

AC Pulverise Herbicide

AXICHEM Pty Ltd

Chemwatch: 5154-19

Version No: 5.1

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

Chemwatch Hazard Alert Code: 3

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L.GHS.AUS.EN

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

Product Identifier

Product name	AC Pulverise Herbicide
Chemical Name	Not Applicable
Synonyms	Not Available
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	For the control of a wide range of annual and perennial broadleaf weeds. Concentrate material is measured and mixed, preferably outdoors, in proportions as recommended by manufacturer. Operators should be trained in procedures for safe use of this material.
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Details of the manufacturer or supplier of the safety data sheet

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

Emergency telephone number

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


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SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	S6
Classification ^[1]	Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
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AC Pulverise Herbicide

Signal word **Danger**

Hazard statement(s)

H302	Harmful if swallowed.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H318	Causes serious eye damage.
H335	May cause respiratory irritation.
H412	Harmful to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

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.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P330	Rinse mouth.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

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

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
32341-80-3	47.2	<u>2,4-dichlorophenoxyacetic acid triisopropanolamine salt</u>
6753-47-5	7.5	<u>picloram, triisopropanolamine salt</u>
Not Available	45.3	other nonhazardous ingredients

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

SECTION 4 First aid measures

Description of first aid measures

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Eye Contact	<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, without delay.
Ingestion	<ul style="list-style-type: none"> ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

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. Alkalisiation 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.
- ▶ 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

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

Special hazards arising from the substrate or mixture

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

Fire Fighting	<ul style="list-style-type: none"> Alert Fire Brigade and tell them location and nature of hazard. Wear breathing apparatus plus protective gloves. Prevent, by any means available, spillage from entering drains or water courses. Use water delivered as a fine spray to control fire and cool adjacent area. DO NOT approach containers suspected to be hot. Cool fire exposed containers with water spray from a protected location. If safe to do so, remove containers from path of fire. Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<ul style="list-style-type: none"> Combustible. Slight fire hazard when exposed to heat or flame. Heating may cause expansion or decomposition leading to violent rupture of containers. On combustion, may emit toxic fumes of carbon monoxide (CO). May emit acrid smoke. Mists containing combustible materials may be explosive. <p>Decomposes on heating and produces toxic fumes of:</p> <p>carbon dioxide (CO₂)</p> <p>hydrogen chloride</p> <p>phosgene</p> <p>nitrogen oxides (NO_x)</p> <p>other pyrolysis products typical of burning organic material.</p>
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

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

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

SECTION 7 Handling and storage

Precautions for safe handling

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

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Polyethylene or polypropylene container. ▶ Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Avoid strong bases. ▶ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
AC Pulverise Herbicide	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
2,4-dichlorophenoxyacetic acid triisopropanolamine salt	Not Available	Not Available
picloram, triisopropanolamine salt	Not Available	Not Available

Occupational Exposure Banding






Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
2,4-dichlorophenoxyacetic acid triisopropanolamine salt	E	≤ 0.01 mg/m ³
picloram, triisopropanolamine salt	E	≤ 0.01 mg/m ³

Notes:

Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.

MATERIAL DATA

Exposure controls

Appropriate engineering controls	Concentrate material is measured and mixed, preferably outdoors, in proportions as recommended by manufacturer. General exhaust is adequate under normal operating conditions.
Individual protection measures, such as personal protective equipment	    

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Eye and face protection	<ul style="list-style-type: none"> ▸ Safety glasses with side shields. ▸ Chemical goggles. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	<ul style="list-style-type: none"> ▸ Wear chemical protective gloves, e.g. PVC. ▸ Wear safety footwear or safety gumboots, e.g. Rubber <p>NOTE:</p> <ul style="list-style-type: none"> ▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p>
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▸ Overalls. ▸ P.V.C apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Clear brown odourless liquid; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	1.185

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Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	6.8	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	100	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	>93.3	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Negligible	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Inhalation of chlorophenoxy pesticide dusts or mist may produce a sore throat and burning sensations in the nasopharynx region and chest, coughing, lachrymation, rhinitis, dizziness and ataxia. Toxic effects may result following absorption from the lungs.
Ingestion	<p>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.</p> <p>Chlorophenoxy compounds may cause irritation of the mouth, throat, and gastrointestinal tract, nausea, vomiting, chest and abdominal pain, and diarrhea. Ingestion of very large doses may produce metabolic acidosis, fever or subnormal temperature, hyperventilation, hypotension, vasodilation, flushing, sweating, cardiac arrhythmias, tachycardia, lethargy, weakness, intercostal paralysis, renal and hepatic disorders, myotonia, coma, and convulsions. Skeletal muscle damage may produce muscle twitching, aching and elevated serum enzymes and myoglobin in both blood and urine. Circulatory collapse may be fatal.</p> <p>Acute exposure to 2,4-dichlorophenoxyacetic acid (2,4-D) and its derivatives and analogues may produce headache, dizziness, nausea, vomiting, raised temperature, low blood pressure, leucocytotoxic heart and liver injury and convulsions.</p> <p>All animal species tested seem to react similarly and there is only a minor difference in potency between various salts and esters of 2,4-D either as pure chemicals or as commercial preparations although the free acid exhibits a somewhat higher toxicity. In several species systemic intoxication after massive doses produces ventricular fibrillation or, if death is delayed, motor disturbances. A disinclination to move progresses to rigidity of skeletal muscles (myotonia) and ataxia (involuntary muscle movement). Severe cases show progressive apathy, depression, muscle weakness of the hind limbs, periodic clonic spasms and coma. Subacute poisonings are characterised by anorexia, eye and nose irritation, and possible epistaxis or bleeding from the mouth. Clinical reports of poisonings are rare although protracted peripheral neuropathies with myopathy appear to be characteristic. Significant cumulative toxicity does not occur with 2,4-D and most of its congeners are not metabolised and do not accumulate in body fat or in the food chain. Urinary excretion is slow with a plasma half-life of about 33 hours.</p>
Skin Contact	<p>Skin contact with the material may be harmful; systemic effects may result following absorption.</p> <p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period.</p>

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	<p>Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <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> <p>2,4-D and its derivatives all penetrate intact skin of laboratory rats and man. Subacute application of 2,4-D esters and of the dimethylamine salt to rabbit skin produced only local irritation due probably to the carrier vehicle (oil). Percutaneous absorption has produced severe peripheral neuropathy in elderly patients exposed to spilled 2,4-D ester, fatigue, nausea, vomiting, anorexia, diarrhoea, swelling and aching of the extremities and muscle fasciculations progressing over a period of days to pain, paraesthesias, and severe limb paralysis. Disability was protracted and continued for several years.</p>
Eye	<p>When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.</p> <p>Corneal injury resulting from 2,4-D exposure may be slow to heal.</p>
Chronic	<p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.</p> <p>Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers</p> <p>Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.</p> <p>Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.</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 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</p> <p>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.</p> <p>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.</p> <p>Repeated overexposure to phenoxy herbicides may cause liver, kidney, gastrointestinal and muscular effects.</p> <p>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.</p> <p>The no-observed effect level (NOAEL) in hamsters was 2 mg/kg 2,4,5-T</p> <p>Clinical symptoms and signs of intoxication following occupational exposure to pyridine, its homologues and derivatives include gastrointestinal disturbance with diarrhoea, abdominal pain and nausea, weakness, headache, insomnia and nervousness..Data indicate that piperidine, pyridine, methyl and alkyl derivatives of pyridine (picolines, lutidines collidines), nicotinonitrile and</p>

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picolinonitrile are slightly to moderately toxic following acute exposures

The available data support the conclusion that the pyridines possess similar human health-related data, and in particular, target organs appear to be the liver and the male reproductive tract.,

The weight-of-evidence suggests that Pyridine and Pyridine Derivatives Category chemicals are not mutagenic. This conclusion is supported by a number of in vivo mutagenicity assays and carcinogenicity studies with negative results for pyridine. Reproductive screening evaluations using several repeated dose toxicity studies indicates that piperidine, pyridine and nicotinonitrile may be male reproductive toxicants.

Exposures less than those which produce overt clinical signs may produce varying levels of liver damage with central lobular fatty degeneration, congestion and cellular infiltration; repeated low level exposures may produce cirrhosis. The kidney is less sensitive to pyridine-induced damage than is the liver. Pyridine, like primidone, phenobarbital and oxazepam induces liver neoplasms in mice, but not in rats, even though in rats these chemicals cause a spectrum of toxic liver lesions. The mouse, an animal with a high background rate of liver neoplasms, is particularly sensitive to the development of malignant liver neoplasms following chemical exposure. There is equivocal evidence (1) that pyridine is carcinogenic in male F344/N rats (based on an increased incidence of renal tubule neoplasms), in female rats of the same species (based on increases in mononuclear cell leukaemia), in male Wistar rats (based on an increased incidence of mono- nuclear cell leukaemia), and clear evidence of carcinogenicity (1) in male and female B6C3F1 mice (based on increased incidences of malignant hepatocellular neoplasms). 1: National Toxicology Program Technical Report Series No. 470, March 2000

AC Pulverise Herbicide	TOXICITY	IRRITATION
	Inhalation (Rat) LC50: >2000 mg/m ³ [2]	Not Available
	Oral (Rat) LD50: >1500 mg/kg[2]	
2,4-dichlorophenoxyacetic acid triisopropanolamine salt	TOXICITY	IRRITATION
	Not Available	Not Available
picloram, triisopropanolamine salt	TOXICITY	IRRITATION
	Not Available	Not Available
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	

2,4-DICHLOROPHENOXYACETIC ACID TRIISOPROPANOLAMINE SALT	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p> <p>Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.</p> <p>For chlorophenoxy pesticides:</p> <p>551chlph</p> <p>WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.</p> <p>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</p> <p>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 hepatotoxicants, 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</p>

compound.

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

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

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

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

Recent findings from Seveso indicate that the biological effects of low level exposure (BELLEs), experienced by a cohort located at a great distance from the plant, may be hormetic, i.e. may be protective AGAINST the development of cancer. The PHAHs do not appear to be genotoxic - they do not alter the integrity of DNA. This contrasts with the effects of the many polycyclic aromatic hydrocarbons (PAHs) (or more properly, their reactive metabolites). TCDD induces carcinogenic effects in the laboratory in all species, strains and sexes tested. These effects are dose-related and occur in many organs. Exposures as low as 0.001 ug/kg body weight/day produce carcinoma. Several studies implicate PCBs in the development of liver cancer in workers as well as multi-site cancers in animals. The second major group of PHAH consists of the non-planar PCB congeners which possess two or more ortho-substituted halogens. These have been shown to produce neurotoxic effects which are thought to reduce the concentration of the brain neurotransmitter, dopamine, by inhibiting certain enzyme-mediated processes. The specific effect elicited by both classes of PHAH seems to depend on the as much on the developmental status of the organism at the time of the exposure as on the level of exposure over a lifetime.

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

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

PICLORAM, TRISOPROPANOLAMINE SALT

For picloram:

Acute toxicity: Picloram is slightly to practically nontoxic via ingestion, with reported oral LD50 values of greater than 5000 mg/kg to 8200 mg/kg in rats, 2000 to 4000 mg/kg in mice, and approximately 2000 mg/kg in rabbits. The reported dermal LD50 in rabbits is greater than 4000 mg/kg, a level which produced no mortality or toxic signs. This indicates slight toxicity via the dermal route as well. Technical picloram is reported to cause no skin and moderate eye irritation in the rabbit, and to cause no skin sensitisation in the guinea pig. Some formulations have caused mild or slight skin irritation and skin sensitization in test animals. The technical grade is moderately toxic by inhalation, with a reported 4-hour inhalation LC50 of greater than 0.35 mg/L. Formulated products may show a lesser toxicity via this route. There is no documented history of human intoxication by picloram, so symptoms of acute exposure are difficult to characterise.

Chronic toxicity: Male mice receiving picloram at dietary doses of 1000 to 2000 mg/kg/day over 32 days showed no clinical signs of toxicity nor changes in blood chemistry, but females did show decreased body weight and increased liver weights. Liver effects were also seen in rats at very high doses of 3000 mg/kg/day over an exposure period of 90 days, and above 225 mg/kg/day for 90 days. Dogs, sheep, and beef cattle fed low levels of picloram for a month experienced no toxic effects. The ester and trisopropanolamine salt showed low toxicity in animal tests. Picloram may show additive effects if mixed with other herbicides such as 2,4-D.

Reproductive effects: In multi-generational studies, pregnant rats exposed during critical periods of gestation to doses of about 180 mg/kg/day of picloram showed no changes in fertility. The fertility of pregnant mice fed 15 mg/kg/day for 4 days before and 14 days after mating was not adversely affected. Other studies showed no effects on fertility or fecundity in rats at doses as high as 1000 mg/kg/day. Picloram does not appear to cause reproductive toxicity.

Teratogenic effects: No teratogenic effects were seen in the offspring of pregnant rats exposed during gestation to 400

AC Pulverise Herbicide

mg/kg/day of the acid or potassium salt, or to 1000 mg/kg/day of the ester or other salt [58]. At 2000 mg/kg/day, maternal toxicity was noted but did not induce malformation in the pups. It appears that picloram is not teratogenic.

Mutagenic effects: One test has shown that picloram is mutagenic (to the bacterium *Saccharomyces cerevisiae*) and another test has shown that it is not mutagenic (Ames test). In tests for unscheduled DNA synthesis and structural chromosome aberrations, the results were also negative. These data suggest that picloram is either nonmutagenic or weakly mutagenic.

Carcinogenic effects: Mice fed average doses of 18 mg/kg/day or 30 mg/kg/day for 80 weeks and observed for another 10 weeks did not display any carcinogenic effects. Male rats fed 17.5 or about 40 mg/kg/day for 80 weeks and observed for 33 weeks showed no carcinogenicity, but females developed benign liver tumor nodules. Other tests have indicated an increased incidence of cancer among animals treated with picloram, but these data are difficult to interpret due to possible interference of hexachlorobenzene contaminants. These data suggest that picloram is noncarcinogenic or weakly carcinogenic.

Organ toxicity: Animal studies show the target organs for picloram to be the liver and kidneys.

Fate in humans and animals: Picloram was rapidly absorbed through the gastrointestinal tract in studies using human volunteers, and was excreted unchanged in the urine. Half of the product was excreted within a day or so. Skin absorption is minimal. Rats showed similar results, with administered doses excreted virtually unchanged in urine and faeces within 48 hours. Picloram does not accumulate in fat. No measurable residues were found in milk from cows fed small amounts of the herbicide in their diets. At higher levels of exposure, milk levels of picloram were low (0.05 to 0.29 ppm) and declined rapidly upon withdrawal of picloram from the diet.

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

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis.

Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

**2,4-DICHLOROPHOXYACETIC
ACID TRIISOPROPANOLAMINE
SALT & PICLORAM,
TRIISOPROPANOLAMINE SALT**

No significant acute toxicological data identified in literature search.

Acute Toxicity	✓	Carcinogenicity	✗
Skin Irritation/Corrosion	✓	Reproductivity	✗
Serious Eye Damage/Irritation	✓	STOT - Single Exposure	✓
Respiratory or Skin sensitisation	✓	STOT - Repeated Exposure	✗
Mutagenicity	✗	Aspiration Hazard	✗

Legend: ✗ – Data either not available or does not fill the criteria for classification
✓ – Data available to make classification

SECTION 12 Ecological information

Toxicity

AC Pulverise Herbicide	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
2,4-dichlorophenoxyacetic acid triisopropanolamine salt	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
picloram, triisopropanolamine salt	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	768h	Fish	7.19mg/L	4
	LC50	96h	Fish	10.2-632mg/l	4
	EC50	48h	Crustacea	120-1712mg/L	4
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					

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
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Continued...

AC Pulverise Herbicide

Ingredient	Persistence: Water/Soil	Persistence: Air
	No Data available for all ingredients	No Data available for all ingredients

Bioaccumulative potential

Ingredient	Bioaccumulation
	No Data available for all ingredients

Mobility in soil

Ingredient	Mobility
	No Data available for all ingredients

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. Recycle wherever possible. Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible material). Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.
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SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

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

Product name	Group
2,4-dichlorophenoxyacetic acid triisopropanolamine salt	Not Available
picloram, triisopropanolamine salt	Not Available

Transport in bulk in accordance with the IGC Code

Product name	Ship Type
2,4-dichlorophenoxyacetic acid triisopropanolamine salt	Not Available
picloram, triisopropanolamine salt	Not Available

SECTION 15 Regulatory information

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

2,4-dichlorophenoxyacetic acid triisopropanolamine salt is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

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

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

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

picloram, triisopropanolamine salt is found on the following regulatory lists

Australia Chemicals with non-industrial uses removed from the Australian Inventory of Chemical Substances (old Inventory)

National Inventory Status

National Inventory	Status
Australia - AIC / Australia Non-Industrial Use	No (2,4-dichlorophenoxyacetic acid triisopropanolamine salt)
Canada - DSL	No (2,4-dichlorophenoxyacetic acid triisopropanolamine salt; picloram, triisopropanolamine salt)
Canada - NDSL	No (2,4-dichlorophenoxyacetic acid triisopropanolamine salt; picloram, triisopropanolamine salt)
China - IECSC	No (2,4-dichlorophenoxyacetic acid triisopropanolamine salt; picloram, triisopropanolamine salt)
Europe - EINEC / ELINCS / NLP	No (2,4-dichlorophenoxyacetic acid triisopropanolamine salt)
Japan - ENCS	No (2,4-dichlorophenoxyacetic acid triisopropanolamine salt; picloram, triisopropanolamine salt)
Korea - KECI	No (2,4-dichlorophenoxyacetic acid triisopropanolamine salt; picloram, triisopropanolamine salt)
New Zealand - NZIoC	No (2,4-dichlorophenoxyacetic acid triisopropanolamine salt)
Philippines - PICCS	No (2,4-dichlorophenoxyacetic acid triisopropanolamine salt; picloram, triisopropanolamine salt)
USA - TSCA	No (2,4-dichlorophenoxyacetic acid triisopropanolamine salt; picloram, triisopropanolamine salt)
Taiwan - TCSI	No (2,4-dichlorophenoxyacetic acid triisopropanolamine salt; picloram, triisopropanolamine salt)
Mexico - INSQ	No (2,4-dichlorophenoxyacetic acid triisopropanolamine salt; picloram, triisopropanolamine salt)
Vietnam - NCI	No (2,4-dichlorophenoxyacetic acid triisopropanolamine salt; picloram, triisopropanolamine salt)
Russia - FBEPH	No (2,4-dichlorophenoxyacetic acid triisopropanolamine salt; picloram, triisopropanolamine salt)
Legend:	<p>Yes = All CAS declared ingredients are on the inventory</p> <p>No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.</p>

SECTION 16 Other information

Revision Date	23/12/2022
Initial Date	14/10/2014

SDS Version Summary

Version	Date of Update	Sections Updated
4.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification
5.1	23/12/2022	Classification review due to GHS Revision change.

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

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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