Apparent Ramjet 75-D Herbicide

AIRR Apparent Pty Ltd.

Chemwatch Hazard Alert Code: **3**

Chemwatch: **9656-49**
Version No: **2.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Initial Date: **05/11/2026**
Revision Date: **05/11/2026**
Print Date: **23/11/2026**
L.GHS.AUS.EN.E

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

Product Identifier

Product name	Apparent Ramjet 75-D Herbicide
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains 2,4-dichlorophenoxyacetic acid triisopropanolamine salt and picloram, triisopropanolamine salt)
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	A liquid broadleaf agricultural herbicide. Use according to manufacturer's directions.

Details of the manufacturer or importer of the safety data sheet

Registered company name	**AIRR Apparent Pty Ltd.**
Address	15/16 Princes Street, Newport NSW 2106 Australia
Telephone	+61 3 5820 8400
Fax	Not Available
Website	www.apparent.com.au
Email	enquiries@apparentag.com.au

Emergency telephone number

Association / Organisation	**AIRR Apparent Pty Ltd.**
Emergency telephone number(s)	1800 033 111 (24 Hours)
Other emergency telephone number(s)	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	S6
Classification [1]	Acute Toxicity (Oral) Category 4, Acute Toxicity (Dermal) Category 4, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Acute Toxicity (Inhalation) Category 4, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Carcinogenicity Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 2
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)	
Signal word	**Danger**

Hazard statement(s)

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H302	Harmful if swallowed.
H312	Harmful in contact with skin.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H318	Causes serious eye damage.
H332	Harmful if inhaled.
H335	May cause respiratory irritation.
H351	Suspected of causing cancer.
H411	Toxic 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.
P202	Do not handle until all safety precautions have been read and understood.
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.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
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.
P391	Collect spillage.
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.

No further product hazard information.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
32341-80-3	30	2,4-dichlorophenoxyacetic acid triisopropanolamine salt
6753-47-5	7.5	picloram, triisopropanolamine salt
Not Available	balance	Ingredients determined not to be hazardous
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available***	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with 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. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs:

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	▸ 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	▸ 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	▸ **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. **Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:** ▸ **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. **NOTE:** Wear a protective glove when inducing vomiting by mechanical means.

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. 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.
- 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 substance or mixture

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

Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ Avoid spraying water onto liquid pools. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	▸ 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. Combustion products include: ▸ carbon dioxide (CO_2)

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	hydrogen chloride phosgene nitrogen oxides (NOx) ▸ other pyrolysis products typical of burning organic material.
HAZCHEM	•3Z

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. ▸ 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	Environmental hazard - contain spillage. Moderate hazard. ▸ 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	▸ Avoid skin contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ Avoid smoking, naked lights or ignition sources. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ 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	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ 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	▸ Metal can or drum ▸ Packaging as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ Avoid strong bases. ▸ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

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MATERIAL DATA

For 2,4-dichlorophenoxyacetic acid (2,4-D):
Evidence for skin absorption and chronic feeding studies in rats and mice necessitate a review of the recommended TLV-TWA:
Occupational exposure will occur for workers involved in the handling, transfer, mixing and spraying of the substance. At a spraying rate of 315-630 g/ha (water-soluble formulation), the calculated inhalation exposure of applicators accounted for less than 2% (median dose 0.2 ug/kg body weight) of the potential cumulative exposure while deposition on the hands accounted for 80-90% (median dose of 120 ug/kg body weight) of the cumulative exposure.

Workers using 2,4-D (the acid) may, on average, absorb 0.1 mg 2,4-D/kg body weight per day. A statistical estimate by NIOSH showed that field spraymen are exposed to air concentrations of 0.001 mg/m3 (TWA). Urinalysis of six volunteer workers (wearing full line protective clothing) involved in mixing and loading 2,4-D ester solutions into aircraft and in guiding the spray aircraft showed a maximum excretion level of 22.2 ug/kg body weight/day. Analyses of the breathing zone air of pilots and ground crew during a three day aerial spray operation showed a mean 2,4-D concentration of 0.002-0.1 mg/m3.
The urinary excretion rate of 2,4-D in 45 commercial lawn care specialists who had been spraying the herbicide for a period of at least 3 weeks ranged from 0.4-6.3 ug/kg body weight/day. Thirty nine children of 197 children living near or away from possible source of contamination had detectable levels of 2,4-D (detection limit 1 ppb) in their urine at a maximum concentration of 9 ppb.

For picloram:
In view of the low irritancy potential of picloram it can be assumed the toxicity due to inhalation exposure will be similar to that due to ingestion. Picloram has low acute and chronic toxicity with hepatic and renal changes recorded, in rats, at doses of 225 mg/kg/day.
The carcinogenic potential in rats remains controversial.
Exposure at or below the TLV-TWA is thought to minimise the risk of systemic effects involving the liver and kidney.

Exposure controls

Appropriate engineering controls

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.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
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

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	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 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	‣ Overalls. ‣ P.V.C apron. ‣ Barrier cream. ‣ Skin cleansing cream. ‣ Eye wash unit.

Respiratory protection

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

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	A-AUS / Class1	-
up to 50	1000	-	A-AUS / Class 1
up to 50	5000	Airline *	-
up to 100	5000	-	A-2
up to 100	10000	-	A-3
100+			Airline**

* - Continuous Flow ** - Continuous-flow or positive pressure demand
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	Dark brown liquid.

Physical state	Liquid	**Relative density (Water = 1)**	1.1-1.3
Odour	No Odour	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	6-7.5	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	0	**Viscosity (mm2/s)**	Not Available

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Initial boiling point and boiling range (°C)	100	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available
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	‣ 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	There is sufficient evidence to classify this material as acutely toxic.
b) Skin Irritation/Corrosion	There is sufficient evidence to classify this material as skin corrosive or irritating.
c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	There is sufficient evidence to classify this material as sensitising to skin or the respiratory system
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	There is sufficient evidence to classify this material as carcinogenic
g) Reproductivity	Based on available data, the classification criteria are not met.
h) STOT - Single Exposure	There is sufficient evidence to classify this material as toxic to specific organs through single exposure
i) STOT - Repeated Exposure	Based on available data, the classification criteria are not met.
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial 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. 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	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. Rats given lethal doses (approximately 1 gm/kg) picloram, exhibited depression, prostration, ataxia, tremours and convulsions preceding death. The 7-day no-observed-adverse effect level (NOAEL) was 400 mg/kg/day picloram in female beagles. The 14-day dog oral NOAEL was 200 mg/kg/day. The lowest-observed-adverse-effect level (LOAEL) based on increased liver weight, was 2700 mg/kg/day in mice fed picloram for 32-days. The subchronic 13-week NOAEL in rats was 50 mg/kg/day. During a 90-day feeding study rats receiving 225 mg/kg/day picloram showed moderate changes in the liver and kidneys and female rats showed a slight reduction in body weight. Renal and hepatic lesions were seen in a 90-day drinking water study with male and female rats - severity was dose-dependant. No adverse effects were seen amongst 6 human volunteers ingesting picloram dissolved in grape juice at 0.5 to 5 mg/kg. Seventy-six percent of the dose was excreted in the urine within 6-hours (half-life 2.9 hours); the remainder was eliminated with an average half-life of 27 hours. 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. 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.

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	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.
Skin Contact	Skin contact with the material may be harmful; systemic effects may result following absorption. Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Open cuts, abraded or irritated skin should not be exposed to this material 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. 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.
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. Corneal injury resulting from 2,4-D exposure may be slow to heal.
Chronic	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. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. 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. 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. Substances than can cuase 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 Wherever it is reasonably practicable, exposure to substances that can cuase 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. 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. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Repeated excessive exposure to high amounts of picloram may cause liver effects. The results of a 2-year feeding study in rats fed picloram at 20-200 mg/kg/day included the development of centrolobular hepatocellular hypertrophy and increased liver weights. The chronic rat NOAEL was 20 mg/kg/day. Beagles given 150 mg/kg/day picloram showed no treatment related changes in body-weight gain, food consumption, behaviour, mortality, haematological and clinical blood chemistry, urinalysis or in histopathologic parameters. Female rats fed up to 723 mg/kg/day picloram for 2-years showed statistically equivocal evidence of increased incidence of benign nodules in the liver; male rats showed a "negative" carcinogenic response. In a life-time study using mice and rats an increased incidence in pituitary and adrenal neoplasia occurred in male and female rats given 7437 and 14875 ppm picloram. In male mice fed 5062 ppm there was an increased incidence of tumours of the spleen. 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. 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 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.

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	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

Apparent Ramjet 75-D Herbicide	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
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***

PICLORAM, TRIISOPROPANOLAMINE 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 triisopropanolamine 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 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 on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.
Apparent Ramjet 75-D Herbicide & 2,4-DICHLOROPHENOXYACETIC ACID TRIISOPROPANOLAMINE SALT	The following information refers to contact allergens as a group and may not be specific to this product. 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. Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production. For 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 hepatoxicants, reproductive toxicants, immunotoxicants and procarcinogens) by interaction with a cytostolic 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 true. This, in part,

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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 thyroxin (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

Apparent Ramjet 75-D Herbicide & 2,4-DICHLOROPHENOXYACETIC 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

Apparent Ramjet 75-D 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
EC50	48h	Crustacea	120-1712mg/L	4

Continued...

	NOEC(ECx)	768h	Fish	7.19mg/L	4
	LC50	96h	Fish	10.2-632mg/L	4

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

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 2,4-dichlorophenoxyacetic acid (2,4-D):
2,4-dichlorophenoxyacetic acid is a hydrophilic compound that tends to remain in water and not adsorb onto soil. Volatilisation into air is considered negligible for most formulations except for the high volatile esters. In water, the acid hydrolyses to the 2,4-D anion and is subject to microbial degradation as the major route of degradation with rates increasing in soils that have been previously treated with the herbicide. 2,4-D is not considered persistent in soil since it tends not to adsorb onto soil unless high levels of organic carbon are present. The compound is relatively mobile, but due to rates and types of applications (usually foliar) along with the relatively high rate of microbial degradation 2,4-D is not likely to leach into subsurface flow. 2,4-D has little tendency to bioconcentrate in animal tissue and is usually excreted unmetabolised via urine except for some of the low-volatile esters which will bioaccumulate in the absence of metabolisation . Some formulations, especially the butyl ester, can be highly toxic to fish. In humans, 2,4-D acid is largely excreted unchanged with no evidence of accumulation in tissues or metabolisation to reactive metabolites.
Environmental fate:
Air Volatilisation plays only a minor role in the breakdown and dissipation of the 2,4-D acid due to its low vapour pressure of 1. x 10-7 mmHg. Further, there is little movement of 2,4-D acid through the air/water barrier, the barrier between atmosphere and surface water or soil moisture, into air due to a low Henry s Law Constant of 1.76 x10-12. If proper application techniques are not used, the high volatile esters may be prone to spray drift causing toxic effects in nearby crops . With the exception of the high volatile ester formulations, the small amount of 2,4-D that gets into the air is subject to photooxidation by reaction with hydroxyl radicals with an estimated half life of 1 day or dissolves into water droplets and is transported back to the earth s surface via wet deposition. The low volatile ester and amine formulations are used in forests so drift from those applications is relatively negligible.
Water: In the aqueous environment, 2,4-D is most commonly found as the free anion . The amine salt formulations dissociate to the anion and ester formulations hydrolyze to the anion, usually within one day . The rate of hydrolysis is pH dependent, with the hydrolysis halflife at pH 9 much shorter than the half-life at pH 6 . Therefore, the persistence of the 2,4-D anion is of primary concern.
Decomposition of the anion appears to result from microbial or photodegradation, with photolysis playing a minor role if microbial degradation is rapid. Both aerobic and anaerobic degradation are possible, although anaerobic degradation is relatively slow with a half-life of 312 days. In water, 2,4-D will biodegrade at a rate dependent upon the level of nutrients present, temperature, availability of oxygen, and whether or not the water has been previously contaminated with 2,4-D or other phenoxyacetic acids . Microbial degradation is a possible route for the breakdown of 2,4-D, but it is very dependent on the characteristics of the water. Microbial metabolism has been found to be biphasic with the first phase being a lag phase where the microbes acclimate to the test compound and the second phase being rapid metabolism with a total half-life of 15.0 days for both phases. 2-Chlorohydroquinone (1,4-dihydroxy 2-chlorophenol) is the major metabolite and 2,4-dichlorophenol and carbon dioxide as minor metabolites. Anaerobic aquatic metabolism is a minor pathway with a half-life of 312 days. Major metabolites are 2,4-DCP and carbon dioxide, with 4-chlorophenol and 2,4-dichloroanisol (2,4-DCA) as minor metabolites .
2,4-D has an aqueous photolysis half-life of 13.0 days at 25 C at the surface of distilled water. In natural surface waters photodecomposition is not expected to be significant due to weaker ultraviolet radiation of natural sunlight and the presence of suspended and organic matter which reduces the effects of solar radiation . The half-life is expected to increase with depth due to reduction in penetration. The major photodegradation product is 1,2,4-benzenetriol . Additional studies showed that 2,4-D is susceptible to photodegradation resulting in the formation of carbon dioxide, 1,2,4-benzenetriol, 2,4-dichlorophenol and then proceeds to a secondary photolysis forming humic acids.
For high volatile ester formulations of 2,4-D, volatilisation may play a larger role for removal from water than hydrolysis. In neutral and acidic waters conversion from ester to anion via hydrolysis is slower than the potential rate of vapourisation.
Soil: Ester hydrolysis decreases with decreasing soil moisture and with increasing molecular weight of the alcohol portion of the ester. Water solubility and the soil adsorption coefficient (Koc) indicate the potential mobility of a chemical in soil..2,4-D has a moderate persistence in soil with a field dissipation half-life of 59.3 days, aerobic half-life of 66 days, and a hydrolysis half-life of 39 days It was found that high organic matter and free iron in soils favoured the adsorption of 2,4-D, while high pH, large surface area, and phyllosilicates as essential clay components decreased adsorption . 2,4-D is in nonionic form at pH less than 6 and is in anionic form at pH greater than 6. In slightly acidic soils, 2,4-D will be adsorbed at pH less than 6 but will not be adsorbed as much if in the anionic form because the negative charges of the soil and chemical repel each other.
Microbial degradation is considered to be the major route in the breakdown of 2,4-D in soil. The rate of microbial degradation is dependent upon the water potential, depth and temperature of the soil..Sandy loam soil containing 2,4-D degrading single-celled bacteria, filamentous bacteria (actinomycetes), and fungi, had the lowest degradation rates. Dry soil conditions contribute to the inhibition of 2,4-D mineralisation by restricting solute mobility, reducing the herbicide degrading activity of organisms, and suppressing the 2,4-D degrading microorganism populations. The rate of microbial degradation is also dependent on soil depth and temperature, with rates of degradation decreasing with increased depths and lower temperatures.
Degradation in soil is affected by the rate of adsorption-desorption of 2,4-D onto soil particles which bind the chemical, making it unavailable for microbial degradation. As soil organic carbon content increased to 12 % the rate of adsorption increased correlating to a decreased rate of degradation due to low concentrations of 2,4-D available for microbial degradation. When the organic carbon content was more than 12% there was an increase in the rate of both adsorption and degradation. The enhanced degradation of 2,4-D was attributed to the increased biological activity of the soil and the decreased 2,4-D-induced inhibitory effect on microbial activity.
Photodecomposition on soil surfaces plays a very minor role in the breakdown of 2,4-D and only occurs on the upper surface of the soil. The potential major soil photodegradation products are 2,4-dichlorophenol, 1,2,4-benzenetriol, and 2-chlorohydroquinone.
The high water solubility of 4.46x10+4 ppm and low soil adsorption coefficient of 0.067-1.1 cm3/g for the 2,4-D free acid suggest that it has a high potential to leach in soil. The principle means of movement would probably be with percolating water, while diffusion is important only for transport over small distances. The adsorption capacity of a given soil affects the potential for leaching of 2,4-D; in soils that promote adsorption, the leaching potential is lower**.**
Biota: 2,4-D formulations having high lipid solubility (such as the esters) rapidly penetrate the cuticle. Salt formulations are most readily absorbed through the roots, while esters are more readily absorbed through foliage. Foliar applied 2,4-D is easily translocated in the phloem and carried with material from photosynthesising leaves to growth sites where it accumulates. Translocation of 2,4-D upward by roots takes place primarily in the transpiration stream of the xylem.
2,4-D mimics the effects of auxins (plant growth regulators) and is found to persist in plant tissues longer than the natural hormone. Ester formulations do not function as growth regulators until they are converted to the acid form, usually within ½ hour after application. The immediate cause of plant death is due to abnormal metabolism of nucleic acids.
Bioconcentration and bioaccumulation: The bioconcentration factor (BCF, a ratio of the test substance concentration in fish to the concentration in water) for 2,4-D ranges from an estimated 7 for the acid to an estimated 37,500 for the isooctyl ester to an estimated 0.1 to 0.47 for the dimethylamine salt . 2,4-D when applied as the acid shows little tendency to bioconcentrate in fish while if applied as the isooctyl ester it is expected to bioconcentrate in the absence of metabolisation. In fish, accumulated 2,4-D is rapidly broken down into hydrocarbon fragments which are utilized by the fish for synthesis of normal body tissue and/or eliminated . In trout 72 ± 4% of 2,4-D is excreted via urine as the unchanged acid within 8 hours after exposure with a half-life of 2.4 hours
Ecotoxicity:
Subacute toxicity tests showed that 2,4-D induced changes in the enzyme activities and morphological changes in the gills, liver, and kidneys of adult fish . A toxicity study done on Salmonids in southeast Alaska showed that the butyl ester was the most toxic of the ester formulations and the isooctyl ester was the least toxic. 100% mortality occurred for pink, chum, and coho fry exposed to 1 ppm 2,4-D butyl ester and sockeye smolts, dolly varden, rainbow and Oregon coho fingerlings exposed to 5 ppm 2,4-D butyl ester . The no-effect level was less than 1 ppm, but not more precisely described. The same study also found the no-effect level for isooctyl ester to range from 1ppm to 10 ppm for all the same fish species except for pink salmon fry and sockeye smolts which showed 40 and 6.7% mortality at 1ppm, respectively. The pure acid form of 2,4-D at 1ppm only showed high mortality in the pink fry
For picloram:
log Kow : 0.3
Koc : 5.87-25.2
Half-life (hr) air : 292.9
Half-life (hr) H2O surface water : 55.2-991.2
Half-life (hr) soil : 1320-2400
Henry's atm m3 /mol: 4.05E-11
Vapour pressure 0.082 mPa (35 C)
Partition coefficient: 0.1461
Adsorption coefficient 16
Environmental fate:

Continued...

Breakdown in soil and groundwater: Picloram is moderately to highly persistent in the soil environment, with reported field half-lives from 20 to 300 days and an estimated average of 90 days . Photodegradation is significant only on the soil surface and volatilisation is practically nil . Degraded slowly by soil microorganisms. DT50 30-330 d. Degradation by microorganisms is mainly aerobic, and dependent upon application rates . Increasing soil organic matter increases the sorption of picloram and increases the soil residence time . Picloram is poorly bound to soils, although it is bound better by soils with higher proportions of soil organic matter . It is soluble in water, and therefore may be mobile . These properties, combined with its persistence, mean it may pose a risk of groundwater contamination.
Breakdown in water: In laboratory studies, sunlight readily broke down picloram in water, with a half-life of 2.6 days . Herbicide levels in farm ponds were 1 mg/L directly following spraying, and decreased to 0.01 mg/L within 100 days, primarily due to dilution and the action of sunlight.
Breakdown in vegetation: Picloram is readily absorbed by plant roots, less so by the foliage, and is readily translocated throughout plants. It remains stable and intact in plants Photodecomposition occurs on plant surfaces, possibly with cleavage of the pyridine ring
Bioconcentration: Picloram is not expected to accumulate in aquatic organisms; the measured bioconcentration factor in bluegill sunfish was less than 0.54
Ecotoxicity:
Bird LD50: duck, pheasant, quail >2000->5000 mg/kg (slightly to practically non-toxic)
Fish LC50 (96 h): rainbow trout 19.3 mg/l; bluegill sunfish 14.5 mg/l; fathead minnow 55 mg/l (slightly to moderately toxic).
Fish LC50 (96 h) - isooctyl ester: rainbow trout 4 mg/l; channel catfish 1.4 mg/l (highly toxic)
Most salts of picloram are of similar or lesser toxicity but the isooctyl ester may be highly toxic.
Daphnia magna LC50 948 h): 50 mg/l (moderately toxic)
Other aquatic invertebrates: 10-68 mg/l
A persistent herbicide (or contains the herbicide)
Certain herbicides can persist on vegetation and in the soil for months or years and these products are called "persistent herbicides". Persistent herbicides are a narrow range of herbicides used to kill broad leaf weeds and thistle that compete with grasses and grain crops. They are "persistent" because they will not be killed by the high temperatures in thermophilic composting and may take over 2 years or more to fully decay
The class of herbicides from the picolinic acid family is of the greatest concern. These chemicals are marketed for use in hayfields, horse pastures, golf courses, roadways, grain crops, and lawns, to kill unwanted broad-leaf weeds. These herbicides do not normally impact grasses and plants like corn, wheat, and oats.
Herbicides vary in their potential to persist in soil. Herbicide families that have persistent members include triazines, uracils, phenylureas, sulfonylureas, dinitroanilines, isoxazolidiones, imidazolinones, and certain plant growth regulators belonging to the pyridine family.
The length of time a herbicide remains active in soil is called "soil persistence," or "soil residual life". For some herbicides, there may be a fine line between controlling weeds for the entire growing season and then planting a sensitive rotation crop. Anything that affects the disappearance or breakdown of herbicides affects persistence.
Persistent herbicides are generally colourless and odorless. Scientific studies reveal that pass unaltered during animal digestion (including microbial digestion) when used at labelled rates. In fact, animal digestion tends to strengthen the impact of these chemicals through concentration because the animal processes the food but passes most of the chemical as a waste.
Persistent herbicide may will kill a tomato plant at a concentration of one part per billion, and impact many other garden plants as well. Plant families sensitive to persistent herbicides include:
· The nitrogen-fixing, legume family of plants such as peas, beans, lentils, and clover
· The nightshade, solanaceous family of plants such as tomatoes and potatoes
· The sunflower, composites family of plants such as sunflower, petunias, daisies, lettuce, and asters
· The cucumber, cucurbit family of plants such as cucumber, squash, pumpkin, and watermelon
Damaged plants may show on or more of the following symptoms:
Stunted growth: the main growth tip stops growing and the lateral buds begin to grow
· Reduced fruit set
· Cupping of leaves
· Failure of secondary leaves to grow after the seed leaves emerge
· In legumes, compound leaves stay single
Depending on the type of herbicide and the level of concentration in the soil, persistent herbicides can last anywhere from several months to three or more years before completely breaking down into inert compounds. The length of time depends upon a variety of factors, including the type and moisture content of the soil.
The most common feed stocks with persistent herbicide contamination are manures and bedding, but grass clippings and many food items can be contaminated.
Persistent pesticides do not break-down in the composting process. At its most fundamental level, composting is digestion by microorganisms, regardless of whether it is an aerated static pile, anaerobic digestion, etc. Simple composting math demonstrates that one ton of feedstock reduces to approximately half that amount as finished compost.
Persistent herbicides break down much more slowly than the materials in compost, and therefore, most of the chemicals pass into the finished compost. Low toxicity to animals is one often cited benefit of persistent herbicides.
According to the USEPA, persistent herbicides are not harmful to people or animals when these products are used at labelled rates.
DO NOT discharge into sewer or waterways.

Persistence and degradability

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	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ 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. 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. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) 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

Continued...

	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. • **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.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	•3Z

Land transport (ADG)

14.1. **UN number or ID number**	3082
14.2. **UN proper shipping name**	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains 2,4-dichlorophenoxyacetic acid triisopropanolamine salt and picloram, triisopropanolamine salt)
14.3. **Transport hazard class(es)**	Class: 9 Subsidiary Hazard: Not Applicable
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: 274 331 335 375 AU01 Limited quantity: 5 L

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082
are not subject to this Code when transported by road or rail in;
(a) packagings;
(b) IBCs; or
(c) any other receptacle not exceeding 500 kg(L).
- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	3082
14.2. **UN proper shipping name**	Environmentally hazardous substance, liquid, n.o.s. (contains 2,4-dichlorophenoxyacetic acid triisopropanolamine salt and picloram, triisopropanolamine salt)
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 2,4-dichlorophenoxyacetic acid triisopropanolamine salt and picloram, triisopropanolamine salt)
14.3. **Transport hazard class(es)**	IMDG Class: 9

Continued...

	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: 274 335 375 969 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
2,4-dichlorophenoxyacetic acid triisopropanolamine salt	Not Applicable
picloram, triisopropanolamine salt	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

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

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)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / 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)
Colombia - Colombia National Industrial Chemicals Inventory	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)
UAE - Control List (Banned/Restricted Substances)	No (2,4-dichlorophenoxyacetic acid triisopropanolamine salt; picloram, triisopropanolamine salt)
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.***

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SECTION 16 Other information

Revision Date	05/11/2026
Initial Date	05/11/2026

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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end of SDS