/day. These data suggest that oxyfluorfen is not carcinogenic.
- **Organ toxicity:** The liver appears to be the main target organ, based on long-term feeding studies.
- **Fate in humans and animals:** Because oxyfluorfen is highly hydrophobic, it may have the potential to bioconcentrate in animal fatty tissues

[* *The Pesticides Manual, Incorporating The Agrochemicals Handbook, 10th Edition, Editor Clive Tomlin, 1994, British Crop Protection Council*]

N-METHYL-2-PYRROLIDONE

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 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 Oxxel	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
liquid hydrocarbons	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
oxyfluorfen	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	96h	Algae or other aquatic plants	<0.001mg/L	4
	EC50	72h	Algae or other aquatic plants	0.00004-0.0001mg/l	4
	NOEC(ECx)	72h	Algae or other aquatic plants	0.00002mg/l	4
	LC50	96h	Fish	0.176-0.419mg/L	4
	EC50	48h	Crustacea	0.081-0.203mg/L	4
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					

Marine pollutant. Dangerous to fish. Do not spray in high winds.
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
oxyfluorfen	HIGH	HIGH
N-methyl-2-pyrrolidone	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
oxyfluorfen	HIGH (LogKOW = 6.0465)
N-methyl-2-pyrrolidone	LOW (BCF = 0.16)

Mobility in soil

Ingredient	Mobility
oxyfluorfen	LOW (KOC = 46840)
N-methyl-2-pyrrolidone	LOW (KOC = 20.94)



SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<div><div>▶ Recycle wherever possible or consult manufacturer for recycling options.</div><div>▶ Consult State Land Waste Authority for disposal.</div><div>▶ Bury or incinerate residue at an approved site.</div><div>▶ Recycle containers if possible, or dispose of in an authorised landfill.</div></div>
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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 oxyfluorfen)	
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 oxyfluorfen)	
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 oxyfluorfen)		
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
liquid hydrocarbons	Not Available
oxyfluorfen	Not Available
N-methyl-2-pyrrolidone	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
liquid hydrocarbons	Not Available
oxyfluorfen	Not Available
N-methyl-2-pyrrolidone	Not Available

SECTION 15 Regulatory information

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

liquid hydrocarbons is found on the following regulatory lists

Not Applicable

oxyfluorfen is found on the following regulatory lists

Australia Chemicals with non-industrial uses removed from the Australian Inventory of Chemical Substances (old Inventory)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

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
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National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (oxyfluorfen)
Canada - NDSL	No (oxyfluorfen; N-methyl-2-pyrrolidone)
China - IECSC	No (oxyfluorfen)
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (oxyfluorfen)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (oxyfluorfen)
USA - TSCA	No (oxyfluorfen)
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	No (oxyfluorfen)
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	10/03/2023
Initial Date	30/09/2014

SDS Version Summary

Version	Date of Update	Sections Updated
8.1	10/12/2021	Classification change due to full database hazard calculation/update.
9.1	10/03/2023	Classification change due to full database hazard calculation/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
- 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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