

Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide)

Titan Ag

Chemwatch: 25169-95

Version No: 2.1

Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Chemwatch Hazard Alert Code: 3

Initial Date: 30/04/2026

Revision Date: 30/04/2026

Print Date: 03/06/2026

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

Product Identifier

Product name	Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA 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	Agricultural herbicide. Use according to manufacturer's directions.
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Details of the manufacturer or importer of the safety data sheet

Registered company name	Titan Ag
Address	Princes Street Marina, Suite 15/16 Princes Street Newport NSW 2106 Australia
Telephone	+61 2 9999 6655
Fax	+61 2 9999 0483
Website	https://www.titanag.com.au/
Email	titanorders@titanag.com.au

Emergency telephone number

Association / Organisation	Titan Ag
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

COMBUSTIBLE LIQUID, regulated for storage purposes only

Poisons Schedule	S7
Classification ^[1]	Flammable Liquids Category 4, 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, Carcinogenicity Category 2, Reproductive Toxicity Category 1A, Specific Target Organ Toxicity - Repeated Exposure Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	   
Signal word	Danger

Hazard statement(s)

H227	Combustible liquid.
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.
H351	Suspected of causing cancer.
H360D	May damage the unborn child.
H373	May cause damage to organs through prolonged or repeated exposure.
H410	Very toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P202	Do not handle until all safety precautions have been read and understood.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P260	Do not breathe mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
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.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.
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

P403	Store in a well-ventilated place.
P405	Store locked up.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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No further product hazard information.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
29450-45-1	<25	MCPA, 2-ethylhexyl ester
872-50-4	<15	N-methyl-2-pyrrolidone
1689-99-2	<15	bromoxynil octanoate
83164-33-4	<2.5	diflufenican
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.
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Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide)

	▶ 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: ▶ 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

Treat symptomatically.

for dinitrophenols:

Marked fatigue, tremendous thirst, profuse sweating, flushing of the face are all characteristics of intoxication by dinitrophenol derivatives. These may be followed by restlessness, anxiety, excitement which may occasionally lead to convulsions. Rises in body temperature (proportional to dose) may result in severe hyperpyrexia. tachycardia, hyperpnoea, dyspnoea, cyanosis and muscle cramps characterise later symptomatology. Late complications include decreased urine output with albuminuria, casts, pigment, and sometimes blood cells due to toxic nephritis. Jaundice and liver tenderness also develop as a result of toxic hepatitis.

TREATMENT for dinitrophenol intoxications:

- ▶ Gastric lavage with large quantities of 5% sodium bicarbonate solution leaving 1-2 pints in the stomach.
- ▶ Saline cathartics e.g. 15-30 gm sodium or magnesium sulfate in water.
- ▶ Cold packs and alcohol sponges to reduce body temperature. Antipyretic drugs are ineffective here. Cold water enemas have been used. Intensive efforts to correct a dinitrophenol fever are justified. If it can be accomplished, mild hypothermia (rectal temperature between 33.5 and 36 deg. C) is desirable because dinitrophenol appears to lose its metabolic activity at reduced temperature. **[DO NOT give atropine, aspirin, and other salicylates to control hyperthermia, as these agents appear likely to enhance the toxicity of phenolic substances. Aspirin also enhances the uncoupling of oxidative phosphorylation.]**
- ▶ Fluids, orally or intravenously (e.g. glucose in saline, 1000 ml) to correct dehydration and acidosis.
- ▶ Because dinitrophenol is actively transported by the renal organic acid transport processes in some species, a trial of forced diuresis with alkalinisation of the urine is warranted.
- ▶ Oxygen therapy. Artificial ventilation is needed. [Administer oxygen by mask to minimise anoxia].
- ▶ Prophylactic measures in anticipation of kidney and liver insufficiency.

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

Diazepam may be used, if necessary, for the treatment of convulsions, to reduce body heat and control agitation. Be prepared to counter respirator depression and hypotension which may occur following administration of anticonvulsants.

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. Alkalinisation 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.

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Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide)

- ▶ Water spray or fog - Large fires only.

Special hazards arising from the substrate or mixture

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

Fire Fighting	▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ 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 (CO₂) hydrogen chloride phosgene nitrogen oxides (NO_x) ▶ other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.
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	Environmental hazard - contain spillage. ▶ 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	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	Consider storage under inert gas.

Continued...

- ▶ 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	▶ Metal can or drum ▶ Packaging as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Nitrophenols are: ▶ combustible solids which may form explosive mixtures with air when finely divided ▶ strong oxidisers which react violently with reducing agents ▶ reactive with combustible, organic and other easily oxidisable materials ▶ thermally unstable burning in the absence of air causing fast pressure rises; closed containers may explode ▶ able to form shock-sensitive explosive mixtures with chlorine trifluoride ▶ incompatible with strong acids, caustics, aliphatic amines, amides, diethylamine, potassium hydride, potassium hydroxide ▶ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	N-methyl-2-pyrrolidone	1-Methyl-2-pyrrolidone	25 ppm / 103 mg/m3	309 mg/m3 / 75 ppm	Not Available	Not Available
Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits	N-methyl-2-pyrrolidone	1-Methyl-2-pyrrolidone	20 ppm / 80 mg/m3	Not Available	Not Available	Not Available

MATERIAL DATA

for N-methyl-2-pyrrolidone (NMP):
Reports of skin and eye irritation and chronic headaches have been reported in workers exposed to 1-methyl-2-pyrrolidone. The Australian ES is based on a 10-fold uncertainty factor of the no-observable-adverse-effect level (NOAEL) of 24 ppm where adverse respiratory effects were observed in a 4-week inhalation study in rats.
These exposure guidelines have been derived from a screening level of risk assessment and should not be construed as unequivocally safe limits. ORGS represent an 8-hour time-weighted average unless specified otherwise.
CR = Cancer Risk/10000; UF = Uncertainty factor:
TLV believed to be adequate to protect reproductive health:
LOD: Limit of detection
Toxic endpoints have also been identified as:
D = Developmental; R = Reproductive; TC = Transplacental carcinogen
Jankovic J., Drake F.: A Screening Method for Occupational Reproductive
American Industrial Hygiene Association Journal 57: 641-649 (1996)

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. <table><tr><th>Type of Contaminant:</th><th>Air Speed:</th></tr><tr><td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td><td>0.25-0.5 m/s (50-100 f/min.)</td></tr><tr><td>aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)</td><td>0.5-1 m/s (100-200 f/min.)</td></tr><tr><td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td><td>1-2.5 m/s (200-500 f/min.)</td></tr><tr><td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td><td>2.5-10 m/s (500-2000 f/min.)</td></tr></table> Within each range the appropriate value depends on: <table><tr><th>Lower end of the range</th><th>Upper end of the range</th></tr><tr><td>1: Room air currents minimal or favourable to capture</td><td>1: Disturbing room air currents</td></tr><tr><td>2: Contaminants of low toxicity or of nuisance value only.</td><td>2: Contaminants of high toxicity</td></tr><tr><td>3: Intermittent, low production.</td><td>3: High production, heavy use</td></tr></table>	Type of Contaminant:	Air Speed:	solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use
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Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide)

	4: Large hood or large air mass in motion 4: Small hood-local control only Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
Individual protection measures, such as personal protective equipment	
Eye and face protection	▶ Safety glasses with side shields. ▶ Chemical goggles.[AS/NZS 1337.1, EN166 or national equivalent] ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].
Skin protection	See Hand protection below
Hands/feet protection	▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber NOTE: ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential 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.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the: "Forsberg Clothing Performance Index". The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection: Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide)

Material	CPI
BUTYL	A
PE/EVAL/PE	A
NATURAL RUBBER	B

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required. Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	AK-AUS	-	AK-PAPR-AUS / Class 1

Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide)

PVA	B
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* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

up to 50 x ES	-	AK-AUS / Class 1	-
up to 100 x ES	-	AK-2	AK-PAPR-2 ^

^ - Full-face

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

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Amber liquid.		
Physical state	Liquid	Relative density (Water = 1)	Not Available
Odour	Characteristic	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	>65	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Combustible.	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	Partly 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

Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide)

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	There is sufficient evidence to classify this material as toxic to reproductivity
h) STOT - Single Exposure	Based on available data, the classification criteria are not met.
i) STOT - Repeated Exposure	There is sufficient evidence to classify this material as toxic to specific organs through repeated exposure
j) Aspiration Hazard	Based on available data, the classification criteria are not met.
Inhaled	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 high vapour concentrations of N-methyl-2-pyrrolidone (NMP) may produce mucous membrane irritation, headache, giddiness, mental confusion and nausea. Fatalities were not recorded following inhalation of 180-200 mg/m³ for 2 hours by mice and following a 6 hour exposure to saturated vapours by rats. Laboratory animals exposed to concentrations of 50 ppm for 8 hours daily for 20 days or 370 ppm for 6 hours daily for 10 days showed no gross or histopathological abnormalities 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. The biochemical action of this class of material (including bromoxynil, ioxynil) is the same as that of dinitrophenol and dinitro-o-cresol. Exposure may cause disturbance of cellular oxidative phosphorylation process, giving rise to pathological sweating followed by thirst, emaciation and functional renal insufficiency. [CISDOC] Hyperaemia of all organs and oedema of the lungs and brains were seen at autopsy in a man who ingested approximately 43 mg/kg of the congener, ioxynil, in combination with alcohol. Signs of poisoning include raised temperature, increased respiration rate, disorientation and excitability. 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. 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. Symptoms of poisoning by dinitrophenol derivatives include an almost immediate increase in oxygen consumption, body temperature, breathing rate and heart rate. As circulation and respiration do not increase at the same rate as oxygen demand, both anoxia and acidosis appear. Body fat is the main fuel for this extra demand. Direct effects on the cerebrum and lower brain centres, consisting of stimulation, followed by depression are also a feature of these poisonings. Later complications involve necrotising tubular injury of the kidneys which produces renal insufficiency, and toxic hepatitis. Symptoms of poisoning may appear up to two days following exposure muscle tremor and coma. Kidney and liver damage may result in oliguria, haematuria and jaundice. Subacute poisoning as a result of repeated exposures may result in lassitude, headache and malaise. Patients receiving dinitrophenol derivatives report a disarming sense of well being, energy and drive. Workers exposed to harmful levels of these substances should be warned against overheating as toxic effects appear to exaggerated.
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 Prolonged contact with N-methyl-2-pyrrolidone (NMP) reportedly causes severe dermatitis with redness, cracking, swelling, blisters and oedema. An instance of severe skin irritation after a few days work with NMP shows latex rubber gloves as giving insufficient protection. A review article casts doubts on reliability of animal single patch tests, i.e Draize tests. [Irritant Cutaneous Reaction to NMP, Contact Dermatitis 27: 148-150, 1992] 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. Direct contact with the liquid N-methyl-2-pyrrolidone (NMP) may produce painful burning or stinging of the eyes and lids, watering and inflammation of the conjunctiva and temporary corneal clouding. 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. Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. 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.

Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide)

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 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. 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.

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.

Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.

Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.

There is sufficient evidence to establish a causal relationship between human exposure to the material and subsequent developmental toxic effects in the off-spring.

There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of:

- clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.

The teratogenic potential, subchronic and long term inhalation toxicity of N-methyl-2-pyrrolidone (NMP) has been studied in rats. No evidence of nephrotoxicity was seen.

No carcinogenic effects were observed. Very high doses are embryotoxic to rats and mice. Reproductive effects have been reported in animals.

2-Ethylhexanoic acid (2-EHA) its esters and its salts are of concern to human health because of their potential to induce carcinogenicity, liver toxicity and developmental/reproductive toxicity. 2-EHA is of low acute oral and dermal toxicity, is a mild skin irritant and a severe eye irritant. It is not mutagenic in Ames test, but is capable of inducing chromosome aberration and sister chromatid exchanges in vitro, liver toxicity and liver tumours after repeated dose treatment. In addition, 2-EHA has been associated with reproductive and developmental toxicity in experimental animals.

2-EHA is quickly resorbed orally, dermally and following inhalation and almost fully excreted mainly in urine. As in the case of fatty acids, degradation mainly takes place by means of peroxisomal beta-oxidation.

2-EHA has been shown to be a liver and developmental toxicant in animal studies at high doses; in developmental toxicity studies, it was postulated that the maternal liver toxicity began a cascade of effects that included metallothionein (MT) induction, zinc accumulation in the liver due to MT binding, and a resulting zinc deficiency in the developing embryo; the zinc deficiency causes the developmental toxicity; a reproductive/developmental toxicity study was also performed with up to 1% dietary di-2-ethylhexyl terephthalate (DEHT), a 2-ethylhexanoic acid precursor; no reproductive or developmental effects were observed, suggesting that the process of metabolic conversion of DEHT to 2-ethylhexanol and subsequent hydrolysis to 2-ethylhexanoic acid results in a time course of 2-ethylhexanoic acid appearance such that allows clearance before sufficient levels can arise to produce acute liver toxicity.

The closely related substance, 2-ethylhexanol is metabolised to form 2-ethylhexanoic acid and so presents a similar toxicity profile..

2-Ethylhexanoic acid (CAS No. 149-57-5) is classified as hazardous, as a Category 3 reproductive toxin, with the risk phrase 'Possible risk of harm to the unborn child (Xn; R63)'. These effects were noted in the absence of signs of maternal toxicity. The lowest observed adverse effect level (LOAEL) for developmental toxicity was reported to be 100 mg/kg bw/day. Effects on fertility were also reported, with evidence being sufficient to warrant classification as potentially toxic to fertility.

2-Ethylhexanol (CAS No. 104-76-7) was reported to cause developmental toxicity, but not teratogenicity, in rats following treatment via the oral route (NICNAS a). These effects were noted in the absence of signs of marked maternal toxicity. The no observed adverse effect level (NOAEL) for developmental toxicity was reported to be 130 mg/kg bw/day.

Various studies on reproduction toxicity have produced indications of an embryotoxic effect of 2-EHA. After oral administration, NOAEL values for maternal toxicity and foetotoxic effects of 2-EHA were determined in rabbits at 25 and >250 mg/kg body weight/day and in rats at 250 and 100 mg/kg body weight/day. The foetotoxic findings in rats were based on a reduced skeleton ossification at the next higher dose (250 mg/kg body weight/day). No teratogenic effects were observed in this study. In comparison with the structural isomer valproic acid, a known human teratogen, 2-EHA does have similar reprotoxic effects at maternal toxic doses in animal experiments but a far lower potency. Following sub-chronic oral administration of 2-EHA, critical effects like liver changes (higher relative liver weight, histological changes in hepatocytes) were observed in rats and mice and histological renal tubule results were observed in mice. Furthermore, statistically significant, higher cholesterol values were found in all treated male rats (61, 303 and 917 mg/kg body weight/day) and in male and female mice in the middle and high dose groups (885-3139 mg/kg body weight/day). In rats the maximum dose with no adverse effect (NOAEL) was 61 mg/kg body weight/day. In bacterial test systems, mutagenicity studies produced negative findings. In test systems with mammalian cells, by contrast, the findings were weakly positive. Cytogenetic and SCE studies involving CHO cells were positive, one SCE test in human lymphocytes was questionably positive and one experiment concerning tritium-thymidine incorporation into the DNA of mouse lymphocytes was negative. Furthermore, An unpublished micronucleus study on the bone marrow of CD-1 mice was conducted in compliance with OECD Guideline 474. No significant increase in the micronuclei was observed at doses of 400, 800 or 1,600 mg/kg body weight (Inveresk Research International Ltd, 1994). Furthermore, *in vitro* and *in vivo* genotoxicity data (micronucleus test, dominant lethal test) are available for 2-ethylhexanol which is rapidly and quantitatively converted into 2-EHA in metabolism studies. This data do not indicate any genotoxic potential which means that such an effect of 2-EHA is not likely either. As 2-EHA can induce both DNA synthesis and inhibition of intercellular communication in hepatic cells, a tumour-promoting potential in rodents, comparable to that of other peroxisome proliferators, cannot be ruled out. The carcinogenic effect of peroxisome proliferators in rodents (e.g. of di(2-ethylhexyl)phthalate, DEHP) is not deemed to be relevant for humans.

Calcium/zinc and barium/zinc salts of 2-EHA are used as thermo-stabilisers for PVC, together with co-stabilisers like polyols or epoxy compounds, in order to capture the hydrochloride cleaved during the thermal loading of PVC; in addition various salts are used in other food and beverage containers as plasticisers. The migration of 2-EHA from the sealing compounds in the metal lids. has been demonstrated in food contamination. The potential for human exposure to 2-EHA therefore is significant.

Chronic poisoning from ionic bromides has historically resulted from medical use of bromides but not from exposure in the environment or workplace. In the absence of other signs of poisoning, there may be depression, hallucinations and schizophrenia-like psychosis. Bromides may also cause sedation, irritability, agitation, delirium, memory loss, confusion, disorientation, forgetfulness, inability to speak, difficulty speaking, weakness, fatigue, a spinning sensation, stupor, coma, decreased appetite, nausea, vomiting, an acne-like rash on the face (bronchoderma), legs and trunk, swelling of the bronchi and a profuse discharge from the nostrils. There may also be inco-ordination and very brisk reflexes. Correlation of nervous system symptoms with blood levels of bromide is inexact. Current day usage of bromides is generally limited to antihistamines such as brompheniramine, which is a covalent compound; ionic compounds are no longer regularly used due to their toxicity.

In test animals, brominated vegetable oils (BVOs), historically used as emulsifiers in certain soda-based soft drinks, produced damage to the heart and kidneys in addition to increasing fat deposits in these organs. In extreme cases, BVOs caused testicular damage, stunted growth and produced lethargy and fatigue.

Brominism (chronic bromine poisoning) produces slurred speech, apathy, headache, decreased memory, anorexia and drowsiness, psychosis resembling paranoid schizophrenia, and personality changes.

Several cases of foetal abnormalities have been described in mothers who took large doses of bromides during pregnancy.

Reproductive effects caused by bromide (which crosses the placenta) include central nervous system depression, brominism, and bronchoderma (an acne-like rash) in the newborn.

Subchronic bromoxynil feeding studies with rats and dogs produced lower body weights. Increased foetal weights, increased uterine deaths and an increased number of foetuses with an extra 14th rib were noted in a study using pregnant rats fed at 35 mg/kg/day. Repeated doses of 60 mg/kg/day in pregnant rabbits produced hydrocephalus, microphthalmia, anophthalmia and severe defects in ossification of the skull. A increased incidence of adenomas and carcinomas of the liver were noted in male mice.

Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide)

Occupational chronic exposure to both ioxynil and bromoxynil produce inordinate sweating and thirst, fever, headache, dizziness, vomiting, asthenia, weight loss, and myalgia of the legs. The onset of these effects is insidious. Removal of effected workers from exposure resulted in prompt recovery. Pathology showed a transitory elevation of creatinine phosphokinase, LDH aldolase and SGOT.

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.

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

Congener substances such as 2,4-dinitrophenol were previously used to produce weight loss. Adverse effects of the treatment included skin eruption, peripheral neuritis, liver and kidney damage, granulocytopenia and, rarely, cataract formation. Repeated or prolonged contact may produce an allergic or irritant dermatitis. Chronic exposures may produce fatigue and weight loss. A yellow discolouration of the sclerae and urine may indicate adsorption of potentially harmful amounts of the substance.

Chronic exposure to nitrophenols may produce kidney and liver damage. Studies in mammals show changes in neurohumoral regulation and pathology. Colitis, enteritis, hepatitis, gastritis, hyperplasia of the spleen and neuritis may occur.

Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.

Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide)	TOXICITY	IRRITATION
	Not Available	Not Available
MCPA, 2-ethylhexyl ester	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Not Available
	Inhalation (Rat) LC50: >5.05 mg/l4h ^[1]	
N-methyl-2-pyrrolidone	Oral (Rat) LD50: >300<=2000 mg/kg ^[1]	
	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 8000 mg/kg ^[2]	Eye (Human): 530ppm/30M - Mild
	Inhalation (Rat) LC50: 3.1-8.8 mg/l4h ^[2]	Eye (Rodent - rabbit): 0.1mL
	Oral (Rat) LD50: 3914 mg/kg ^[2]	Eye (Rodent - rabbit): 100mg - Moderate
bromoxynil octanoate		Eye: adverse effect observed (irritating) ^[1]
		Skin: adverse effect observed (irritating) ^[1]
	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 1675 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
diflufenican	Inhalation (Rat) LC50: 0.721 mg/l4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: 141 mg/kg ^[1]	
diflufenican	TOXICITY	IRRITATION
	dermal (rat) LD50: 2000 mg/kg ^[2]	Not Available
diflufenican	Oral (Mouse) LD50: >1000 mg/kg ^[2]	

Legend:

1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances

Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide)

Most people have very few pyrroles in their system at any given time; certain individuals, however, have an unusually high number of pyrroles in their bodies, resulting in a condition known as pyroluria. Pyroluria is becoming more and more prevalent both in children with ADHD and autism and in the general population. It is a little known condition; it was first given the name 'mauve factor'.

Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide)

Pyroluria is a genetically acquired chemical imbalance in which the body produces an abnormally large number of pyrroles. A pyrrole is a chemical that is the by-product of haemoglobin synthesis and have no known function in the body; they are normally excreted in the urine. Pyroluria occurs when the pyrroles bind to pyroxidine (vitamin B6) and zinc, causing these vital nutrients to be excreted from the body in large amounts.

Deficiencies of B6 and zinc are associated with a wide range of emotional and psychiatric problems. Vitamin B6 is critical for the production of neurotransmitters and deficiencies can have a profound effect on both mental and physical health. Nervousness, extreme irritability, anxiety, depression, short-term memory problems, and explosive anger have all been linked to Pyroluria.

A large percentage of patients with psychiatric disorders such as schizophrenia exhibit high levels of pyrroles; emotionally dysregulated children and those with an excessive alcohol intake also tend to have an abnormally high pyrrole count.

In addition, zinc deficiencies have been associated with a number of physiological disorders, including poor immune function, poor growth, and delayed sexual development. Because zinc and B6 are so important to both our overall physical and mental health, identifying and treating this devastating condition is critical.

Included in this condition are changes to fatty acid metabolism which lead to high levels of an omega 6 fatty acid call arachidonic acid.

Current research shows that neurological disorders such as ASD as well as psychiatric disorders have a high rate of remission with pyrrole treatment.

Pyrrole Disorder can also be triggered by a traumatic event and high stress levels and is also often part of the picture for PDD, ODD, Tics, Tourette s Syndrome, Depression, and Bipolar Disorder.

Treatment of Pyroluria consists of a replacement of zinc and vitamin B6. Because the treatment is replacing deficiencies not pharmacologic, it needs to be titrated to individual requirements.

Common symptoms associated with Pyrrole Disorder in children (may not include all symptoms):

- Sleep problems, particularly taking a long time to fall asleep and waking up easily at night
- Sensory sensitivity i.e. to the labels on clothes, to specific fabrics – e.g. 'feels prickly', to noises.
- Anxiety. (Anxiety can contribute to digestive upsets such as IBS)
- Poor tolerance of stress
- Depression, pessimistic outlook, mood swings, emotional lability
- Anger and violence out of proportion to the situation, including rage
- Poor functioning of the digestive system, including poor absorption of nutrients as well as food sensitivities
- Low immunity – constantly suffering with coughs and colds
- Motion sickness
- Reading and learning problems
- Poor memory
- Slow growth
- Delayed puberty
- Unpleasant body odour
- Stretch marks on skin
- Pain in joints and extremities
- Sweet fruity breath and body odour, problems with sugar metabolism, allergies

MCPA, 2-ETHYLHEXYL ESTER

551chlph

N-METHYL-2-PYRROLIDONE

for N-methyl-2-pyrrolidone (NMP):

Acute toxicity: In rats, NMP is absorbed rapidly after inhalation, oral, and dermal administration, distributed throughout the organism, and eliminated mainly by hydroxylation to polar compounds, which are excreted via urine. About 80% of the administered dose is excreted as NMP and NMP metabolites within 24 h. A probably dose-dependent yellow coloration of the urine in rodents is observed. The major metabolite is 5-hydroxy-*N*-methyl-2-pyrrolidone.

Studies in humans show comparable results. Dermal penetration through human skin has been shown to be very rapid. NMP is rapidly biotransformed by hydroxylation to 5-hydroxy-*N*-methyl-2-pyrrolidone, which is further oxidized to *N*-methylsuccinimide; this intermediate is further hydroxylated to 2-hydroxy-*N*-methylsuccinimide. These metabolites are all colourless. The excreted amounts of NMP metabolites in the urine after inhalation or oral intake represented about 100% and 65% of the administered doses, respectively.

NMP has a low potential for skin irritation and a moderate potential for eye irritation in rabbits. Repeated daily doses of 450 mg/kg body weight administered to the skin caused painful and severe haemorrhage and eschar formation in rabbits. These adverse effects have not been seen in workers occupationally exposed to pure NMP, but they have been observed after dermal exposure to NMP used in cleaning processes. No sensitisation potential has been observed.

In acute toxicity studies in rodents, NMP showed low toxicity. Uptake of oral, dermal, or inhaled acutely toxic doses causes functional disturbances and depressions in the central nervous system. Local irritation effects were observed in the respiratory tract when NMP was inhaled and in the pyloric and gastrointestinal tracts after oral administration. In humans, there was no irritative effect in the respiratory system after an 8-h exposure to 50 mg/m3.

Repeat dose toxicity: There is no clear toxicity profile of NMP after multiple administration. In a 28-day dietary study in rats, a compound-related decrease in body weight gain was observed in males at 1234 mg/kg body weight and in females at 2268 mg/kg body weight.

Testicular degeneration and atrophy in males and thymic atrophy in females were observed at these dose levels. The no-observed-adverse-effect level (NOAEL) was 429 mg/kg body weight in males and 1548 mg/kg body weight in females. In a 28-day intubation study in rats, a dose-dependent increase in relative liver and kidney weights and a decrease in lymphocyte count in both sexes were observed at 1028 mg/kg body weight. The NOAEL in this study was 514 mg/kg body weight. In another rat study, daily dietary intake for 90 days caused decreased body weights at doses of 433 and 565 mg/kg body weight in males and females, respectively. There were also neurobehavioural effects at these dose levels. The NOAELs in males and females were 169 and 217 mg/kg body weight, respectively.

The toxicity profile after exposure to airborne NMP depends strongly on the ratio of vapour to aerosol and on the area of exposure (i.e., head-only or whole-body exposure). Because of higher skin absorption for the aerosol, uptake is higher in animals exposed to aerosol than in those exposed to vapour at similar concentrations. Studies in female rats exposed head only to 1000 mg/m3 showed only minor nasal irritation, but massive mortality and severe effects on major organs were observed when the females were whole-body exposed to the same concentration of coarse droplets at high relative humidity. Several studies in rats following repeated exposure to NMP at concentrations between 100 and 1000 mg/m3 have shown systemic toxicity effects at the lower dose levels. In most of the studies, the effects were not observed after a 4-week observation period.

In rats, exposure to 3000 mg NMP/m3 (head only) for 6 h/day, 5 days/week, for 13 weeks caused a decrease in body weight gain, an increase in erythrocytes, haemoglobin, haematocrit, and mean corpuscular volume, decreased absolute testis weight, and cell loss in the germinal epithelium of the testes. The NOAEL was 500 mg/m3.

There are no data in humans after repeated-dose exposure.

Carcinogenicity: NMP did not show any clear evidence for carcinogenicity in rats exposed to concentrations up to 400 mg/m3 in a long-term inhalation study.

Genotoxicity: The mutagenic potential of NMP is weak. Only a slight increase in the number of revertants was observed when tested in a *Salmonella* assay with base-pair substitution strains. NMP has been shown to induce aneuploidy in yeast *Saccharomyces cerevisiae* cells. No investigations regarding mutagenicity in humans were available.

Reproductive toxicity: In a two-generation reproduction study in rats, whole-body exposure of both males and females to 478 mg/m3 of NMP vapour for 6 h/day, 7 days/week, for a minimum of 100 days (pre-mating, mating, gestation, and lactation periods) resulted in a 7% decrease in fetal weight in the F1 offspring. A 4-11% transient, non-dose-dependent decrease was observed in the average pup weight at all exposure levels tested (41, 206, and 478 mg/m3).

Developmental toxicity: When NMP was administered dermally, developmental toxicity was registered in rats at 750 mg/kg body weight. The observed effects were increased preimplantation losses, decreased fetal weights, and delayed ossification. The NOAEL for both developmental effects and maternal toxicity (decreased body weight gain) was 237 mg/kg body weight.

Inhalation studies in rats (whole-body exposure) demonstrated developmental toxicity as increased preimplantation loss without significant effect on implantation rate or number of live fetuses at 680 mg/m3 and behavioural developmental toxicity at 622 mg/m3. In an inhalation study (whole-body exposure), the NOAEL for maternal effects was 100 mg/m3, and the NOAEL for developmental effects was 360 mg/m3.

A tolerable inhalation concentration, 0.3 mg/m3, based on mortality and organ damage, is expected to be protective against any possible reproductive toxicity. Similarly, an oral tolerable intake of 0.6 mg/kg body weight per day, based on a 90-day study, is expected to provide

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Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide)

	adequate protection against possible reproductive effects. Because of non-existent data on the exposure of the general population and very limited information on occupational exposure, no meaningful risk characterisation can be performed A substance (or part of a group of chemical substances) of very high concern (SVHC) - or product containing an SVHC: It is proposed that use within the European Union be subject to authorisation under the REACH Regulation. Indeed, listing of a substance as an SVHC by the European Chemicals Agency (ECHA) is the first step in the procedure for authorisation or restriction of use of a chemical. The criteria are given in article 57 of the REACH Regulation. A substance may be proposed as an SVHC if it meets one or more of the following criteria: ▶ it is carcinogenic *; ▶ it is mutagenic *; ▶ it is toxic for reproduction *; ▶ it is persistent, bioaccumulative and toxic (PBT substances); ▶ it is very persistent and very bioaccumulative (vPvB substances); ▶ there is "scientific evidence of probable serious effects to human health or the environment which give rise to an equivalent level of concern"; such substances are identified on a case-by-case basis. * Collectively described as CMR substances The "equivalent concern" criterion is significant because it is this classification which allows substances which are, for example, neurotoxic, endocrine-disrupting or otherwise present an unanticipated environmental health risk to be regulated under REACH] Simply because a substance meets one or more of the criteria does not necessarily mean that it will be proposed as an SVHC. Many such substances are already subject to restrictions on their use within the European Union, such as those in Annex XVII of the REACH Regulation SVHCs are substances for which the current restrictions on use (where these exist) might be insufficient. There are three priority groups for assessment: ▶ PBT substances and vPvB substances; ▶ substances which are widely dispersed during use; ▶ substances which are used in large quantities.
BROMOXYNIL OCTANOATE	NOEL (90 day) for rats 15.6 mg/kg, dogs 5 mg/kg daily.
DIFLUFENICAN	ADI: 0.2 mg/kg/day NOEL: 16.3 mg/kg/day
Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide) & BROMOXYNIL OCTANOATE	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.
Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide) & N-METHYL-2-PYRROLIDONE	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide) & MCPA, 2-ETHYLHEXYL ESTER	For chlorophenoxy pesticides: WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. Side-reactions during manufacture of the parent compound may result in the production of trace amounts of polyhalogenated aromatic hydrocarbon(s). Halogenated phenols, and especially their alkali salts, can condense above 300 deg. C. to form polyphenoxyphenols or, in a very specific reaction, to form dibenzo-p-dioxins Polyhalogenated aromatic hydrocarbons (PHAHs) comprise two major groups. The first group represented by the halogenated derivatives of dibenzodioxins (the chlorinated form is PCDD), dibenzofurans (PCDF) and biphenyls (PCB) exert their toxic effect (as hepatocarcinogens, reproductive toxicants, immunotoxicants and procarcinogens) by interaction with a cytosolic protein known as the Ah receptor. In guinea pigs the Ah receptor is active in a mechanism which "pumps" PHAH into the cell whilst in humans the reverse appears to be true. This, in part, may account for species differences often cited in the literature. This receptor exhibits an affinity for the planar members of this group and carries these to the cellular nucleus where they bind, reversibly, to specific genomes on DNA. This results in the regulation of the production of certain proteins which elicit the toxic response. The potency of the effect is dependent on the strength of the original interaction with the Ah receptor and is influenced by the degree of substitution by the halogen and the position of such substitutions on the parent compound. The most potent molecule is 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) while the coplanar PCBs (including mono-ortho coplanars) possess approximately 1% of this potency. Nevertheless, all are said to exhibit "dioxin-like" behaviour and in environmental and health assessments it has been the practice to assign each a TCDD-equivalence value. The most subtle and important biological effects of the PHAHs are the effects on endocrine hormones and vitamin homeostasis. TCDD mimics the effect of thyroxine (a key metamorphosis signal during maturation) and may disrupt patterns of embryonic development at critical stages. Individuals from exposed wildlife populations have been observed to have altered sexual development, sexual dysfunction as adults and immune system suppression. Immunotoxic effects of the PHAHs (including the brominated congener, PBB) have been the subject of several studies. No clear pattern emerges in human studies however with T-cell numbers and function (a blood marker for immunological response) increasing in some and decreasing in others. Developmental toxicity (e.g. cleft palate, hydronephrosis) occurs in relatively few species; functional alterations following TCDD exposure leads to deficits in cognitive functions in monkeys and to adverse effects in the male reproductive system of rats. Three incidences have occurred which have introduced abnormally high levels of dioxin or dioxin-like congeners to humans. The explosion at a trichlorophenol-manufacturing plant in Seveso, Italy distributed TCDD across a large area of the country-side, whilst rice-oil contaminated with heat-transfer PCBs (and dioxin-like contaminants) has been consumed by two groups, on separate occasions (one in Yusho, Japan and another in Yu-cheng, Taiwan). The only symptom which can unequivocally be related to all these exposures is the development of chloracne, a disfiguring skin condition, following each incident. Contaminated oil poisonings also produced eye-discharge, swelling of eyelids and visual disturbances. The Babies born up to 3 years after maternal exposure (so-called "Yusho-babies") were characteristically brown skinned, coloured gums and nails and (frequently) produced eye-discharges. Delays in intellectual development have been noted. It has been estimated that Yu-cheng patients consumed an average level of 0.06 mg/kg body weight/day total PCB and 0.0002 mg/kg/day of PCDF before the onset of symptoms after 3 months. When the oil was withdrawn after 6 months they had consumed 1 gm total PCB containing 3.8 mg PCDF. Taiwanese patients consumed 10 times as much contaminated oil as the Japanese patients (because of later withdrawal); however since PCB/PCDF concentration in the Japanese oil was 10 times that consumed in Taiwan, patients from both countries consumed about the same amount of PCBs/PCDFs. Preliminary data from the Yusho cohort suggests a six-fold excess of liver cancer mortality in males and a three-fold excess in women. Recent findings from Seveso indicate that the biological effects of low level exposure (BELLEs), experienced by a cohort located at a great distance from the plant, may be hormetic, i.e. may be protective AGAINST the development of cancer. The PHAHs do not appear to be genotoxic - they do not alter the integrity of DNA. This contrasts with the effects of the many polycyclic aromatic hydrocarbons (PAHs) (or

Continued...

Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide)

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

Animal Metabolism – MCPA is rapidly absorbed and eliminated from mammalian systems. Rats eliminated nearly all of a single oral dose within 24 hours, mostly in urine with little or no metabolism. In another rat study, three quarters of the dose was eliminated within two days. All was gone the by the eighth day. Humans excreted about half of a 5 mg dose in the urine within a few days. No residues were found after day five. Cattle and sheep fed MCPA in low to moderate doses in the diet for two weeks had no residues from levels less than about 18 mg/kg. The major metabolite of MCPA is 2-methyl-4-chlorophenol in the free and conjugated form, which is formed in the liver

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

Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide)	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
MCPA, 2-ethylhexyl ester	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	0.11mg/l	1
	EC50	48h	Crustacea	0.29mg/l	1
	EC50	96h	Algae or other aquatic plants	0.17mg/l	1
	EC10(ECx)	120h	Algae or other aquatic plants	0.002mg/l	1
	LC50	96h	Fish	>0.283mg/l	2
N-methyl-2-pyrrolidone	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>500mg/l	1
	EC50	48h	Crustacea	ca.4897mg/l	1
	LC50	96h	Fish	464mg/l	1
	NOEC(ECx)	504h	Crustacea	12.5mg/l	2
bromoxynil octanoate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	0.015-0.056mg/L	4
	NOEC(ECx)	840h	Fish	0.003mg/L	2
	EC50	96h	Algae or other aquatic plants	4.43mg/l	4
	LC50	96h	Fish	0.028-0.062mg/L	4
diflufenican	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	<0.001mg/L	4
	LC50	96h	Fish	56mg/l	Not Available
	EC50(ECx)	72h	Algae or other aquatic plants	<0.001mg/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				

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

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.

for N-methyl-2-pyrrolidinone (NMP):

log Kow : -0.44-0.1

Environmental Fate

Continued...

Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide)

NMP may enter the environment as emissions to the atmosphere, as the substance is volatile and widely used as a solvent, or it may be released to water as a component of municipal and industrial wastewaters. The substance is mobile in soil, and leaching from landfills is thus a possible route of contamination of groundwater.

In air, NMP is expected to be removed by wet deposition or by photochemical reactions with hydroxyl radicals. As the substance is completely miscible in water, it is not expected to adsorb to soil, sediments, or suspended organic matter or to bioconcentrate. NMP is not degraded by chemical hydrolysis. Data from screening tests on the biodegradability of NMP show that the substance is rapidly biodegraded.

This material is not expected to persist in the environment. It is water soluble and is expected to have low volatility. Hydrolysis is not expected to be an important factor in the environmental fate process for this material.

Persistence and Degradability
Biodegradation: BOD (Modified MITI Method) = 73% (28 days). BOD (Modified MITI Method) = 92% (14 days). This material is expected to be readily biodegradable.
Bioaccumulation: BCF = 0.16. This material is not expected to bioaccumulate

Ecotoxicity
This material is expected to be non-hazardous to aquatic species.
Fish LC50 (96 h): bluegill. 832 mg/l, fathead minnow 1072 mg/l; rainbow trout 3048 mg/l
Daphnia magna EC50 (24 h): > 1000 mg/l
Algae EC50 (72 h): Scenedesmus subspicatus > 500 mg/l

for chlorophenoxy herbicides:

Environmental fate:
Residues of chlorophenoxy herbicides in the environment are the consequence of the direct application of these compounds to agricultural and non-agricultural areas. Biodegradation is the primary route of elimination from the environment; photolysis and hydrolysis also contribute to their removal.

The chlorophenoxy herbicides are considered to have only marginal potential for leaching to groundwater. In basic waters, phenoxy herbicide esters are hydrolysed to the anionic forms; in acidic waters, photodegradation or vaporisation predominates, depending on the ester.

Chlorophenoxy herbicides may be transported in the atmosphere in the form of droplets, vapour, or powder following application by spraying.

Chlorophenoxy herbicides may be present in food as a result of their direct application to crops; however, concentrations are normally low for bromoxynil:

Environmental fate:
Soil: Bromoxynil has a low persistence in soil. It is broken down in the environment by photolytic degradation, abiotic hydrolysis and microbial-mediated metabolism under both aerobic and anaerobic conditions. In sandy soil, the half-life is about 10 days. Degradation in clay was slower, with half of the bromoxynil degraded to its metabolites in about a 2-week period at 25°C. The persistence of the compound is also slightly longer in peat field soils than in the sandy soils.

Water – Environmental fate studies indicate that bromoxynil (phenol and octanoate) should not persist in surface waters. The aerobic aquatic metabolism study shows rapid degradation with a half-life of <12 hours

Transport: Based on the available data, U.S. EPA concluded that the potential for ground water contamination from bromoxynil octanoate is low; it does not exhibit the mobility or persistence characteristics of pesticides that are normally found in ground water.

Metabolism
Mode of Action : Bromoxynil is a nitrile herbicide that acts by inhibiting photosynthesis in target species of plant. Its primary use is for control of post-emergent broadleaf weeds

Plant Metabolism: The herbicide works by disrupting the plants ability to produce energy for cell-related activities. It is not readily translocated throughout the plant once it has been absorbed.

Animal Metabolism – No bromoxynil was present in the milk or feces of cows 9 days after exposure to low doses of the herbicide. Less than 20% of the compound was excreted in urine as the parent compound

The group of acidic herbicides, including the phenoxy acids, possess functional groups that ionise in aqueous systems yielding pKa values of less than 4. The behaviour of these materials is closely correlated with their acid character. The most significant factor with respect to soil mobility is the organic content of the soil which readily absorbs these compounds. Furthermore in acidic systems these compounds are also absorbed by clay particles. The esters and ethers are expected to behave differently from the acid forms although hydrolysis may influence subsequent binding. In general the esters and ethers are considered non-persistent in the environment.

MCPA and its formulations are rapidly degraded by soil microorganisms and it has low persistence, with a reported field half-life of 14 days to 1 month, depending on soil moisture and soil organic matter. Decreased soil moisture and microbial activity, as well as increased soil organic matter, will prolong the field half-life for MCPA.

Water – MCPA is relatively stable to light breakdown, but can be rapidly broken down by microorganisms. In sterilized water, it takes about five weeks for half of the compound to degrade due to the action of sunlight. In rice paddy water, however, MCPA is almost totally degraded by aquatic microorganisms in under two weeks.

Transport
MCPA readily leaches in most soils, but its mobility decreases with increasing organic matter. MCPA and its formulations show little affinity for soil.

Metabolism
Mode of Action – MCPA works by concentrating in the actively growing regions of a plant (meristematic tissue) where it interferes with protein synthesis, cell division and ultimately the growth of the plant .

Plant Metabolism – MCPA is absorbed, translocated, and actively broken down by vegetation. The metabolite found in plants is 2-methyl-4-chlorophenol.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
MCPA, 2-ethylhexyl ester	HIGH	HIGH
N-methyl-2-pyrrolidone	LOW	LOW
bromoxynil octanoate	LOW (Half-life = 44 days)	LOW (Half-life = 2.65 days)
diflufenican	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
MCPA, 2-ethylhexyl ester	HIGH (LogKOW = 6.1705)
N-methyl-2-pyrrolidone	LOW (BCF = 0.16)
bromoxynil octanoate	HIGH (LogKOW = 5.4)
diflufenican	HIGH (LogKOW = 4.9)

Mobility in soil

Ingredient	Mobility
MCPA, 2-ethylhexyl ester	LOW (Log KOC = 10510)
N-methyl-2-pyrrolidone	LOW (Log KOC = 20.94)
bromoxynil octanoate	LOW (Log KOC = 4847)
diflufenican	LOW (Log KOC = 109900)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	► Containers may still present a chemical hazard/ danger when empty.
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Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide)

- ▶ 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 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

COMBUSTIBLE LIQUID	COMBUSTIBLE LIQUID, regulated for storage purposes only
Marine Pollutant	
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

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
MCPA, 2-ethylhexyl ester	Not Applicable
N-methyl-2-pyrrolidone	Not Applicable
bromoxynil octanoate	Not Applicable
diflufenican	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
MCPA, 2-ethylhexyl ester	Not Applicable
N-methyl-2-pyrrolidone	Not Applicable
bromoxynil octanoate	Not Applicable
diflufenican	Not Applicable

SECTION 15 Regulatory information

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

MCPA, 2-ethylhexyl ester is found on the following regulatory lists

Australia Chemicals with non-industrial uses removed from the Australian Inventory of Chemical Substances (old Inventory)
 Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

N-methyl-2-pyrrolidone is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
 Australian Inventory of Industrial Chemicals (AIIC)
 Chemical Footprint Project - Chemicals of High Concern List

bromoxynil octanoate is found on the following regulatory lists

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

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7
Chemical Footprint Project - Chemicals of High Concern List

diflufenican is found on the following regulatory lists
Australia Chemicals with non-industrial uses removed from the Australian Inventory of Chemical Substances (old Inventory)
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

Additional Regulatory Information
Not Applicable

National Inventory	Status
Australia - AIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (MCPA, 2-ethylhexyl ester; bromoxynil octanoate; diflufenican)
Canada - NDSL	No (MCPA, 2-ethylhexyl ester; N-methyl-2-pyrrolidone; diflufenican)
China - IECSC	No (diflufenican)
Europe - EINEC / ELINCS / NLP	No (diflufenican)
Japan - ENCS	No (MCPA, 2-ethylhexyl ester; bromoxynil octanoate; diflufenican)
Korea - KECI	No (MCPA, 2-ethylhexyl ester; bromoxynil octanoate; diflufenican)
New Zealand - NZIoC	Yes
Philippines - PICCS	No (MCPA, 2-ethylhexyl ester; bromoxynil octanoate; diflufenican)
USA - TSCA	TSCA Inventory 'Active' substance(s) (N-methyl-2-pyrrolidone; bromoxynil octanoate); No (MCPA, 2-ethylhexyl ester; diflufenican)
Taiwan - TCSI	No (MCPA, 2-ethylhexyl ester; bromoxynil octanoate)
Mexico - INSQ	No (MCPA, 2-ethylhexyl ester; bromoxynil octanoate; diflufenican)
Vietnam - NCI	Yes
Russia - FBEPH	Yes
UAE - Control List (Banned/Restricted Substances)	No (MCPA, 2-ethylhexyl ester; N-methyl-2-pyrrolidone; bromoxynil octanoate; diflufenican)
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	30/04/2026
Initial Date	30/04/2026

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
 - AIC: 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

Titan Triforto EC (Titan Diflufenican Bromoxynil MCPA Herbicide)

- ▶ 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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