Titan Pinoxaden 100 EC (Titan Pinoxaden 100EC Herbicide)

Titan Ag

Chemwatch Hazard Alert Code: 3

Chemwatch: **7655-237**
Version No: **2.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Initial Date: **25/03/2026**
Revision Date: **02/06/2026**
Print Date: **09/06/2026**
L.GHS.AUS.EN.E

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

Product Identifier

Product name	Titan Pinoxaden 100 EC (Titan Pinoxaden 100EC Herbicide)
Chemical Name	Not Applicable
Synonyms	APVMA Approval Number: 85289
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains solvent naphtha petroleum, heavy aromatic)
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.

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	S5
Classification [1]	Flammable Liquids Category 4, Acute Toxicity (Oral) Category 4, Aspiration Hazard Category 1, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2A, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Reproductive Toxicity Category 1A, Hazardous to the Aquatic Environment Acute Hazard Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 2
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI***

Label elements

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

Hazard statement(s)

H227	Combustible liquid.
H302	Harmful if swallowed.
H304	May be fatal if swallowed and enters airways.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H319	Causes serious eye irritation.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.
H360D	May damage the unborn child.
H411	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.
P271	Use only a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P331	Do NOT induce vomiting.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
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.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P330	Rinse mouth.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

No further product hazard information.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
64742-94-5	60-<65	solvent naphtha petroleum, heavy aromatic
98-86-2	10-15	acetophenone
872-50-4	10-15	N-methyl-2-pyrrolidone
243973-20-8	10	pinoxaden
99734-09-5	8-10	tristyrylphenol, ethoxylated
99607-70-2	2.5	cloquintocet-mexyl
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available***	

SECTION 4 First aid measures

Description of first aid measures

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Eye Contact	If this product comes in contact with the eyes: ▸ Wash out immediately with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	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 do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice. ▸ Avoid giving milk or oils. ▸ Avoid giving alcohol. ▸ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

Indication of any immediate medical attention and special treatment needed

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.
Treat symptomatically.
For petroleum distillates
· In case of ingestion, gastric lavage with activated charcoal can be used promptly to prevent absorption - decontamination (induced emesis or lavage) is controversial and should be considered on the merits of each individual case; of course the usual precautions of an endotracheal tube should be considered prior to lavage, to prevent aspiration.
· Individuals intoxicated by petroleum distillates should be hospitalized immediately, with acute and continuing attention to neurologic and cardiopulmonary function.
· Positive pressure ventilation may be necessary.
· Acute central nervous system signs and symptoms may result from large ingestions of aspiration-induced hypoxia.
· After the initial episode,individuals should be followed for changes in blood variables and the delayed appearance of pulmonary oedema and chemical pneumonitis. Such patients should be followed for several days or weeks for delayed effects, including bone marrow toxicity, hepatic and renal impairment Individuals with chronic pulmonary disease will be more seriously impaired, and recovery from inhalation exposure may be complicated.
· Gastrointestinal symptoms are usually minor and pathological changes of the liver and kidneys are reported to be uncommon in acute intoxications.
· Chlorinated and non-chlorinated hydrocarbons may sensitize the heart to epinephrine and other circulating catecholamines so that arrhythmias may occur.Careful consideration of this potential adverse effect should precede administration of epinephrine or other cardiac stimulants and the selection of bronchodilators.
Treatment regime for chloroquine and its congeners :
- In gross overdose prompt treatment is essential.
- Empty the stomach by inducing emesis or by aspiration and lavage.
- The use of charcoal has been suggested.
- Respiration may require assistance and intravenous fluids and vasopressors may be given for hypotension.
- Ammonium chloride in doses of up to 8 gm daily by mouth has been recommended to enhance urinary
- excretion but hazards exist in forced diuresis.
- Sodium lactate injection has been given intravenously to counter depressant effects of
- chloroquinone on the heart.
- Electrical pacing of the heart may be required.
- Dialysis procedures seem to offer little benefit

For acetophenone:
- Sympathomimetics, such as ephedrine and phenylephrine have been suggested as potential antidotes.
- Drugs which block the autonomic nervous system (such as atropine and scopolamine) are contraindicated.
- Acetophenone is converted to methylphenylcarbinol and benzoic acid. The benzoic acid is conjugated with glycine and excreted in the urine as hippuric acid.

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

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

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.

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	▸ Mists containing combustible materials may be explosive. Combustion products include: ▸ carbon dioxide (CO_2) nitrogen oxides (NO_x) ▸ other pyrolysis products typical of burning organic material.
HAZCHEM	●3Z

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. ▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Control personal contact with the substance, by using protective equipment. ▸ Contain and absorb spill with sand, earth, inert material or vermiculite. ▸ Wipe up. ▸ Place in a suitable, labelled container for waste disposal.
Major Spills	Environmental hazard - contain spillage. Chemical Class: aliphatic hydrocarbons For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
LAND SPILL - SMALL				
cross-linked polymer - particulate	1	shovel	shovel	R, W, SS
cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT
wood fiber - pillow	2	throw	pitchfork	R, P, DGC, RT
treated wood fibre- pillow	2	throw	pitchfork	DGC, RT
sorbent clay - particulate	3	shovel	shovel	R, I, P
foamed glass - pillow	3	throw	pitchfork	R, P, DGC, RT
LAND SPILL - MEDIUM				
cross-linked polymer - particulate	1	blower	skiploader	R,W, SS
cross-linked polymer - pillow	2	throw	skiploader	R, DGC, RT
sorbent clay - particulate	3	blower	skiploader	R, I, P
polypropylene - particulate	3	blower	skiploader	W, SS, DGC
expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC
polypropylene - mat	4	throw	skiploader	DGC, RT

Legend
DGC: Not effective where ground cover is dense
R; Not reusable
I: Not incinerable
P: Effectiveness reduced when rainy
RT:Not effective where terrain is rugged
SS: Not for use within environmentally sensitive sites
W: Effectiveness reduced when windy
Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;
R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988
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	The conductivity of this material may make it a static accumulator., A liquid is typically considered nonconductive if its conductivity is below 100 pS/m and is considered semi-conductive if its conductivity is below 10 000 pS/m., Whether a liquid is nonconductive or semi-conductive, the precautions are the same., A number of factors, for example liquid temperature, presence of contaminants, and anti-static additives can greatly influence the conductivity of a liquid. ▸ Containers, even those that have been emptied, may contain explosive vapours. ▸ Do NOT cut, drill, grind, weld or perform similar operations on or near containers.

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	▸ **DO NOT** allow clothing wet with material to stay in contact with skin ▸ 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.
Other information	Consider storage under inert gas. ▸ 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	For alkyl aromatics: The alkyl side chain of aromatic rings can undergo oxidation by several mechanisms. The most common and dominant one is the attack by oxidation at benzylic carbon as the intermediate formed is stabilised by resonance structure of the ring. ▸ Following reaction with oxygen and under the influence of sunlight, a hydroperoxide at the alpha-position to the aromatic ring, is the primary oxidation product formed (provided a hydrogen atom is initially available at this position) - this product is often short-lived but may be stable dependent on the nature of the aromatic substitution; a secondary C-H bond is more easily attacked than a primary C-H bond whilst a tertiary C-H bond is even more susceptible to attack by oxygen ▸ Monoalkylbenzenes may subsequently form monocarboxylic acids; alkyl naphthalenes mainly produce the corresponding naphthalene carboxylic acids. ▸ Oxidation in the presence of transition metal salts not only accelerates but also selectively decomposes the hydroperoxides. ▸ Hock-rearrangement by the influence of strong acids converts the hydroperoxides to hemiacetals. Peresters formed from the hydroperoxides undergo Criegee rearrangement easily. ▸ Alkali metals accelerate the oxidation while CO2 as co-oxidant enhances the selectivity. ▸ Microwave conditions give improved yields of the oxidation products. ▸ Photo-oxidation products may occur following reaction with hydroxyl radicals and NOx - these may be components of photochemical smogs. Oxidation of Alkylaromatics: T.S.S Rao and Shubhra Awasthi: E-Journal of Chemistry Vol 4, No. 1, pp 1-13 January 2007 ▸ Vigorous reactions, sometimes amounting to explosions, can result from the contact between aromatic rings and strong oxidising agents. ▸ Aromatics can react exothermically with bases and with diazo compounds. Acetophenone: ▸ is incompatible with strong acids, aldehydes, aliphatic amines, oxidisers, perchloric acid, hydrogen peroxide ▸ attacks some plastics, coatings and rubber.

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)

Exposed individuals are **NOT** reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

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Classification into classes follows:

Class	OSF	Description
A	550	Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities
B	26-550	As "A" for 50-90% of persons being distracted
C	1-26	As "A" for less than 50% of persons being distracted
D	0.18-1	10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached
E	<0.18	As "D" for less than 10% of persons aware of being tested

Odour threshold: 0.25 ppm.
The TLV-TWA is protective against ocular and upper respiratory tract irritation and is recommended for bulk handling of gasoline based on calculations of hydrocarbon content of gasoline vapour. A STEL is recommended to prevent mucous membrane and ocular irritation and prevention of acute depression of the central nervous system. Because of the wide variation in molecular weights of its components, the conversion of ppm to mg/m3 is approximate. Sweden recommends hexane type limits of 100 ppm and heptane and octane type limits of 300 ppm. Germany does not assign a value because of the widely differing compositions and resultant differences in toxic properties.
Odour Safety Factor (OSF)
OSF=0.042 (gasoline)
For acetophenone:
Odour Threshold Value: 0.17 ppm
Acetophenone produces narcosis and anaesthesia when swallowed or injected intravenously or subcutaneously. Contact with the skin produces irritation and, if confined, may burn. Eye contact may produce marked irritation and transient corneal injury depending on the extent and duration of contact.
Acute inhalation studies indicate that a saturated atmosphere (nominally 400 ppm) produced no fatalities in rats exposed for 8 hours. Whether the substance is irritating at or around the reported odour threshold is inconclusive. Subchronic oral studies indicate no effect on growth or haematology at doses of up to 10000 ppm in the diet. The TLV-TWA is thought to be protective against the potential irritant or odour effects of acetophenone.

Exposure controls

Appropriate engineering controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.

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

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

Within each range the appropriate value depends on:

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

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

Individual protection measures, such as personal protective equipment

Eye and face protection

- Safety glasses with side shields.
- Chemical goggles.[AS/NZS 1337.1, EN166 or national equivalent]
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].

Skin protection

See Hand protection below

Hands/feet protection

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

NOTE:

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

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	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 Pinoxaden 100 EC (Titan Pinoxaden 100EC Herbicide)

Material	CPI
BUTYL	C
NATURAL RUBBER	C
PE/EVAL/PE	C
PVA	C
TEFLON	C

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	AK-AUS P2	-	AK-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AK-AUS / Class 1 P2	-
up to 100 x ES	-	AK-2 P2	AK-PAPR-2 P2 ^

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

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Clear yellow to orange coloured liquid with aromatic odour; mixes with water.		
Physical state	Liquid	**Relative density (Water = 1)**	1.03

Continued...

Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Applicable	**Viscosity (cSt)**	8.097 @ 20 C
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	79	**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	Miscible	**pH as a solution (1%)**	3-7
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available
Heat of Combustion (kJ/g)	Not Available	**Ignition Distance (cm)**	Not Available
Flame Height (cm)	Not Available	**Flame Duration (s)**	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	**Enclosed Space Ignition Deflagration Density (g/m3)**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

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

Inhaled	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 vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. 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 High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations.

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	Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal. The acute toxicity of inhaled alkylbenzene is best described by central nervous system depression. These compounds may also act as general anaesthetics. Whole body symptoms of poisoning include light-headedness, nervousness, apprehension, a feeling of well-being, confusion, dizziness, drowsiness, ringing in the ears, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness, depression of breathing, and arrest. Heart stoppage may result from cardiovascular collapse. A slow heart rate and low blood pressure may also occur. Alkylbenzenes are not generally toxic except at high levels of exposure. Their breakdown products have low toxicity and are easily eliminated from the body. Acetophenone is hypnotic in high concentrations and may cause narcosis and central nervous system depression. Overexposure by inhalation is unlikely because of low volatility and odour-warning properties. Vapours do not irritate throat. Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.
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. Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result. Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis). Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosal ulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea and vomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injury may produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Light aromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration into the lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage. Side-effects of chloroquine and its congeners include: headache, gastrointestinal disturbances (nausea,vomiting, diarrhoea and abdominal cramps), pruritis, and macular, urticarial and purpuric skin eruptions. Occasional psychotic episodes, convulsions, hypotension and cardiovascular collapse, ECG changes, double vision and difficulty in accommodation have been reported. Overdose is expected to produce the same effects as overdose with chloroquine viz: respiratory and cardiovascular depression, arrhythmias, shock, followed by convulsions, respiratory and cardiac arrest and death. Ingestion of acetophenone may produce anaesthetic effects.
Skin Contact	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 Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. 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] One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants Open cuts, abraded or irritated skin should not be exposed to this material 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. Rabbit skin subject to continuous contact with acetophenone, for 24 hours with undiluted product, showed irritation described as "mild burn". When applied to rabbit skin under occlusive cover, acetophenone produced severe skin injury (necrosis), and kidney injury. 2% acetophenone in petrolatum (maximisation test) produced no skin sensitisation in humans. No evidence for sensitisation was observed following intradermal injection in guinea pigs of 0.1 ml of 0.6% acetophenone, followed by a challenge treatment two weeks later. Aromatic hydrocarbons may produce skin irritation, vasodilation with erythema and changes in endothelial cell permeability. Systemic intoxication, resulting from contact with the light aromatics, is unlikely due to the slow rate of permeation. Branching of the side chain appears to increase percutaneous absorption.
Eye	This material causes serious eye irritation. 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. Some nonionic surfactants may produce a localised anaesthetic effect on the cornea; this may effectively eliminate the warning discomfort produced by other substances and lead to corneal injury. Irritant effects range from minimal to severe dependent on the nature of the surfactant, its concentration and the duration of contact. Pain and corneal damage represent the most severe manifestation of irritation. Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may also result. The aromatic fraction may produce irritation and lachrymation. Introduction of 0.005 ml of a 15% solution in propylene glycol produced severe corneal necrosis in rabbits. Application of 2 drops of saturated aqueous solution of acetophenone to rabbit eyes caused discomfort despite prior application of local anaesthetic. Acetophenone vapours do not appear to irritate the eye.
Chronic	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. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers

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Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.
Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.

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.
Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.
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.
Prolonged administration of high doses of chloroquine (common in the treatment of rheumatoid arthritis) and its congeners may lead to pigmented deposits and opacities in the cornea which are reversible if treatment is withdrawn. There is a risk of retinopathy with lesions, defects in colour vision, optic nerve atrophy, scotomas, field defects and blindness. Uncommon adverse effects include loss of hair, bleaching of hair pigment, bluish-black pigmentation of mucous membranes and skin, photosensitivity, lichen planus like eruptions, aural defects, neuromyopathy and myopathy. Blood disorders such as agranulocytosis, thrombocytopenia and neutropenia have been reported on rare occasions. Aplastic anaemia may develop.
Chronic dermatoses may be lichenoid, eczemoid or exfoliative in nature.
Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses. Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.
Hydrocarbon solvents are liquid hydrocarbon fractions derived from petroleum processing streams, containing only carbon and hydrogen atoms, with carbon numbers ranging from approximately C5-C20 and boiling between approximately 35-370 deg C. Many of the hydrocarbon solvents have complex and variable compositions with constituents of 4 types, alkanes (normal paraffins, isoparaffins, and cycloparaffins) and aromatics (primarily alkylated one- and two-ring species). Despite the compositional complexity, most hydrocarbon solvent constituents have similar toxicological properties, and the overall toxicological hazards can be characterized in generic terms.
Hydrocarbon solvents can cause chemical pneumonitis if aspirated into the lung, and those that are volatile can cause acute CNS effects and/or ocular and respiratory irritation at exposure levels exceeding occupational recommendations. Otherwise, there are few toxicologically important effects. The exceptions, n-hexane and naphthalene, have unique toxicological properties

Animal studies:
No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) was observed in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar
naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of a alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human.
Pyrazole is teratogenic in rats producing high levels of ocular and urinary bladder anomalies.
Pyrazole produces adrenal necrosis in experimental animals.

Titan Pinoxaden 100 EC (Titan Pinoxaden 100EC Herbicide)

TOXICITY	IRRITATION
Not Available	Not Available

solvent naphtha petroleum, heavy aromatic

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye (Rodent - rabbit): 100uL/24H - Moderate
Inhalation (Rat) LC50: >0.003 mg/L4h[1]	Eye: adverse effect observed (irritating)[1]
Oral (Rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Skin (Rodent - rabbit): 500uL/24H - Mild
	Skin (Rodent - rabbit): 500uL/24H - Moderate
	Skin: adverse effect observed (irritating)[1]
	Skin: no adverse effect observed (not irritating)[1]

acetophenone

TOXICITY	IRRITATION
Dermal (rabbit) LD50: 1760 mg/kg[2]	Eye (Rodent - rabbit): 750ug - Severe
Inhalation (Mouse) LC50: 1.2 mg/L4h[2]	Eye: no adverse effect observed (not irritating)[1]
Oral (Mouse) LD50; 740 mg/kg[2]	Skin (Rodent - rabbit): 10mg/24H
	Skin (Rodent - rabbit): 515mg - Mild
	Skin: no adverse effect observed (not irritating)[1]

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N-methyl-2-pyrrolidone	**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
		Eye: adverse effect observed (irritating)[1]
		Skin: adverse effect observed (irritating)[1]
pinoxaden	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[2]	Not Available
	Inhalation (Rat) LC50: >5 mg/L4h[2]	
	Oral (Rat) LD50: 3129 mg/kg[2]	
tristyrylphenol, ethoxylated	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[2]	Not Available
	Oral (Rat) LD50: 5000 mg/kg[2]	
cloquintocet-mexyl	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: >0.935 mg/L4h[2]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 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 Pinoxaden 100 EC (Titan Pinoxaden 100EC 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.
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 Proluria.
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
Data demonstrate that during inhalation exposure,aromatic hydrocarbons undergo substantial partitioning into adipose tissues. Following cessation of exposure, the level of aromatic hydrocarbons in body fats rapidly declines. Thus, the aromatic hydrocarbons are unlikely to bioaccumulate in the body. Selective partitioning of the aromatic hydrocarbons into the non-adipose tissues is unlikely. No data is available regarding distribution following dermal absorption. However, distribution following this route of exposure is likely to resemble the pattern occurring with inhalation exposure.
Aromatics hydrocarbons may undergo several different Phase I dealkylation, hydroxylation and oxidation reactions which may or may not be followed by Phase II conjugation to glycine, sulfation or glucuronidation. However, the major predominant biotransformation pathway is typical of that of the alkylbenzenes and consists of: (1) oxidation of one of the alkyl groups to an alcohol moiety; (2) oxidation of the hydroxyl group to a carboxylic acid; (3) the carboxylic acid is then conjugated with glycine to form a hippuric acid. The minor metabolites can be expected to consist of a complex mixture of isomeric triphenols, the sulfate and glucuronide conjugates of dimethylbenzyl alcohols, dimethylbenzoic acids and dimethylhippuric acids. Consistent with the low propensity for bioaccumulation of aromatic hydrocarbons, these substances are likely to be significant inducers of their own metabolism.
The predominant route of excretion of aromatic hydrocarbons following inhalation exposure involves either exhalation of the unmetabolized parent compound, or urinary excretion of its metabolites. When oral administration occurs, there is little exhalation of unmetabolized these

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	hydrocarbons, presumably due to the first pass effect in the liver. Under these circumstances, urinary excretion of metabolites is the dominant route of excretion.
ACETOPHENONE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin. A member or analogue of a group of of aromatic substituted secondary alcohols, ketones, and related esters generally regarded as safe (GRAS) based, in part, on their rapid absorption, metabolic detoxication, and excretion in humans and other animals; their low level of flavor use; the wide margins of safety between the conservative estimates of intake and the no-observed-adverse effect levels determined from subchronic and chronic studies and the lack of significant genotoxic and mutagenic potential. Acute rat oral LD50 values have been reported for 17 of the 38 agents in this group. These values are in the range from 400 to greater than 10,000 mg/kg bw. However, the majority of these values are >1000 mg/kg bw, demonstrating that the oral acute toxicity of aromatic substituted secondary alcohols, ketones, and related esters is extremely low. Short-term toxicity studies have been performed for a representative number (12 of 38) of aromatic substituted secondary alcohols, ketones, and related esters. The results of these studies provided no-observable adverse effect levels (NOAELs) that are at least 1000 times (most 100,000–1,000,000 times) the daily per capita intake (‘‘eaters only) of the respective substances for their intended use as flavoring ingredients. Predominantly negative results are reported in in vitro genotoxicity assays for aromatic ketones, secondary alcohols, and related esters. The most studied substance, benzoin, is also the one exhibiting the most positive results in in vitro assays. . The absence of any consistent evidence of in vivo genotoxicity or carcinogenicity of benzoin in rats and mice supports the conclusion that benzoin exhibits a low potential for genotoxicity. Results of in vivo genotoxicity assays were predominantly negative Simple aromatic ketones and aromatic secondary alcohols have been shown to be rapidly absorbed from the gut, metabolised by the liver and excreted primarily in the urine, and to a very minor extent, in the faeces. The side-chain length of the aromatic ketone or secondary alcohol does not significantly affect metabolism. The ketone and alcohol are interconvertible. In major metabolic pathways, the ketone is stereoselectively reduced to the corresponding alcohol that is subsequently excreted as the glucuronic acid conjugate. If the alkyl chain is even numbered, the ketone may undergo oxidation and cleavage to yield a phenylacetic acid derivative or, if the alkyl chain is odd numbered, oxidative cleavage yields mainly a benzoic acid derivative. The acids are excreted almost exclusively as glycine conjugates Hydrolysis of esters occurs in all animals leading to the corresponding alcohols and carboxylic acids. Aromatic esters are hydrolysed in vivo through the catalytic activity of carboxylesterases, the most important of which are the A-esterases Hydrolysis of alpha-methylbenzyl esters yields alpha-methylbenzyl alcohol and simple aliphatic carboxylic acids. In vitro hydrolysis of structurally related benzyl esters (benzyl acetate, benzyl 2-methylbutanoate, benzyl cinnamate, and benzyl phenylacetate) in simulated intestinal fluid containing pancreatin indicates that significant ester hydrolysis is expected prior to absorption. After absorption, rapid in vivo hydrolysis is also expected in the blood and liver. Flavor and Extract Manufacturers' Association (FEMA)
N-METHYL-2-PYRROLIDONE	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.
PINOXADEN	Sensitisation Did not cause sensitisation on laboratory animals. Chronic exposure Did not show carcinogenic, teratogenic or mutagenic effects in animal experiments. * Sigma Aldrich SDS Acute studies Pinoxaden had low acute oral toxicity in rats, with a LD50 of greater than 5000 mg/kg (one death). In rats it also had low dermal toxicity of LD50 greater than 2000 mg/kg (no deaths) and a low inhalation toxicity of LC50 greater than 5220 mg/m3 (no deaths). It was a severe eye irritant but did not cause skin irritation in rabbits and was not a skin sensitiser in guinea pigs. Short term studies Rats received daily dermal applications of 0, 10, 100 or 1000 mg/kg of bw pinoxaden for six hours daily for 28 days. There were no treatment-related effects observed. Rats were dosed by oral gavage with pinoxaden at 0, 300, 600 or 1000 mg/kg bw/d for 28 days. At 1000 mg/kg bw/d, body weight gain was reduced in males without changes to food consumption. Higher neutrophil and lymphocyte counts were observed in males at 1000 mg/kg bw/d. At a dosage rate of 600 mg/kg bw/d and above, plasma urea and creatinine levels were increased in males, while increased inorganic phosphate and decreased chloride levels were seen in both sexes. Increased bilirubin levels were observed in males at 1000 mg/kg bw/d. Urinary volume and ketones levels were increased in both sexes at 600 mg/kg bw/d and above. Higher kidney and liver weights were observed in both sexes at 600 mg/kg bw/d and above. Increased incidence and severity of renal tubular atrophy and dilatation at 600 mg/kg bw/d was seen in both sexes. Single cell necrosis was also observed in three quarters of males at 1000 mg/kg bw/d, but not in females. The NOEL was 300 mg/kg bw/d. In a dose range finding study, two dogs were treated orally with pinoxaden at various concentrations ranging from 125?500 mg/kg bw/d for up to 36 days. Lower food consumption and vomiting were observed at 325, 400 and 500 mg/kg bw/d. At 250 mg/kg bw/d, no clinical signs were seen in either animal. Dogs were dosed with pinoxaden in gelatine capsules at 0, 250, 500 or 1000 mg/kg bw/d for 28 days. The only notable observation was a high incidence of vomit and food regurgitation at the dosage rate of 500 and 1000 mg/kg bw/d. The NOEL was 1000 mg/kg bw/d, which was the highest dose tested. Dogs received pinoxaden orally at 0, 25, 100 or 250 mg/kg bw/d for 28 days. There were no treatment-related effects observed other than loose stools in animals at 100 and 250 mg/kg bw/d. Subchronic studies In a dose range finding study, mice received pinoxaden orally by gavage at 0, 10, 100, 400, 700 or 1000 mg/kg bw/d for 13 weeks. A slight anaemia was seen in females at 400 mg/kg bw/d and above. Platelet counts were significantly increased at 1000 mg/kg bw/d in females. Increases in liver weights were observed in both sexes at 400 mg/kg bw/d and above, but liver histology was not affected. Microscopic examinations revealed a high incidence and grading of tubular basophilia at 700 mg/kg bw/d and above in males. The NOEL was 100 mg/kg bw/d for both sexes based on mild anaemia in females, increased liver weights in both sexes at 400 mg/kg bw/d and above and on an increased incidence of tubular basophilia in males at 700 mg/kg bw/d and above. Rats received pinoxaden orally by gavage at 0, 3, 10, 30, 100 or 300 mg/kg bw/d for 13 weeks. Both sexes had elevated serum inorganic phosphate at 30 mg/kg bw/d and above, creatinine and urea at 300 mg/kg bw/d, ketones in urine at 100 and 300 mg/kg bw/d, urine volume at 300 mg/kg bw/d and acidity at 100 and 300 mg/kg bw/d. Serum levels of chloride were decreased at 100 and 300 mg/kg bw/d. Liver weights were increased at 300 mg/kg bw/d in both sexes. The NOEL was 10 mg/kg bw/d for both sexes based on increased levels of inorganic phosphate at 30 mg/kg bw/d and on changes in serum chemistry, urinalysis and liver weights at higher doses. Rats were dosed with pinoxaden at 0, 150, 1000, 5000 or 10000 ppm in the diet for four weeks for the satellite group and for three months for the main group. At 10000 ppm, decreased RBC counts, haemoglobin concentration and haematocrit were observed in females of the main group. At 5000 and 10000 ppm, serum creatinine levels were increased in males of the satellite group and in females of the main group. Total cholesterol levels were lower at 10000 ppm in both sexes of the satellite group and at 5000 ppm and above in the main group.

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Females of the main group had lower urinary volume and pH at 10000 ppm. Increased incidences of mild cortical basophilia with tubular dilation and/or atrophy were noted in both sexes at 10000 ppm. The NOEL was 1000 ppm (approximately 100 mg/kg bw/d). Dogs were dosed with pinoxaden in gelatine capsules at 0, 25, 100, 250 or 500 mg/kg bw/d for 90 days. All females treated with 500 mg/kg bw/d were terminated at week five due to excessive loss of bodyweight. Reduced bodyweight gains with a slight reduction in food consumption were observed in females at 250 mg/kg bw/d. Total serum protein was lower in males at 500 mg/kg bw/d throughout the study and in females at 250 mg/kg bw/d at week 13. The NOEL was 100 mg/kg bw/d. Chronic studies Mice Mice were dosed by oral gavage with pinoxaden at 0, 5, 40, 300 or 750 mg/kg bw/d for 18 months. Bodyweight gains were reduced in both sexes at 750 mg/kg bw/d and in females at 300 mg/kg bw/d without changes to food consumption. Increased platelet counts were seen in males at 300 and 750 mg/kg bw/d. Increased relative liver weights were observed in both sexes at 300 mg/kg bw/d and above, while relative kidney weights were increased in females at 750 mg/kg bw/d. Increased incidences of foamy outflow from the bronchi were observed in both sexes at 300 mg/kg bw/d and above. At 300 and 700 mg/kg bw/d, higher incidences of epithelial thickening in the small intestine and lower incidences of cytoplasmic vacuolation in the epididymides were observed in males at 300 and 700 mg/kg bw/d. The incidences and nature of tumours were comparable across all treated and control groups. The NOEL was 40 mg/kg bw/d. In a chronic toxicity and carcinogenic study, rats received pinoxaden orally by gavage at 0, 1, 10, 100, 250 or 500 mg/kg bw/d for 105 weeks. Mortality rates were high in males at 500 mg/kg bw/d during the first year and this group was terminated in week 61. Bodyweight gains were significantly reduced at 500 mg/kg bw/d, and to a lesser extent at 250 mg/kg bw/d in both sexes during the first 52 weeks of the study. At weeks 27 and 53, both sexes had a slight anaemia at 250 and 500 mg/kg bw/d, elevated serum urea and creatinine and decreased serum sodium and chloride at 500 mg/kg bw/d. Increased serum inorganic phosphate was observed at 100 mg/kg bw/d and above in both sexes at weeks 53 and 79. Serum potassium was higher at 250 and 500 mg/kg bw/d in males and at 100 mg/kg bw/d and above in females at week 53, while ketones in urine were higher in males at 250 and 500 mg/kg bw/d at week 27 only and in females throughout the course of the study. At week 105, females had lower haemoglobin and haematocrit at 500 mg/kg bw/d, higher inorganic phosphate at 250 and 500 mg/kg bw/d and higher potassium at 500 mg/kg bw/d compared to control. Serum sodium and chloride were lower in males at 250 mg/kg bw/d and in females at 100 mg/kg bw/d and above. Liver weights were increased in both sexes at 250 and 500 mg/kg bw/d at weeks 53 and 105 for females and at week 53 for males, but was not associated with any histological changes. Higher incidences of kidneys with granulated surface and chronic nephropathy were observed, particularly in males, at 250 and 500 mg/kg bw/d at weeks 53 and 105. In females, the incidence and grading of tubular vacuolation were increased at 100, 250 and 500 mg/kg bw/d at 105 weeks. The incidence and nature of tumours was comparable across all treated and control groups. The NOEL was 10 mg/kg bw/d based on increased inorganic phosphate levels at 100 mg/kg bw/d and on changes in haematology, serum chemistry, urinalysis, liver weights and histopathological evidence of renal toxicity at higher doses. Reproduction study Rats received pinoxaden orally by gavage at 0, 10, 50, 250 or 500 mg/kg bw/d throughout two generations. There were no effects of treatment on parent mortality, clinical signs, reproductive performance and food consumption. Liver weights were higher in first generation male parents at 250 and 500 mg/kg bw/d and in female parents at 50 mg/kg bw/d and above. Kidney weights were higher in first generation parental males at 250 and 500 mg/kg bw/d and in second generation males at 50 mg/kg bw/d and above. Microscopic findings revealed higher incidences of chronic nephropathy and tubular atrophy in both sexes at 500 mg/kg bw/d compared to the control groups. At 500 mg/kg bw/d, bodyweight gains of pups from birth to day seven were reduced (10 to 15 per cent) during the first half of the lactation period but were similar thereafter. There were no other treatment-related effects on number of pups delivered, sex ratio, clinical signs and microscopic findings. The NOEL was 10 mg/kg bw/d for parents based on increased liver and kidney weights at higher doses and 250 mg/kg bw/d for offspring based on reduced bodyweight gains at 500 mg/kg bw/d. Developmental studies Pregnant rats were administered pinoxaden orally by gavage at 0, 3, 30, 300 or 800 mg/kg bw/d from days six to 20 of gestation. One animal at 800 mg/kg was sacrificed in a moribund condition on day 17 of gestation. From days six to 21, mean maternal body weight gain and daily food consumption were significantly reduced at 800 mg/kg bw/d, and to a lesser extent at 300 mg/kg bw/d. Implantation sites, post-implantation losses, foetal viability and foetal sex ratios were similar in control and treated groups. At 800 mg/kg bw/d, mean foetal bodyweight was eight per cent lower and incidences of foetal shortening of innominate artery, accessory lobulets of the liver and renal pelvic dilatation were slightly higher. No foetal malformations were observed, but incidences of incomplete ossification of cranial bones as well as unossified and incomplete ossification of digits were higher at 300 and 800 mg/kg bw/d. Lower foetal bodyweight and increased incidences of foetal visceral and skeletal anomalies were likely to be secondary to decreased maternal nutritional status and not due to teratogenic effects by pinoxaden. The NOEL for both maternal and foetal toxicity was 30 mg/kg bw/d, based on reduced body weight gain and food consumption (maternal) and on delayed ossification of cranial bones and digits (foetal) at higher doses. The NOEL for developmental toxicity was 800 mg/kg bw/d, the highest dose tested. Female rabbits were artificially inseminated with semen from only one male donor. Pregnant rabbits were dosed by oral gavage with pinoxaden at 0, 30, 150, 300, 700 or 1000 mg/kg bw/d from days 7?28 of gestation. Because of severe toxic effects at 300, 700 and 1000 mg/kg bw/d, these groups were terminated. At 150 mg/kg bw/d, one animal was sacrificed due to moribund condition. Bodyweight gains were reduced but food consumption was only slightly reduced. At 150 mg/kg bw/d, increased incidences of early resorption, implantation loss, lower number of live foetuses and reduced foetal weight were observed. The NOEL was 30 mg/kg bw/d for both maternal and foetal toxicity. Female rabbits were artificially inseminated with semen from only one male donor. Pregnant rabbits were dosed by oral gavage with pinoxaden at 0, 3, 10, 30 or 100 mg/kg bw/d from days 7?28 of gestation. Maternal bodyweight gains and food consumption were reduced at 100 mg/kg bw/d. At this dose, foetal body weight was reduced and higher incidences of malformed diaphragm were seen. To determine if the higher incidences of malformed diaphragm were reproducible, pregnant female rabbits (artificially inseminated with semen from only one male donor) were dosed by oral gavage with pinoxaden at 0 or 100 mg/kg bw/d from days 7-28 of gestation. Maternal bodyweight gains and food consumption were reduced at 100 mg/kg bw/d. There were no other abnormal findings. Therefore, the NOEL was 30 mg/kg bw/d for maternal toxicity and 100 mg/kg bw/d for developmental toxicity. Pregnant rabbits (artificially inseminated with semen from multiple male donors) were dosed by oral gavage with pinoxaden at 0 or 100 mg/kg bw/d from days 7-28 of gestation. At 100 mg/kg bw/d, three out of 12 animals died and another two were sacrificed due to moribund condition. Maternal bodyweight gains were reduced without changes to food consumption at 100 mg/kg bw/d. Also at this dose, increased incidences of early resorption, post-implantation loss and lower number of live foetuses were seen. There were no foetal abnormal findings. Pregnant rabbits (artificially inseminated with semen from only one male donor) were dosed by oral gavage with pinoxaden at 0, 3, 10, 30 or 100 mg/kg bw/d from days 7-28 of gestation. At 100 mg/kg bw/d, four out of 24 animals died and another two were sacrificed due to moribund condition. Maternal bodyweight gains were reduced at 30 mg/kg bw/d and above. At 100 mg/kg bw/d, increased incidences of early resorption, post-implantation loss and lower number of live foetuses were seen. There were no abnormal foetal findings. The NOEL was 10 mg/kg bw/d for maternal toxicity and 100 mg/kg bw/d for developmental toxicity. Genotoxicity studies Pinoxaden did not induce mutations in S. typhimurium or E. coli, nor did it induce micronuclei in the bone marrow of mice administered single oral doses of 500, 1000 or 2000 mg/kg. Pinoxaden was not mutagenic to mouse lymphoma cells. It was positive for chromosomal aberrations in Chinese hamster lung cells, but was negative for unscheduled DNA synthesis both in vitro and in vivo. It was concluded that pinoxaden was unlikely to be genotoxic in vivo. Neurotoxicity studies Rats were dosed with a single dose of pinoxaden by oral gavage at 0, 100, 500 or 2000 mg/kg bw and were observed for the following 14 days. There were no changes in the battery of functional observations or in motor activity that could be attributed to treatment. Rats were administered pinoxaden by oral gavage at 0, 10, 100 or 500 mg/kg bw/d for 13 weeks. There were no changes in the battery of functional observations or in motor activity that could be attributed to treatment. ** APMVA Evaluation May 2006, Australia

A member or an analogue of a group of pyrazine derivatives generally considered as safe (GRAS) based in part on their extremely low aroma thresholds and their self-limiting properties in food: their rapid absorption, metabolic detoxification and excretion in humans; the wide margins of safety between the conservative estimates of intake and the no-adverse-effect levels determined from subchronic and chronic studies and the lack of genotoxic and mutagenic potential, This evidence is supported by the intake of pyrazine derivatives as natural components of traditional foods is much greater than their intake as intentionally added flavouring substances.

Acute oral rat LD50 values are available for 17 of 41 pyrazines included in this group and indicate varying levels of toxicity, in rats, ranging from 158 mg/kg for a thiol derivative to greater than 4000 mg/kg. However almost all of the LD50 values are in the narrower range of approximately 500-2000 mg/kg. All available mouse acute oral LD50s are 2000 mg/kg or greater.

Subchronic effects in six studies showed slight to moderate decrease in growth rate and efficiency of food utilisation due to palability problems.

Unsubstituted and alkyl-substituted pyrazine derivatives all induced significant percentages of chromosome aberrations (breaks and exchanges) in metaphase plates in CHO cells with and without S9 activation. However these results remain problematic for a number of reasons.

There seems to be no evidence of reproductive or developmental effects.

Pyrazine derivatives participate in common pathways of metabolic detoxication principally involving oxidation of side-chain alkyl or oxygenated functional groups and hydroxylation of the ring. When sulfur is present in the side-chain, oxidation to sulfoxides and then sulfones is catalysed by three enzyme systems.

Pyrazine is a weaker base (pKb=13.4) than pyridine)pKb=8.8), pyrimidine (pKb=12.7) or pyrazidine (pKb=11.7). At intestinal pH (5-7), absorption of weak amine bases such as pyrazine derivatives, is optimal. In human and laboratory rodents, orally administered substituted

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	pyrazines are rapidly absorbed from the gastrointestinal tract and excreted. Flavor and Extract Manufacturers' Association (FEMA)
TRISTYRYLPHENOL, ETHOXYLATED	Rhodia Canada Mutagenicity Assay: Salmonella, Ames test; negative. For tristyrylphenol ethoxylates (CAS RNs: 70559-25-0, 99734-09-5, 90093-37-1, 105362-40-1, 119432-41-6, 163436-84-8, 396089-99-9) **Acute toxicity:** Based on available animal studies conducted on several members of the group or by suitable analogy with structurally related substances, the tristyrylphenol ethoxylates exhibit low acute oral toxicity and low to moderate subchronic oral toxicity to terrestrial species. **Repeat dose toxicity:** In a 90-day study of beagle dogs, using CAS RN 105362-40-1, the predominant pathological changes noted were: hyperaemia of the stomach and intestines and hepatic (liver) degenerative changes. Hepatic pathological changes, significantly increased mean liver weights/ ratios were noted in the mid to high dose groups (500, 1000 mg/kg/day) and food consumption was significantly increased in the high dose group. In a 90-day oral study with Sprague Dawley rats, using CAS RN: 105362-40-1, mean-liver weights were increased in high-dose males (4000 mg/kg/day) and all dose levels in females and mean kidney weights were increased in high-dose females. There are no apparent morphological changes to explain organ weight differences. Mean liver and kidney to final body weight ratios were increased in all dose levels except low-dose males (500 mg/kg/day). Additionally, effects in thyroid and pituitary glands were observed. Gavage administration of this chemical produced increased thyroid activity characterised by increased follicular epithelial height, decreased follicle size and altered colloid. These alterations are typical of physiological hypertrophy due to hormonal stimulation - this may indicate a compound-induced interference with the feedback control of hormone synthesis between the pituitary and thyroid. **Developmental toxicity:** In a developmental study with CAS RN: 119432-41-6, administered by gavage to Sprague Dawley rats, slight maternal toxicity was seen at 300 mg/kg/day (expressed as reduced weight gain, reduced food consumption and clinical signs of toxicity) and slight foetotoxicity at 1000 mg/kg/day (expressed as reduced skeletal ossification in the foot phalanges in the absence of reduced foetal weight). Thee were no treatment-related increased incidence of malformations at any dose level;. The NOEL was 100 mg/kg/day for maternal toxicity and the NOEL was 300 mg/kg/day for developmental toxicity. **Genotoxicity:** In an Ames study with CAS RN 105362-40-1), there was no mutagenic activity with Salmonella typhimurium strains both with and without activation. Results of acceptable mutagenicity studies, including a chromosomal aberration assay in Chinese hamster ovary cells CHO) and a test for unscheduled DNA synthesis in primary hepatocyte cultures were negative.
CLOQUINTOCET-MEXYL	*Merck Index NOEL (2 y) for rats 4 mg/kg b.w. daily; (18 mo) for mice 106.5 mg/kg b.w. daily; (1 y) 44 mg/kg b.w. daily ADI 0.04 mg/kg b.w. Toxicity Class WHO (a.i.) III Cloquintocet-mexyl technical has been extensively tested and no evidence of mutagenic, carcinogenic, teratogenic, or reproductive effects was obtained.
Titan Pinoxaden 100 EC (Titan Pinoxaden 100EC Herbicide) & CLOQUINTOCET-MEXYL	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 Pinoxaden 100 EC (Titan Pinoxaden 100EC Herbicide) & N-METHYL-2-PYRROLIDONE & PINOXADEN & TRISTYRYLPHENOL, ETHOXYLATED	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 Pinoxaden 100 EC (Titan Pinoxaden 100EC Herbicide) & PINOXADEN	ACCase plays a vital role in mammalian systems for fatty acid biosynthesis, the enzyme existing in two isozymic forms.This enzyme is highly regulated in the brain and non-neural tissue. None of these forms are inhibited by cyclohexanediones or aryloxyphenoxypropionates. Although these herbicides affect common target sites in plants and mammals, they have no effect on ACCase in mammals. Phenylpyrazole insecticides function by blocking glutamate-activated chloride channels in insects. Mammals do not have this type of chloride channel, making them much less susceptible to its effects. However, they do have the capacity to disrupt epithelial cells in the human intestine and adversely impact human health The disruption of the epithelial barrier was attributed to severe ATP depletion independent of cell viability, as revealed by lactic dehydrogenase (LDH) release. The origin of energetic metabolism failure was investigated and revealed a transient enhancement of tetrazolium salt reduction and an increase in lactate production by Caco-2 cells, suggesting an increase in glucose metabolism by pesticides. Cellular symptoms observed in these experiments leads to a hypothesis that phenylpyrazole insecticides interact with mitochondria. Inhibitors of acetyl CoA carboxylase, the target enzyme of certain herbicides, have the capacity, in mammals, to alter blood lipid levels. In the male rat, a reduction ($p < 0.05$) in blood cholesterol and total lipids in a chronic study may be a reflection of inhibition of this enzyme. However, in the female mouse, there was an increase in blood cholesterol at the highest dose tested, in a subchronic study . Male mice in this study showed an increase in total lipids at the two highest doses. It is therefore possible that many of the effects reported in acute, subchronic and chronic studies are manifestations of a compromise of normal liver function. The inhibition of fatty acid biosynthesis, in the liver. may account for the majority of the effects observed. However, increases in liver weight, seen in acute and sub-chronic studies, and decreases in liver weight, which are seen in chronic studies, alone, do not necessarily reflect an adverse effect. This is because liver weight changes have often been found to be reversible, in subchronic studies following the discontinuation of dosing, or through adaptation mechanisms, with the continued dietary intake of fenoxaprop-ethyl. in chronic studies.
Titan Pinoxaden 100 EC (Titan Pinoxaden 100EC Herbicide) & TRISTYRYLPHENOL, ETHOXYLATED	No significant acute toxicological data identified in literature search.
Titan Pinoxaden 100 EC (Titan Pinoxaden 100EC Herbicide) & 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.

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	Testicular degeneration and atrophy in males and thymic atrophy in females were observed at these dose levels. The no-observed-adverse-effect level (NOAEL) was 429 mg/kg body weight in males and 1548 mg/kg body weight in females. In a 28-day intubation study in rats, a dose-dependent increase in relative liver and kidney weights and a decrease in lymphocyte count in both sexes were observed at 1028 mg/kg body weight. The NOAEL in this study was 514 mg/kg body weight. In another rat study, daily dietary intake for 90 days caused decreased body weights at doses of 433 and 565 mg/kg body weight in males and females, respectively. There were also neurobehavioural effects at these dose levels. The NOAELs in males and females were 169 and 217 mg/kg body weight, respectively. The toxicity profile after exposure to airborne NMP depends strongly on the ratio of vapour to aerosol and on the area of exposure (i.e., head-only or whole-body exposure). Because of higher skin absorption for the aerosol, uptake is higher in animals exposed to aerosol than in those exposed to vapour at similar concentrations. Studies in female rats exposed head only to 1000 mg/m3 showed only minor nasal irritation, but massive mortality and severe effects on major organs were observed when the females were whole-body exposed to the same concentration of coarse droplets at high relative humidity. Several studies in rats following repeated exposure to NMP at concentrations between 100 and 1000 mg/m3 have shown systemic toxicity effects at the lower dose levels. In most of the studies, the effects were not observed after a 4-week observation period. In rats, exposure to 3000 mg NMP/m3 (head only) for 6 h/day, 5 days/week, for 13 weeks caused a decrease in body weight gain, an increase in erythrocytes, haemoglobin, haematocrit, and mean corpuscular volume, decreased absolute testis weight, and cell loss in the germinal epithelium of the testes. The NOAEL was 500 mg/m3. There are no data in humans after repeated-dose exposure. **Carcinogenicity:** NMP did not show any clear evidence for carcinogenicity in rats exposed to concentrations up to 400 mg/m3 in a long-term inhalation study. **Genotoxicity:** The mutagenic potential of NMP is weak. Only a slight increase in the number of revertants was observed when tested in a ***Salmonella*** assay with base-pair substitution strains. NMP has been shown to induce aneuploidy in yeast ***Saccharomyces cerevisiae*** cells. No investigations regarding mutagenicity in humans were available. **Reproductive toxicity:** In a two-generation reproduction study in rats, whole-body exposure of both males and females to 478 mg/m3 of NMP vapour for 6 h/day, 7 days/week, for a minimum of 100 days (pre-mating, mating, gestation, and lactation periods) resulted in a 7% decrease in fetal weight in the F1 offspring. A 4-11% transient, non-dose-dependent decrease was observed in the average pup weight at all exposure levels tested (41, 206, and 478 mg/m3). **Developmental toxicity:** When NMP was administered dermally, developmental toxicity was registered in rats at 750 mg/kg body weight. The observed effects were increased preimplantation losses, decreased fetal weights, and delayed ossification. The NOAEL for both developmental effects and maternal toxicity (decreased body weight gain) was 237 mg/kg body weight. Inhalation studies in rats (whole-body exposure) demonstrated developmental toxicity as increased preimplantation loss without significant effect on implantation rate or number of live fetuses at 680 mg/m3 and behavioural developmental toxicity at 622 mg/m3. In an inhalation study (whole-body exposure), the NOAEL for maternal effects was 100 mg/m3, and the NOAEL for developmental effects was 360 mg/m3. A tolerable inhalation concentration, 0.3 mg/m3, based on mortality and organ damage, is expected to be protective against any possible reproductive toxicity. Similarly, an oral tolerable intake of 0.6 mg/kg body weight per day, based on a 90-day study, is expected to provide adequate protection against possible reproductive effects. Because of non-existent data on the exposure of the general population and very limited information on occupational exposure, no meaningful risk characterisation can be performed
Titan Pinoxaden 100 EC (Titan Pinoxaden 100EC Herbicide) & SOLVENT NAPHTHA PETROLEUM, HEAVY AROMATIC	Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length,with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo-paraffins. The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption,is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver. For petroleum: This product contains benzene, which can cause acute myeloid leukaemia, and n-hexane, which can be metabolized to compounds which are toxic to the nervous system. This product contains toluene, and animal studies suggest high concentrations of toluene lead to hearing loss. This product contains ethyl benzene and naphthalene, from which animal testing shows evidence of tumour formation. Cancer-causing potential: Animal testing shows inhaling petroleum causes tumours of the liver and kidney; these are however not considered to be relevant in humans. Mutation-causing potential: Most studies involving gasoline have returned negative results regarding the potential to cause mutations, including all recent studies in living human subjects (such as in petrol service station attendants). Reproductive toxicity: Animal studies show that high concentrations of toluene (>0.1%) can cause developmental effects such as lower birth weight and developmental toxicity to the nervous system of the foetus. Other studies show no adverse effects on the foetus. Human effects: Prolonged or repeated contact may cause defatting of the skin which can lead to skin inflammation and may make the skin more susceptible to irritation and penetration by other materials. Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable.

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

	Endpoint	Test Duration (hr)	Species	Value	Source
Titan Pinoxaden 100 EC (Titan Pinoxaden 100EC Herbicide)	Not Available	Not Available	Not Available	Not Available	Not Available

Continued...

solvent naphtha petroleum, heavy aromatic	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	<1mg/l	1
	EC50	48h	Crustacea	0.95mg/l	1
	EC50	96h	Algae or other aquatic plants	11.7mg/l	2
	EC50(ECx)	48h	Crustacea	0.95mg/l	1
	LC50	96h	Fish	0.58mg/l	2
acetophenone	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	1mg/L	4
	EC50(ECx)	72h	Algae or other aquatic plants	1mg/L	4
	LC50	96h	Fish	155mg/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
pinoxaden	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	18.56mg/L	4
	EC50	48h	Crustacea	52mg/l	Not Available
	EC50	96h	Algae or other aquatic plants	2.436-5.916mg/L	4
	EC50(ECx)	48h	Crustacea	52mg/l	Not Available
	LC50	96h	Fish	10.3mg/l	Not Available
tristyrylphenol, ethoxylated	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96h	Fish	21mg/l	Not Available
cloquintocet-mexyl	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	0.42mg/l	2
	EC50	48h	Crustacea	>0.82mg/l	2
	EC50	96h	Algae or other aquatic plants	>0.24mg/l	2
	ErC50	72h	Algae or other aquatic plants	5.3mg/l	2
	NOEC(ECx)	504h	Crustacea	0.002mg/l	2
	LC50	96h	Fish	>0.97mg/l	2
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***				

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.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
acetophenone	LOW	LOW
N-methyl-2-pyrrolidone	LOW	LOW
cloquintocet-mexyl	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
solvent naphtha petroleum, heavy aromatic	LOW (BCF = 159)
acetophenone	LOW (LogKOW = 1.58)
N-methyl-2-pyrrolidone	LOW (BCF = 0.16)
cloquintocet-mexyl	HIGH (LogKOW = 5.672)

Mobility in soil

Ingredient	Mobility
acetophenone	LOW (Log KOC = 46.2)
N-methyl-2-pyrrolidone	LOW (Log KOC = 20.94)
cloquintocet-mexyl	LOW (Log KOC = 38070)

Continued...

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	•3Z

Land transport (ADG)

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

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

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	3082
14.2. **UN proper shipping name**	Environmentally hazardous substance, liquid, n.o.s. (contains solvent naphtha petroleum, heavy aromatic)
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 9 ICAO / IATA Subsidiary Hazard: Not Applicable ERG Code: 9L
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: A97 A158 A197 A215

Continued...

Cargo Only Packing Instructions	964
Cargo Only Maximum Qty / Pack	450 L
Passenger and Cargo Packing Instructions	964
Passenger and Cargo Maximum Qty / Pack	450 L
Passenger and Cargo Limited Quantity Packing Instructions	Y964
Passenger and Cargo Limited Maximum Qty / Pack	30 kg G

Sea transport (IMDG-Code / GGVSee)

14.1. **UN number**	3082
14.2. **UN proper shipping name**	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains solvent naphtha petroleum, heavy aromatic)
14.3. **Transport hazard class(es)**	IMDG Class: 9 IMDG Subsidiary Hazard: Not Applicable
14.4. **Packing group**	III
14.5 **Environmental hazard**	Marine Pollutant
14.6. **Special precautions for user**	EMS Number: F-A, S-F Special provisions: 274 335 375 969 Limited Quantities: 5 L

14.7. Maritime transport in bulk according to IMO instruments

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

Not Applicable

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

Product name	Group
solvent naphtha petroleum, heavy aromatic	Not Applicable
acetophenone	Not Applicable
N-methyl-2-pyrrolidone	Not Applicable
pinoxaden	Not Applicable
tristyrylphenol, ethoxylated	Not Applicable
cloquintocet-mexyl	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
solvent naphtha petroleum, heavy aromatic	Not Applicable
acetophenone	Not Applicable
N-methyl-2-pyrrolidone	Not Applicable
pinoxaden	Not Applicable
tristyrylphenol, ethoxylated	Not Applicable
cloquintocet-mexyl	Not Applicable

SECTION 15 Regulatory information

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

solvent naphtha petroleum, heavy aromatic is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

acetophenone 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
Australian Inventory of Industrial Chemicals (AIIC)

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

pinoxaden 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

Continued...

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

tristyrylphenol, ethoxylated is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

cloquintocet-mexyl is found on the following regulatory lists

Not Applicable

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (pinoxaden; cloquintocet-mexyl)
Canada - DSL	No (pinoxaden; cloquintocet-mexyl)
Canada - NDSL	No (solvent naphtha petroleum, heavy aromatic; acetophenone; N-methyl-2-pyrrolidone; pinoxaden; cloquintocet-mexyl)
China - IECSC	No (pinoxaden)
Europe - EINEC / ELINCS / NLP	No (pinoxaden; tristyrylphenol, ethoxylated; cloquintocet-mexyl)
Japan - ENCS	No (pinoxaden; cloquintocet-mexyl)
Korea - KECI	No (pinoxaden; cloquintocet-mexyl)
New Zealand - NZIoC	Yes
Philippines - PICCS	No (pinoxaden; cloquintocet-mexyl)
USA - TSCA	TSCA Inventory 'Active' substance(s) (solvent naphtha petroleum, heavy aromatic; acetophenone; N-methyl-2-pyrrolidone; tristyrylphenol, ethoxylated; cloquintocet-mexyl); No (pinoxaden)
Taiwan - TCSI	No (pinoxaden)
Mexico - INSQ	No (pinoxaden; tristyrylphenol, ethoxylated; cloquintocet-mexyl)
Vietnam - NCI	Yes
Russia - FBEPH	No (pinoxaden; tristyrylphenol, ethoxylated; cloquintocet-mexyl)
UAE - Control List (Banned/Restricted Substances)	No (solvent naphtha petroleum, heavy aromatic; acetophenone; N-methyl-2-pyrrolidone; tristyrylphenol, ethoxylated; cloquintocet-mexyl)
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	02/06/2026
Initial Date	25/03/2026

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	07/04/2026	Toxicological information - Acute Health (eye), Toxicological information - Acute Health (inhaled), Toxicological information - Acute Health (skin), Toxicological information - Acute Health (swallowed), Physical and chemical properties - Appearance, Toxicological information - Chronic Health, Hazards identification - Classification, Disposal considerations - Disposal, Ecological Information - Environmental, First Aid measures - First Aid (eye), First Aid measures - First Aid (swallowed), Exposure controls / personal protection - Personal Protection (hands/feet), Identification of the substance / mixture and of the company / undertaking - Supplier Information, Identification of the substance / mixture and of the company / undertaking - Use, Name

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

Continued...

- IMSBC: International Maritime Solid Bulk Cargoes Code
- IGC: International Gas Carrier Code
- IBC: International Bulk Chemical Code

- AIIC: Australian Inventory of Industrial Chemicals
- DSL: Domestic Substances List
- NDSL: Non-Domestic Substances List
- IECSC: Inventory of Existing Chemical Substance in China
- EINECS: European INventory of Existing Commercial chemical Substances
- ELINCS: European List of Notified Chemical Substances
- NLP: No-Longer Polymers
- ENCS: Existing and New Chemical Substances Inventory
- KECI: Korea Existing Chemicals Inventory
- NZIoC: New Zealand Inventory of Chemicals
- PICCS: Philippine Inventory of Chemicals and Chemical Substances
- TSCA: Toxic Substances Control Act
- TCSI: Taiwan Chemical Substance Inventory
- INSQ: Inventario Nacional de Sustancias Químicas
- NCI: National Chemical Inventory
- FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

end of SDS