

Oxy 500 SC®

Arxada NZ Limited

Chemwatch: 5479-38

Version No: 7.2

Safety Data Sheet according to the Health and Safety at Work (Hazardous Substances) Regulations 2017

Chemwatch Hazard Alert Code: 2

Initial Date: 22/07/2021

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Print Date: 18/09/2025

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

Product Identifier

Product name	Oxy 500 SC®
Chemical Name	Not Applicable
Synonyms	ACVM approval: P007996
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. Use according to manufacturer's directions.
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Details of the manufacturer or importer of the safety data sheet

Registered company name	Arxada NZ Limited
Address	13-15 Hudson Road Bell Block New Plymouth 4312 New Zealand
Telephone	+64 6 755 9234
Fax	+64 6 755 1174
Website	www.arxada.co.nz
Email	office-newplymouth@arxada.com

Emergency telephone number

Association / Organisation	Arxada NZ Limited
Emergency telephone number(s)	0800 243 622
Other emergency telephone number(s)	+64 4 917 9888 (International)

SECTION 2 Hazards identification

Classification of the substance or mixture

Classification ^[1]	Serious Eye Damage/Eye Irritation Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 2, Hazardous to the Aquatic Environment Acute Hazard Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
Signal word	Warning

Hazard statement(s)

H319	Causes serious eye irritation.
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

P260	Do not breathe mist/vapours/spray.
P273	Avoid release to the environment.

P280	Wear protective gloves, protective clothing, eye protection and face protection.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P314	Get medical advice/attention if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.

Precautionary statement(s) Storage

Not Applicable

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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No further product hazard information.

SECTION 3 Composition / information on ingredients**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
42874-03-3	<60	<u>oxyfluorfen</u>
9038-95-3	<3	<u>monobutyl ether ethoxylated, propoxylated</u>
Legend:	1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 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 or combustion products are inhaled remove from contaminated area. Lay patient down. Keep warm and rested. Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. Transport to hospital, or doctor.
Ingestion	<ul style="list-style-type: none"> IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY. For advice, contact a Poisons Information Centre or a doctor. Urgent hospital treatment is likely to be needed. In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist. If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS. <p>Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:</p> <ul style="list-style-type: none"> INDUCE vomiting with fingers down the back of the throat, ONLY IF CONSCIOUS. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. <p>NOTE: Wear a protective glove when inducing vomiting by mechanical means.</p>

Indication of any immediate medical attention and special treatment needed

for chlorinated diphenyl ethers

If large amounts are ingested, gastric lavage is suggested. In the case of splashes in the eyes, a petrolatum-based ophthalmic ointment may be applied to the eye to relieve the irritating effects.

Preplacement and annual medical examinations of workers, with emphasis on liver function, skin condition, reproductive history, are recommended

Treat symptomatically.

Following exposures to chlorophenoxy compounds:

- Acute toxic reactions are rare. The by-product of production, dioxin, may be implicated in subacute features such as hepatic enlargement, chloracne, neuromuscular symptoms and deranged porphyrin metabolism.
- Large intentional overdoses result in coma, metabolic acidosis, myalgias, muscle weakness, elevated serum creatine kinase, myoglobinuria, irritation of the skin, eyes, respiratory tract and gut and mild renal and hepatic dysfunction.
- Several cases of sensorimotor peripheral neuropathies have been associated with chronic dermal exposure to 2,4-D. For acute exposures the usual methods of gut and skin contamination (lavage, charcoal, cathartic) are recommended in the first several hours. Alkalisation of the urine and generous fluid replacement have the added benefit of treating any myoglobinuria present. Monitor metabolic acidosis, hyperthermia, hyperkalaemia, myoglobinuria and hepatic/renal dysfunction. for 2,4-dichlorophenoxyacetic acid (2,4-D) and its derivatives
- Gastric lavage if there are no signs of impending convulsions.
- Cautious administration of short-acting anticonvulsant drug if convulsions appear imminent.

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- ▶ General supportive measures for central nervous system depression.
- ▶ If hypotension appears, search vigorously for a contributing cause (e.g. dehydration, electrolyte balance, acidosis, myocardial disturbances and hyperpyrexia).
- ▶ As appropriate, treat dehydration, electrolyte disturbances, acidosis, and hyperexia.
- ▶ To promote excretion of 2,4-D, initiate alkaline diuresis, as in salicylate poisoning by injecting sodium bicarbonate, intravenously, until the urine pH exceeds 7.5 and then infuse mannitol; renal clearance rises sharply as urine pH rises above 7.5 - above pH 8.0, it is said to be 100-fold greater than pH 6.0.
- ▶ If cardiac disturbances are suspected, monitor ECG continuously when possible. Prepare to deliver defibrillating shocks in the event of ventricular fibrillation.
- ▶ If hypotension intensifies, a trial with a vasopressor drug may be appropriate. Adrenalin (epinephrine) should be avoided because of possible fibrillation.
- ▶ If myotonia appears, a trial with quinidine may be helpful.
- ▶ Physiotherapy may be necessary for motion disorders associated with peripheral neuritis, myopathy or brain stem dysfunction.

GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products, 5th Ed.

In general, chlorophenoxy herbicides are rapidly absorbed from the gastrointestinal tract and evenly distributed throughout the body; accumulation in human tissues is not expected. A steady-state level in the human body will be achieved within 3–5 days of exposure. The herbicides are eliminated mainly in the urine, mostly unchanged, although fenoprop may be conjugated to a significant extent. Biological half-lives of chlorophenoxy herbicides in mammals range from 10 to 33 h; between 75% and 95% of the ingested amount is excreted within 96 h. Dogs appear to retain chlorophenoxy acids longer than other species as a result of relatively poor urinary clearance and thus may be more susceptible to their toxic effects. Metabolic conversions occur only at high doses. The salt and ester forms are rapidly hydrolysed and follow the same pharmacokinetic pathways as the free acids.

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	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves in the event of a fire. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Use fire fighting procedures suitable for surrounding 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"> ▶ The material is not readily combustible under normal conditions. ▶ However, it will break down under fire conditions and the organic component may burn. ▶ Not considered to be a significant fire risk. ▶ Heat may cause expansion or decomposition with violent rupture of containers. ▶ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. <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>

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	<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. ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Neutralise/decontaminate residue (see Section 13 for specific agent). ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▶ If contamination of drains or waterways occurs, advise emergency services. <p>Environmental hazard - contain spillage.</p>

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	<p>▶ DO NOT allow clothing wet with material to stay in contact with skin</p> <p>The tendency of many ethers to form explosive peroxides is well documented. Ethers lacking non-methyl hydrogen atoms adjacent to the ether link are thought to be relatively safe</p> <p>▶ DO NOT concentrate by evaporation, or evaporate extracts to dryness, as residues may contain explosive peroxides with DETONATION potential.</p> <p>▶ Any static discharge is also a source of hazard.</p> <p>▶ Before any distillation process remove trace peroxides by shaking with excess 5% aqueous ferrous sulfate solution or by percolation through a column of activated alumina.</p> <p>▶ Distillation results in uninhibited ether distillate with considerably increased hazard because of risk of peroxide formation on storage.</p> <p>▶ Add inhibitor to any distillate as required.</p> <p>▶ When solvents have been freed from peroxides by percolation through columns of activated alumina, the absorbed peroxides must promptly be desorbed by treatment with polar solvents such as methanol or water, which should then be disposed of safely.</p> <p>▶ Avoid all personal contact, including inhalation.</p> <p>▶ Wear protective clothing when risk of exposure occurs.</p> <p>▶ Use in a well-ventilated area.</p> <p>▶ Avoid contact with moisture.</p> <p>▶ Avoid contact with incompatible materials.</p> <p>▶ When handling, DO NOT eat, drink or smoke.</p> <p>▶ Keep containers securely sealed when not in use.</p> <p>▶ Avoid physical damage to containers.</p> <p>▶ Always wash hands with soap and water after handling.</p> <p>▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use.</p> <p>▶ Use good occupational work practice.</p> <p>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</p> <p>▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.</p>
Other information	<p>▶ Store in original containers.</p> <p>▶ Keep containers securely sealed.</p> <p>▶ No smoking, naked lights or ignition sources.</p> <p>▶ Store in a cool, dry, well-ventilated area.</p> <p>▶ Store away from incompatible materials and foodstuff containers.</p> <p>▶ Protect containers against physical damage and check regularly for leaks.</p> <p>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</p>

Conditions for safe storage, including any incompatibilities

Suitable container	<p>HDPE jerry can.</p> <p>▶ Polyethylene or polypropylene container.</p> <p>▶ Packing as recommended by manufacturer.</p> <p>▶ Check all containers are clearly labelled and free from leaks.</p>
Storage incompatibility	<p>▶ Avoid reaction with oxidising agents, bases and strong reducing agents.</p> <p>▶ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.</p>



X — Must not be stored together

O — May be stored together with specific preventions

+ — May be stored together

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
New Zealand Workplace Exposure Standards (WES)	oxyfluorfen	Inhalable dust (not otherwise classified)	10 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	oxyfluorfen	Respirable dust (not otherwise classified)	3 mg/m3	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
oxyfluorfen	Not Available	Not Available
monobutyl ether ethoxylated, propoxylated	Not Available	Not Available

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

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. An approved self contained breathing apparatus (SCBA) may be required in some situations. Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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

Within each range the appropriate value depends on:

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

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

Individual protection measures, such as personal protective equipment



Eye and face protection

- ▶ 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

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

NOTE:

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

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.

The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.

Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

- frequency and duration of contact,
- chemical resistance of glove material,
- glove thickness and
- dexterity

Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).

· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.

· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.

· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.

- Contaminated gloves should be replaced.

As defined in ASTM F-739-96 in any application, gloves are rated as:

- Excellent when breakthrough time > 480 min
- Good when breakthrough time > 20 min
- Fair when breakthrough time < 20 min
- Poor when glove material degrades

For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.

It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.

Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.

Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:

· Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.

· Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential

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	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.
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.

Respiratory protection

Type A-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 10 x ES	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

^ - Full-face

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

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

SECTION 9 Physical and chemical properties**Information on basic physical and chemical properties**

Appearance	Light yellow suspension; dispersible in water.		
Physical state	Liquid	Relative density (Water = 1)	1.19
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable
pH (as supplied)	7-8	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Applicable	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Dispersible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Heat of Combustion (kJ/g)	Not Available	Ignition Distance (cm)	Not Available
Flame Height (cm)	Not Available	Flame Duration (s)	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	Enclosed Space Ignition Deflagration Density (g/m3)	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

a) Acute Toxicity	Based on available data, the classification criteria are not met.
b) Skin Irritation/Corrosion	Based on available data, the classification criteria are not met.
c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	Based on available data, the classification criteria are not met.
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	Based on available data, the classification criteria are not met.
g) Reproductivity	Based on available data, the classification criteria are not met.
h) STOT - Single Exposure	Based on available data, the classification criteria are not met.
i) STOT - Repeated Exposure	There is sufficient evidence to classify this material as toxic to specific organs through repeated exposure
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	<p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>In 134-day inhalation experiments with chlorinated diphenyl ethers the 54% and 57% chlorinated materials produced liver injury, in rats, characterised by congestion and fatty liver degeneration.</p> <p>Inhalation hazard is increased at higher temperatures.</p> <p>Acute effects from inhalation of high vapour concentrations may be chest and nasal irritation with coughing, sneezing, headache and even nausea.</p>
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.
Skin Contact	<p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</p> <p>Limited evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	<p>On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.</p> <p>Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.</p> <p>There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals.</p> <p>Studies with some glycol ethers (principally the monoethylene glycols) and their esters indicate reproductive changes, testicular atrophy, infertility and kidney function changes. The metabolic acetic acid derivatives of glycol ethers (alkoxyacetic acids), not the ether itself, have been found to be the proximal reproductive toxin in animals. The potency of these metabolites decreases significantly as the chain length of the ether increases. Consequently glycol ethers with longer substituents (e.g diethylene glycols, triethylene glycols) have not generally been associated with reproductive effects. One of the most sensitive indicators of toxic effects observed from many of the glycol ethers is an increase in the erythrocytic osmotic fragility in rats Which produces haemolytic anaemia). This appears to be related to the development of haemoglobinuria (blood in the urine) at higher exposure levels or as a result of chronic exposure.</p> <p>Glycol ethers based on propylene oxides, propylene glycol ethers, dipropylene glycol ethers and tripropylene glycol ethers are mainly available, commercially, as alpha-isomers (because of thermodynamic considerations); these are incapable of forming alkoxyacetic or alkoxypropionic acids as metabolites and therefore do not produce erythrocyte fragility unless contaminated by ethylene glycol ethers or to a significant degree by the beta-isomer. beta-Isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).</p> <p>Chlorinated diphenyl ethers may penetrate into the human body by cutaneous, respiratory or digestive exposure. Laboratory workers handling the chlorinated phenyl ethers, have shown liver damage after prolonged exposure</p> <p>Prolonged contact with chlorinated diphenyl ethers (CDPE) and their halogenated analogues may cause skin irritation, weight loss and liver injury. Repeated absorption has produced liver damage in animals. Research has shown that very low doses of the brominated derivative (BDPE) given to baby mice, leads to irreparable brain damage, causing reduced learning capacity and hyperactive behaviour.</p> <p>CDPE and other halogenated congeners are often by-products in the manufacture of halogenated phenols</p> <p>Workers exposed to chlorophenoxy herbicides show a significant increase in soft-tissue sarcoma, malignant lymphomas and bronchial carcinomas. Prolonged or repeated contact with solutions may result in non-allergic dermatoses.</p> <p>Until recently, most epidemiological studies of the effects of chlorophenoxy herbicides dealt with populations exposed in the 1950s and 1960s, when the trichlorophenol-based herbicides 2,4,5-T and fenoprop were contaminated with polychlorinated dioxins and furans, including 2,3,7,8-tetrachlorodibenzodioxin (TCDD); the effects observed may therefore have been a consequence of the presence of the dioxin contaminants. In addition, most epidemiological studies on chlorophenoxy herbicides conducted to date have involved multiple exposures to chemical agents, including other pesticides and synthetic organic compounds. In a series of case-referent studies conducted in Sweden in the late 1970s and early 1980s, strong associations were noted between soft tissue sarcomas (STS) and multiple lymphomas (including Hodgkin disease (HD) and non-Hodgkin lymphoma (NHL)) and the use of chlorophenoxy herbicides by agricultural or forestry workers. The association between STS and chlorophenoxy herbicide use observed in the Swedish studies has not been confirmed in other case-referent studies. Although a number of cohort studies of occupationally exposed workers have been conducted, the small size of many of them limits their usefulness in assessing the relationship between STS and the herbicides. The risk for malignant lymphoma (HD + NHL) was almost five times greater for agricultural and forestry workers exposed to a mixture of chlorophenoxy herbicides than for controls in the case-referent study in Sweden but was not</p>

significantly elevated in a Danish cohort study of 3390 workers in a chemical plant manufacturing MCPA, dichlorprop, mecoprop, and 2,4-D, as well as other industrial chemicals and dyes

Chronic exposure to 2,4-dichlorophenoxyacetic acid(2,4-D), its salts and its esters and its analogues may result in nausea, liver function changes, contact toxic dermatitis, irritation of the airways and eyes, as well as neurological changes. Persons with chronic diseases of the central nervous system, liver, heart, kidneys, lungs and skin, as well as those with endocrinological or immunological disturbances should not be exposed to herbicides (ILO Encyclopaedia). Groups of rats receiving 2,4-D in their diets for 13 weeks showed growth retardation and decreased food intake at 150 mg/kg/day dosage and an increased serum glutamic pyruvic transaminase (SGPT). A statistically significant incidence of astrocytoma was seen in the brains of male rats receiving 45 mg/kg/day for 104 weeks suggesting a possible carcinogenic effect although the prevalence of naturally occurring tumours in controls makes this result equivocal. A controversial study implicating 2,4-D as the cause of non-Hodgkin's lymphoma among male Kansas residents, aged 21 years or older, was difficult to evaluate because of a number of confounding factors. Agent Orange, a mixture of 2,4-D and 2,4,5-T, with contamination from 2,3,7,8-tetrachlorodibenzo-p-dioxin (also referred to as "dioxin" or TCDD) has been studied due to exposure of military personnel during its use as a herbicide in Vietnam. Neurological, reproductive and carcinogenic effects, purported to have occurred amongst veterans may be related to 2,4-D and 2,4,5-T but given the toxicity of the other components this remains the subject of conjecture.

Most, if not all, occupational illnesses associated with 2,4,5-trichlorophenoxyacetic acids (2,4,5-T) and its derivatives actually result from TCDD contamination.

Repeated overexposure to phenoxy herbicides may cause liver, kidney, gastrointestinal and muscular effects.

Subchronic exposure by dogs to phenoxy herbicides produced a reduction in circulating lymphocytes Teratogenic response was exhibited in mice (but not rats). Cleft palate was demonstrated. No such findings occurred in non-human primates given up to 10 mg/kg/day (containing 0.05 ppm TCDD) from gestation day 22 to 38.

The no-observed effect level (NOAEL) in hamsters was 2 mg/kg 2,4,5-T

Besides PCBs, other structurally related compounds have been demonstrated to elicit dioxin-like activity. These include any or all of the following classes of polyhalogenated compounds: benzenes, naphthalenes, diphenyl ethers, diphenyl toluenes, phenoxy anisoles, biphenyl anisoles, xanthenes, xanthones, anthracenes, fluorenes, dihydroanthracenes, biphenyl methanes, phenylxylethanes, dibenzothiophenes, quaterphenyls, quaterphenyl ethers, and biphenylenes. However, due to the lack of information on environmental occurrence and the limited data to compare their toxicity relative to that of 2,3,7,8-TCDD, no international agreement has yet been reached to derive Toxic Equivalency Factors (TEFs) for compounds other than the group of dioxin-like PCBs

Oxy 500 SC®	TOXICITY	IRRITATION
	Not Available	Not Available
oxyfluorfen	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >10000 mg/kg ^[2]	Not Available
	Oral (Dog) LD50; >5000 mg/kg ^[2]	
monobutyl ether ethoxylated, propoxylated	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >20000 mg/kg ^[2]	Eye (Rodent - rabbit): 50mg - Severe
	Inhalation (Rat) LC50: 0.147 mg/L4h ^[2]	Skin (Rodent - rabbit): 500mg - Mild
	Oral (Rat) LD50: >5000 mg/kg ^[2]	Skin (Rodent - rabbit): 500mg - Mild
		Skin (Rodent - rabbit): 500mg - Mild
		Skin (Rodent - rabbit): 500mg - Mild
		Skin (Rodent - rabbit): 500mg - Mild
		Skin (Rodent - rabbit): 500mg - Mild
		Skin (Rodent - rabbit): 500mg - Mild
		Skin (Rodent - rabbit): 500mg - Mild
		Skin (Rodent - rabbit): 500mg - Mild

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

OXYFLUORFEN	<p>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 *</p> <p>For Protoporphyrinogen Oxidase (PPO) Inhibitors:</p> <p>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.</p> <p>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</p> <p>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</p> <p>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 haematopoiesis 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</p> <p>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</p>
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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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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.

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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 foetuses 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]

MONOBUTYL ETHER ETHOXYLATED, PROPOXYLATED

Polyalkylene glycol monobutyl ether (PGME) polymers have a low degree of toxicity. Toxicity by ingestion is low, but highest for lower molecular weight products *(molecular weight <1500). Thus, the FDA has restricted the use of PGME in sanitizing solutions that may contact surfaces that contact foods to a 0.05% aqueous solution of polymers that have an average molecular weight of 2,400-3,300 and a cloud-point of 90-100 degrees C. In this application, large PGME polymers engulf (micellize) oils and smaller particulates that are subsequently precipitated at temperatures of 90-100 deg C.

Toxicity by skin contact is low for all PGME species. Higher molecular weight PGME polymers have been found to be toxic in animals upon inhalation of mechanically generated mists. The products of thermal degradation are also toxic.

Eye injury is possible only for the lowest molecular weight species.

There have been no reported effects of PGME on human health. However, data from toxicity studies performed in appropriate animal species provides information on potential human toxicity. PGME polymers with molecular weight greater than 1500, have limited acute toxicity by the oral (LD50, rat, 8630 mg/kg) and dermal (LD50, rabbit, >8,000 mg/kg) routes, while toxicity of the high molecular weight products through inhalation is increased (LC50, 4 hours, rat, > 5 mg/kg). Oral and dermal toxicity in general increases with lower molecular weight products.

This product has not been evaluated for genetic, developmental, or reproductive toxicity. PGME is not known to be an irritant or an allergen. Human beings have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents, and other cleaning products. Exposure to these chemicals can occur through ingestion, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that volumes well above a reasonable intake level would have to occur to produce any toxic response. Moreover, no fatal case of poisoning with alcohol ethoxylates has ever been reported. Multiple studies investigating the acute toxicity of alcohol ethoxylates have shown that the use of these compounds is of low concern in terms of oral and dermal toxicity.

Clinical animal studies indicate these chemicals may produce gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhea, and lethargy. Similarly, slight to severe irritation of the skin or eye was generated when undiluted alcohol ethoxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotoxin, carcinogen, or mutagen (HERA 2007). No information was available on levels at which these effects might occur, though toxicity is thought to be substantially lower than that of nonylphenol ethoxylates.

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture.

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autooxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose allergic contact dermatitis (ACD) to these compounds by patch testing

Overall, alcohol alkoxyates (AAs) are not expected to be systemically toxic, although some short chain ethylene glycol ethers, e.g. methyl and ethyl homologues are of concern for a range of adverse health effects. They include skin and eye irritation, liver and kidney damage, bone marrow and central nervous system (CNS) depression, testicular atrophy, developmental toxicity, and immunotoxicity. For higher propyl and butyl homologues, the toxicity involves haemolysis (anaemia) with secondary effects relating to haemosiderin accumulation in the spleen, liver and kidney, and compensatory haematopoiesis in the bone marrow. Systemic toxicity was shown to decrease with increasing alkyl chain lengths and/or alkoxylation degrees (ECETOC, 2005; US EPA, 2010). The chemicals ethylene glycol hexyl ether (with a longer alkyl chain length, CAS No. 112-25-4) and diethylene glycol butyl ether (with a higher ethoxylation degree, CAS No. 112-34-5) have no evidence of systemic effects including haemolysis.

Commercially available AAs are mixtures of homologues of varying carbon chain lengths and it is possible that some of the chemicals with an average alkyl chain length C ≥ 6 may also contain shorter alkyl chains C < 6. It is not practical to quantify the proportion of shorter C < 6 chain lengths present in such chemicals, or these shorter chain lengths may not be present at all. The available data suggest a lack of systemic toxicity for the AE chemicals with potential short alkyl chain presence (NICNASa); therefore, the toxicity of the chemicals in this assessment is unlikely to be significantly affected by the presence of shorter chain alkyl groups.

For high boiling ethylene glycol ethers (typically triethylene- and tetraethylene glycol ethers):

Skin absorption: Available skin absorption data for triethylene glycol ether (TGBE), triethylene glycol methyl ether (TGME), and triethylene glycol ethylene ether (TGEE) suggest that the rate of absorption in skin of these three glycol ethers is 22 to 34 micrograms/cm2/hr, with the methyl ether having the highest permeation constant and the butyl ether having the lowest. The rates of absorption of TGBE, TGEE and TGME are at least 100-fold less than EGME, EGEE, and EGBE, their ethylene glycol monoalkyl ether counterparts, which have absorption rates that range from 214 to 2890 micrograms/cm2/hr. Therefore, an increase in either the chain length of the alkyl substituent or the number of ethylene glycol moieties appears to lead to a decreased rate of percutaneous absorption. However, since the ratio of the change in values of the ethylene glycol to the diethylene glycol series is larger than that of the diethylene glycol to triethylene glycol series, the effect of the length of the chain and number of ethylene glycol moieties on absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetraethylene glycol methyl ether (TetraME) and

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tetraethylene glycol butyl ether (TetraBE) are expected to be less permeable to skin than TGME and TGBE, the differences in permeation between these molecules may only be slight.

Metabolism: The main metabolic pathway for metabolism of ethylene glycol monoalkyl ethers (EGME, EGEE, and EGBE) is oxidation via alcohol and aldehyde dehydrogenases (ALD/ADH) that leads to the formation of an alkoxy acids. Alkoxy acids are the only toxicologically significant metabolites of glycol ethers that have been detected *in vivo*. The principal metabolite of TGME is believed to be 2-[2-(2-methoxyethoxy)ethoxy] acetic acid. Although ethylene glycol, a known kidney toxicant, has been identified as an impurity or a minor metabolite of glycol ethers in animal studies it does not appear to contribute to the toxicity of glycol ethers.

The metabolites of category members are not likely to be metabolized to any large extent to toxic molecules such as ethylene glycol or the mono alkoxy acids because metabolic breakdown of the ether linkages also has to occur.

Acute toxicity: Category members generally display low acute toxicity by the oral, inhalation and dermal routes of exposure. Signs of toxicity in animals receiving lethal oral doses of TGBE included loss of righting reflex and flaccid muscle tone, coma, and heavy breathing. Animals administered lethal oral doses of TGEE exhibited lethargy, ataxia, blood in the urogenital area and piloerection before death.

Irritation: The data indicate that the glycol ethers may cause mild to moderate skin irritation. TGEE and TGBE are highly irritating to the eyes. Other category members show low eye irritation.

Repeat dose toxicity: Results of these studies suggest that repeated exposure to moderate to high doses of the glycol ethers in this category is required to produce systemic toxicity.

In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME.

Testicular effects included spermatid giant cells, focal tubular hypospermatogenesis, and increased cytoplasmic vacuolisation. Due to a high incidence of similar spontaneous changes

in normal New Zealand White rabbits, the testicular effects were considered not to be related to treatment. Thus, the NOAELs for TGME, TGEE and TGBE were established at 1000 mg/kg/day. Findings from this report were considered unremarkable.

A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day. In this study, significantly-increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500 mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or

haemolysed blood in the stomach. These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats.

In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity.

Mutagenicity: Mutagenicity studies have been conducted for several category members. All *in vitro* and *in vivo* studies were negative at concentrations up to 5,000 micrograms/plate and 5,000 mg/kg, respectively, indicating that the category members are not genotoxic at the concentrations used in these studies. The uniformly negative outcomes of various mutagenicity studies performed on category members lessen the concern for carcinogenicity.

Reproductive toxicity: Although mating studies with either the category members or surrogates have not been performed, several of the repeated dose toxicity tests with the surrogates have included examination of reproductive organs. A lower molecular weight glycol ether, ethylene glycol methyl ether (EGME), has been shown to be a testicular toxicant. In addition, results of repeated dose toxicity tests with TGME clearly show testicular toxicity at an oral dose of 4,000 mg/kg/day four times greater than the limit dose of 1,000 mg/kg/day recommended for repeat dose studies. It should be noted that TGME is 350 times less potent for testicular effects than EGME. TGBE is not associated with testicular toxicity, TetraME is not likely to be metabolised by any large extent to 2-MAA (the toxic metabolite of EGME), and a mixture containing predominantly methylated glycol ethers in the C5-C11 range does not produce testicular toxicity (even when administered intravenously at 1,000 mg/kg/day).

Developmental toxicity: The bulk of the evidence shows that effects on the foetus are not noted in treatments with 1,000 mg/kg/day during gestation. At 1,250 to 1,650 mg/kg/day TGME (in the rat) and 1,500 mg/kg/day (in the rabbit), the developmental effects observed included skeletal variants and decreased body weight gain.

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

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

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

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

>20 EO is not classified (CESIO 2000)

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

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

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

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

AEs are not contact sensitizers. Neat AE are irritating to eyes and skin. The irritation potential of aqueous solutions of AEs depends on concentrations. Local dermal effects due to direct or indirect skin contact in certain use scenarios where the products are diluted are not of concern as AEs are not expected to be irritating to the skin at in-use concentrations. Potential irritation of the respiratory tract is not a concern given the very low levels of airborne AE generated as a consequence of spray cleaner aerosols or laundry powder detergent dust.

In summary, the human health risk assessment has demonstrated that the use of AE in household laundry and cleaning detergents is safe and does not cause concern with regard to consumer use.

Acute Toxicity

✗

Carcinogenicity

✗

Skin Irritation/Corrosion

✗

Reproductivity

✗

Continued...

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

Oxy 500 SC®	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	72h	Algae or other aquatic plants	<0.001mg/L	4
	EC50	48h	Crustacea	0.081-0.203mg/L	4
	EC50	96h	Algae or other aquatic plants	<0.001mg/L	4
	EC50(ECx)	96h	Algae or other aquatic plants	<0.001mg/L	4
	LC50	96h	Fish	0.176-0.419mg/L	4
monobutyl ether ethoxylated, propoxylated	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
Legend:		Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 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 allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
for oxyfluorfen
log Kow : 4.468
Environmental fate:
Soil and water: Strongly adsorbed on soil, not readily desorbed; shows negligible leaching.
Koc from 2891 (sand) to 32381 (silty clay loam) Photodecomposition in water is rapid and on soil slow.
Microbial action is not significant.
Field dissipation DT 5-55 days; soil DT50 (in dark)(aerobic) 292 d, (anaerobic) 580 d approx.
Half-life (hr) sediment : >336
Breakdown in soil and groundwater: Oxyfluorfen is moderately persistent in most soil environments, with a representative field half-life of about 30 to 40 days . Oxyfluorfen is not subject to microbial degradation or hydrolysis . The main mechanism of degradation in soils may be photodegradation and evaporation/codistillation in moist soils . In laboratory studies, its soil half-life was 6 months, indicating very low rates of microbial degradation . Oxyfluorfen is very well-sorbed to most soils . Soil binding is highest in soils with high organic matter and clay content . Once oxyfluorfen is adsorbed to soil particles, it is not readily removed . It is practically insoluble in water, and therefore is unlikely to be appreciably mobile in most instances, unless the sorptive capacity of the soil is exceeded. Oxyfluorfen did not leach below 4 inches in any soil except sand .
Breakdown in water: In water, oxyfluorfen is rapidly decomposed by light . Because oxyfluorfen is nearly insoluble in water and has a tendency to adsorb to soil, it will be sorbed to suspended particles or sediments .
Breakdown in vegetation: There is very little movement of oxyfluorfen within treated plants. It is not readily metabolised by plants, but since it is not readily taken up by roots, residues in plants are generally very low . Residues of oxyfluorfen accumulated in carrots and oats grown on previously treated fields, but not in cotton or lettuce
Bioaccumulation: Oxyfluorfen accumulated up to 13 mg/kg (13,000 ug/kg) in bluegill sunfish exposed to 10 ug/L for 40 days. This represents a bioconcentration factor (BCF) of 1300. The BCF in channel catfish was 700 to 5000 in one 30-day study . These results indicate a low to moderate potential for bioaccumulation in aquatic species
Ecotoxicity:
Practically non-toxic to birds.
Birds: Acute LD50 for bobwhite quail >2150 mg/kg; mallard duck >4000 mg/kg
Eight day dietary LC50 for mallard ducks and bobwhite quail >5000 ppm
Dietary concentrations as high as 100 ppm had no effect on reproduction in mallards or bobwhite quail
Oxyfluorfen is highly toxic to aquatic invertebrates, freshwater clams, oysters, aquatic plants, and fish
Fish: LC50 (96 h) for bluegill 0.2, trout 0.41, channel catfish 0.4 mg/l; fathead minnow 0.15 mg/l
Grass-shrimp, oysters: 0.032 mg/l
Freshwater clams LC50 (96 h): 0.01 ug/l
Daphnia magna EC50 (48 h): 1.5 mg ai/l
Daphnia magna LC50 (96 h): 1.5 mg/l (Goal 2E)
Bees; non-toxic to honeybees at 0.025 mg ai/bee

DO NOT discharge into sewer or waterways.

Persistence and degradability

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

Bioaccumulative potential

Ingredient	Bioaccumulation
oxyfluorfen	HIGH (LogKOW = 6.0465)
monobutyl ether ethoxylated, propoxylated	LOW (LogKOW = 1.1)

Mobility in soil

Ingredient	Mobility
oxyfluorfen	LOW (Log KOC = 46840)

SECTION 13 Disposal considerations**Waste treatment methods**

Product / Packaging disposal	<ul style="list-style-type: none"> Containers may still present a chemical hazard/ danger when empty. Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. Where possible retain label warnings and SDS and observe all notices pertaining to the product. Due to their environmental persistence and potential health hazards, PCBs, PBBs, dioxins and their derivatives or congeners (including chlorinated diphenyl ethers), cannot be disposed of in landfills or dumped at sea. Environmentally acceptable method of disposal include high temperature incineration. However this option is costly and uncertain. Other acceptable disposal technologies include base-catalysed dechlorination in the BCD (Base-Catalyzed Decomposition) Process. Currently, most wastes must be stored in an approved manner until satisfactory arrangements can be made for their disposal. All wastes and residues containing these substances (e.g. wiping clothes, absorbent materials, used disposable protective gloves, contaminated clothing, etc.) should be collected, placed in proper containers, labelled and disposed of in the manner prescribed by government regulations. Regulations may require the compulsory reporting of all spills. <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> Reduction Reuse Recycling Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> DO NOT allow wash water from cleaning or process equipment to enter drains. It may be necessary to collect all wash water for treatment before disposal. In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. Where in doubt contact the responsible authority. Recycle wherever possible or consult manufacturer for recycling options. Consult State Land Waste Authority for disposal. Bury or incinerate residue at an approved site. Recycle containers if possible, or dispose of in an authorised landfill.

Ensure that the hazardous substance is disposed in accordance with the Hazardous Substances (Disposal) Notice 2017

Disposal Requirements



Packages that have been in direct contact with the hazardous substance must be only disposed if the hazardous substance was appropriately removed and cleaned out from the package. The package must be disposed according to the manufacturer's directions taking into account the material it is made of. Packages which hazardous content have been appropriately treated and removed may be recycled.

The hazardous substance must only be disposed if it has been treated by a method that changed the characteristics or composition of the substance and it is no longer hazardous.

Only dispose to the environment if a tolerable exposure limit has been set for the substance.

Only deposit the hazardous substance into or onto a landfill or sewage facility or incinerator, where the hazardous substance can be handled and treated appropriately.

SECTION 14 Transport information**Labels Required**

	
Marine Pollutant	
HAZCHEM	•3Z

Land transport (UN)

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)	<table> <tr> <td>Class</td><td>9</td></tr> <tr> <td>Subsidiary Hazard</td><td>Not Applicable</td></tr> </table>	Class	9	Subsidiary Hazard	Not Applicable
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
	Limited quantity	5 L

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. Maritime transport in bulk according to IMO instruments

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
oxyfluorfen	Not Available
monobutyl ether ethoxylated, propoxylated	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
oxyfluorfen	Not Available
monobutyl ether ethoxylated, propoxylated	Not Available

SECTION 15 Regulatory information

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

This substance is to be managed using the conditions specified in an applicable Group Standard

HSR Number	Group Standard
HSR100031	Not Available

Please refer to Section 8 of the SDS for any applicable tolerable exposure limit or Section 12 for environmental exposure limit.

oxyfluorfen is found on the following regulatory lists

- International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
- New Zealand Approved Hazardous Substances with controls
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
- New Zealand Inventory of Chemicals (NZIoC)

New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 1 Quantity limits for dangerous goods
New Zealand Workplace Exposure Standards (WES)

monobutyl ether ethoxylated, propoxylated is found on the following regulatory lists

- New Zealand Approved Hazardous Substances with controls
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
- New Zealand Inventory of Chemicals (NZIoC)

Additional Regulatory Information

Not Applicable

Hazardous Substance Location

Subject to the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Quantities
Not Applicable	Not Applicable

Certified Handler

Subject to Part 4 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Class of substance	Quantities
Not Applicable	Not Applicable

Refer Group Standards for further information

Maximum quantities of certain hazardous substances permitted on passenger service vehicles

Subject to Regulation 13.14 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Gas (aggregate water capacity in mL)	Liquid (L)	Solid (kg)	Maximum quantity per package for each classification
Not Applicable	Not Applicable	Not Applicable	Not Applicable	Not Applicable

Tracking Requirements

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (oxyfluorfen)
Canada - NDSL	No (oxyfluorfen; monobutyl ether ethoxylated, propoxylated)
China - IECSC	No (oxyfluorfen)
Europe - EINEC / ELINCS / NLP	No (monobutyl ether ethoxylated, propoxylated)
Japan - ENCS	No (oxyfluorfen)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (oxyfluorfen)
USA - TSCA	TSCA Inventory 'Active' substance(s) (monobutyl ether ethoxylated, propoxylated); No (oxyfluorfen)
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	No (oxyfluorfen)
UAE - Control List (Banned/Restricted Substances)	No (monobutyl ether ethoxylated, propoxylated)
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	15/09/2025
Initial Date	22/07/2021

SDS Version Summary

Version	Date of Update	Sections Updated
7.1	15/09/2025	Identification of the substance / mixture and of the company / undertaking - Synonyms
7.2	17/09/2025	Identification of the substance / mixture and of the company / undertaking - Synonyms

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
- ▶ MARPOL: International Convention for the Prevention of Pollution from Ships
- ▶ IMSBC: International Maritime Solid Bulk Cargoes Code
- ▶ IGC: International Gas Carrier Code
- ▶ IBC: International Bulk Chemical Code

- ▶ 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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