Titan Dual Grain Treatment (Titan Dual Grain Treatment)

Titan Ag

Chemwatch Hazard Alert Code: 3

Chemwatch: **7655-370**
Version No: **3.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Initial Date: **19/03/2026**
Revision Date: **05/05/2026**
Print Date: **03/06/2026**
L.GHS.AUS.EN.E

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

Product Identifier

Product name	Titan Dual Grain Treatment (Titan Dual Grain Treatment)
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	ORGANOPHOSPHORUS PESTICIDE, LIQUID, TOXIC (contains fenitrothion)
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 insecticide for use as described on the product label. 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

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

Label elements

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

Hazard statement(s)

H302	Harmful if swallowed.
H312	Harmful in contact with skin.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H318	Causes serious eye damage.
H331	Toxic if inhaled.
H361fd	Suspected of damaging fertility. Suspected of damaging the unborn child.
H373	May cause damage to organs through prolonged or repeated exposure.
H410	Very toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P202	Do not handle until all safety precautions have been read and understood.
P260	Do not breathe mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P330	Rinse mouth.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

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

No further product hazard information.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
65733-16-6	6	methoprene
122-14-5	60	fenitrothion
Not Available	balance	Ingredients determined not to be hazardous
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available***	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If product comes in contact with skin: ▸ Contact a Poisons Information Centre or a doctor. ▸ **DO NOT** allow clothing wet with product to remain in contact with skin, strip all contaminated clothing including boots. ▸ Quickly wash affected areas vigorously with soap and water.

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	▸ **DO NOT** give anything by mouth to a patient showing signs of narcosis, i.e. losing consciousness. ▸ Give atropine if instructed. ▸ **DO NOT** delay, get to a doctor or hospital quickly.
Inhalation	▸ If spray mist, vapour are inhaled, remove from contaminated area. ▸ Contact a Poisons Information Centre or a doctor at once. ▸ Lay patient down in a clean area and strip any clothing wet with spray. ▸ 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. ▸ **DO NOT** give anything by mouth to a patient showing signs of narcosis, i.e. losing consciousness. ▸ Give atropine if instructed. ▸ Get to doctor or hospital quickly.
Ingestion	If swallowed: ▸ Contact a Poisons Information Centre or a doctor at once. ▸ If swallowed, activated charcoal may be advised. ▸ Give atropine if instructed. ▸ **REFER FOR MEDICAL ATTENTION WITHOUT DELAY.** ▸ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. ▸ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. ▸ Further action will be the responsibility of the medical specialist. ▸ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

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 fire fighting procedures suitable for surrounding area. ▸ **Do not** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire. ▸ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	▸ Combustible. ▸ Slight fire hazard when exposed to heat or flame. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke. ▸ Mists containing combustible materials may be explosive. Combustion products include: ▸ carbon dioxide (CO_2) nitrogen oxides (NO_x) phosphorus oxides (PO_x) sulfur oxides (SO_x) ▸ other pyrolysis products typical of burning organic material. May emit poisonous fumes.
HAZCHEM	2X

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

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

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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	1	throw	pitchfork	R,P, DGC, RT
foamed glass - pillow	2	shovel	shovel	R, W, P, DGC
sorbent clay - particulate	2	shovel	shovel	R, I, P
wood fibre - particulate	3	shovel	shovel	R,W, P, DGC
LAND SPILL - MEDIUM				
cross-linked polymer -particulate	1	blower	skiploader	R, W, SS
sorbent clay - particulate	2	blower	skiploader	R, I, P
polypropylene - particulate	2	blower	skiploader	R, SS, DGC
expanded mineral - particulate	3	blower	skiploader	R,I, W, P, DGC
wood fiber- particulate	3	blower	skiploader	R, W, P, DGC
polypropylene - mat	3	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
Slippery when spilt.

- **DO NOT** touch the spill material
- Clear area of personnel and move upwind.
- 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.
- Stop leak if safe to do so.
- Contain spill with sand, earth or vermiculite.
- Collect recoverable product into labelled containers for recycling.
- Neutralise/decontaminate residue (see Section 13 for specific agent).
- Collect solid residues and seal in labelled drums for disposal.
- Wash area and prevent runoff into drains.
- After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.
- If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling

- **DO NOT** allow clothing wet with material to stay in contact with skin

Australian Standard (AS2639) and the National Institute of Health (USA) recommends that the preparation of injectable antineoplastic drugs should be performed in a Class II laminar flow biological safety cabinet and that personnel preparing drugs of this class should wear appropriate personal protective gear. Emphasise controls on containment.

- 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.
- **DO NOT** allow material to come in direct contact with human skin or eyes.
- **DO NOT** allow material to come in contact with exposed food or food contact surfaces.
- Suitable PPE must be worn at all times.
- 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. Launder contaminated clothing before re-use.
- 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 are maintained.

Other information

Antineoplastics (cytotoxics):

- should be clearly identifiable to all personnel involved in their handling
- should be stored in impervious break-resistant containers
- should be stored in separate, clearly marked storage areas to minimise the risk of breakage, and to limit contamination in the event of leakage.

Spill kits should be available in storage areas.

- Store in original containers.
- Keep containers securely sealed.
- Store in a cool, dry, well-ventilated area.
- Store away from incompatible materials and foodstuff containers.
- Protect containers against physical damage and check regularly for leaks.
- Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container

- Glass container is suitable for laboratory quantities

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	▸ **DO NOT** use unlined steel containers ▸ Lined metal can, lined metal pail/ can. ▸ Plastic pail. ▸ Polyliner drum. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks. For low viscosity materials ▸ Drums and jerricans must be of the non-removable head type. ▸ Where a can is to be used as an inner package, the can must have a screwed enclosure. For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.): ▸ Removable head packaging; ▸ Cans with friction closures and ▸ low pressure tubes and cartridges may be used. - Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages *. - In addition, where inner packagings are glass and contain liquids of packing group I and II there must be sufficient inert absorbent to absorb any spillage *. - * unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	▸ A number of phosphate and thiophosphate esters are of limited thermal stability and undergo highly exothermic self-accelerating decomposition reactions which may be catalysed by impurities. ▸ The potential hazards can be reduced by appropriate thermal control measures. BRETHERICK L.: Handbook of Reactive Chemical Hazards Thermal decomposition of organophosphate esters, in the presence of trimethylolpropane or its homologues (common components of synthetic lubricants), may produce bicyclic phosphates and phosphites. These may occur be produced in as little as 5 minutes at 650 deg C. These bicyclic compounds are a class of materials with neurotoxic properties which produce convulsive seizures in test animals. The formation of these compounds does not occur, for example, in the presence of a pentaerythritol base (another common component of synthetic lubricants). ▸ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

MATERIAL DATA

CEL TWA: 0.001 mg/m3
(CEL=Chemwatch Exposure Limit)

Exposure controls

Appropriate engineering controls	**Unless written procedures, specific to the workplace are available, the following is intended as a guide:** ▸ **For Laboratory-scale handling of Substances assessed to be toxic by inhalation.** ***Quantities of up to 25 grams*** may be handled in Class II biological safety cabinets *; ***Quantities of 25 grams to 1 kilogram*** may be handled in Class II biological safety cabinets* or equivalent containment systems; ***Quantities exceeding 1 kg*** may be handled either using specific containment, a hood or Class II biological safety cabinet*, ▸ HEPA terminated local exhaust ventilation should be considered at point of generation of dust, fumes or vapours. ▸ The need for respiratory protection should also be assessed where incidental or accidental exposure is anticipated. Dependent on levels of contamination, PAPR, full face air purifying devices with P2 or P3 filters or air supplied respirators should be evaluated. When handling: ***Quantities of up to 25 grams***, an approved respirator with HEPA filters or cartridges should be considered; ***Quantities of 25 grams to 1 kilogram,*** a half-face negative pressure, full negative pressure, or powered helmet-type air purifying respirator should be considered. ***Quantities in excess of 1 kilogram*****,** a full face negative pressure, helmet-type air purifying, or supplied air respirator should be considered. Written procedures, specific to a particular work-place, may replace these recommendations * For Class II Biological Safety Cabinets, Types B2 or B3 should be considered. Where only Class I, open fronted Cabinets are available, glove panels may be added, Laminar flow cabinets do not provide sufficient protection when handling these materials unless especially designed to do so. **Pilot Plant and Production** ▸ Wear appropriate gloves; lab coat, nylon coveralls or disposable Tyvek suit; safety glasses, safety shoes, and disposable booties. Use good manufacturing practices (i.e., cGMPs). ▸ Protective garment (coveralls, Tyvek, lab coat) is not to be worn outside the work area. ▸ Clean/dirty/decontamination areas are to be established. ▸ Negative/positive air pressure relationships and buffer zones required (i.e., ante-room/degowning room/airlock). ▸ Area access is to be restricted. ▸ High-energy operations such as milling, particle sizing, spraying or fluidising should be done within an approved emission control or containment system. ▸ Develop cleaning procedures and techniques that limit potential exposure **For potent pharmacological agents:** **Solutions Handling:** ▸ Solutions can be handled outside a containment system or without local exhaust ventilation during procedures with no potential for aerosolisation. If the procedures have a potential for aerosolisation, an air-purifying respirator is to be worn by all personnel in the immediate area. ▸ Solutions used for procedures where aerosolisation may occur (e.g., vortexing, pumping) are to be handled within a containment system or with local exhaust ventilation. ▸ In situations where this is not feasible (may include animal dosing), an air-purifying respirator is to be worn by all personnel in the immediate area. If using a ventilated enclosure that has not been validated, wear a half-mask respirator equipped with HEPA cartridges until the enclosure is validated for use. ▸ Ensure gloves are protective against solvents in use.
Individual protection measures, such as personal protective equipment	

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Eye and face protection	▸ Chemical protective goggles with full seal.[AS/NZS 1337.1, EN166 or national equivalent] ▸ Shielded mask (gas-type). ▸ 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	**NOTE:** ▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. ▸ Rubber gloves (nitrile or low-protein, powder-free latex, latex/ nitrile). Employees allergic to latex gloves should use nitrile gloves in preference. ▸ Double gloving should be considered. ▸ PVC gloves. ▸ Change gloves frequently and when contaminated, punctured or torn. ▸ Wash hands immediately after removing gloves. ▸ Protective shoe covers. [AS/NZS 2210] ▸ Head covering.
Body protection	See Other protection below
Other protection	▸ When handling antineoplastic materials, it is recommended that a disposable work-uniform (such as Tyvek or closed front surgical-type gown with knit cuffs) is worn. ▸ Potentially contaminated bodily fluids should be handled in accordance with local standards or codes of practice (appendix 10 of 'Cytotoxic Drugs and Related Waste' - Workcover New South Wales, HSE Information Sheet MISC615, OSHA Technical Manual (OTM) Section VI: Chapter 2) ▸ For quantities up to 500 grams a laboratory coat may be suitable. ▸ For quantities up to 1 kilogram a disposable laboratory coat or coverall of low permeability is recommended. Coveralls should be buttoned at collar and cuffs. ▸ For quantities over 1 kilogram and manufacturing operations, wear disposable coverall of low permeability and disposable shoe covers. ▸ For manufacturing operations, air-supplied full body suits may be required for the provision of advanced respiratory protection. ▸ Eye wash unit. ▸ Ensure there is ready access to an emergency shower. ▸ For Emergencies: Vinyl suit ▸ Ensure that there is a supply of atropine tablets on hand ▸ Ensure all employees have been informed of symptoms of organophosphorus or carbamate poisoning and that the use of atropine in first aid is understood .

Respiratory protection

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

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

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	A-AUS / Class1 P2	-
up to 50	1000	-	A-AUS / Class 1 P2
up to 50	5000	Airline *	-
up to 100	5000	-	A-2 P2

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up to 100	10000	-	A-3 P2
100+			Airline**

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

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Slightly viscous liquid with a characteristic odour; partially mixes with water.

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

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

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

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Inhaled	Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation. The material is not thought to produce respiratory irritation (as classified by EC Directives using animal models). Nevertheless inhalation of vapours, fumes or aerosols, especially for prolonged periods, may produce respiratory discomfort and occasionally, distress. Symptoms of acute exposure to cholinesterase-inhibiting compounds may include the following: numbness, tingling sensations, incoordination, headache, dizziness, tremor, nausea, abdominal cramps, sweating, blurred vision, difficulty breathing or respiratory depression, slow heartbeat. Very high doses may result in unconsciousness, incontinence, and convulsions or fatality. Some cholinesterase-inhibitors may cause delayed symptoms beginning 1 to 4 weeks after an acute exposure that may or may not have produced immediate symptoms. In such cases, numbness, tingling, weakness, and cramping may appear in the lower limbs and progress to incoordination and paralysis. Improvement may occur over months or years, but some residual impairment may remain The early warnings of poisonings associated with cholinesterase inhibition include nasal hyperaemia (localised engorgement with blood), watery discharge, chest discomfort, dyspnoea and wheezing due to increased bronchial secretions and bronchioconstriction. Other effects may include tearing, urination, chest pains, breathing difficulties, low blood pressure, irregular heartbeat, loss of reflexes, twitching, visual disturbances, dilated or pin-point pupils, convulsion, lung congestion, coma and heart-include ataxia, slurred speech, tremors of the tongue and eyelids, and eventual paralysis of the extremities and respiratory muscles. Fatalities in man are generally due to respiratory failure on the basis of central nervous system paralysis although cardiac arrest may also occur. Where cholinesterase inhibitors have been used as miotic eyedrops there has occasional evidence of toxic effects on the crystalline lens and obstruction of the nasolachrymal canals. Inhalation hazard is increased at higher temperatures. Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may produce severely toxic effects. Relatively small amounts absorbed from the lungs may prove fatal.
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. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing. Symptoms may include nausea, headache, giddiness, blurred vision, contraction of pupils, vomiting.
Skin Contact	Skin contact with the material may produce toxic effects; systemic effects may result following absorption. Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact. The material may accentuate any pre-existing dermatitis condition 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.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. Direct contact with the eyes may produce lachrymation (tears), twitching of the eyelids, miosis (contraction of the pupils) and ciliary muscle spasm mydriasis (dilation of the pupils). Absorption may produce generalised cholinesterase inhibition.
Chronic	Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. Toxic: 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. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects. Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects. On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

Titan Dual Grain Treatment (Titan Dual Grain Treatment)	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

methoprene	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 3000 mg/kg[2]	Not Available
	Inhalation (Rat) LC50: >210 mg/L4h[2]	
	Oral (Rabbit) LD50; 3038 mg/kg[2]	

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fenitrothion	**TOXICITY** / **IRRITATION** dermal (rat) LD50: 890 mg/kg[2] / Eye (Rodent - rabbit): 0.1mL Inhalation (Rat) LC50: 0.378 mg/L4h[2] / Eye: no adverse effect observed (not irritating)[1] Oral (Rat) LD50: 250 mg/kg[2] / Skin: no adverse effect observed (not irritating)[1]
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***

METHOPRENE	Toxicity Class WHO Table 5; EPA IV * ADI 0.1 mg/kg b.w. * Olfaction changes, somnolence, body temperature decrease, changes in adrenal weight recorded. NOEL: In 2-year feeding trials, rats 5000 mg/kg diet, mice 2500 mg/kg diet No teratogenic effects on rats at 1000 mg/kg, rabbits at 5000 mg/kg. No mutagenic effects on rats at 2000 mg/kg. No adverse reproductive effects in a 3-generation study on rats at 2500 mg/kg diet * 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. Epoxidation of double bonds is a common bioactivation pathway for alkenes. The allylic epoxides, so formed, were found to possess sensitising capacity in vivo and in vitro and to chemically reactive towards a common hexapeptide containing the most common nucleophilic amino acids. Further-more, a SAR study of potentially prohaptenic alkenes demonstrated that conjugated dienes in or in conjunction with a six-membered ring are prohaptens, whereas related alkenes containing isolated double bonds or an acyclic conjugated diene were weak or nonsensitizing compounds.This difference in sensitizing capacity of conjugated dienes as compared to alkenes with isolated double bonds was found to be due to the high reactivity and sensitizing capacity of the allylic epoxides metabolically formed from conjugated dienes. Allergic Contact Dermatitis—Formation, Structural Requirements,and Reactivity of Skin Sensitizers. Ann-Therese Karlberg et al: Chem. Res. Toxicol. 2008, 21, pp 53–69 https://ftp.cdc.gov/pub/Documents/OEL/06.%20Dotson/References/Karlberg_2008.pdf For methoprene: **Acute toxicity:** Methoprene is practically nontoxic when ingested or inhaled and slightly toxic by dermal absorption. The oral LD50 for methoprene in rats is greater than 34,600 mg/kg, and in dogs is greater than 5000 mg/kg . It is slightly toxic by skin exposure, with reported dermal LD50 values of greater than 2000 to 3000 mg/kg in rabbits . Methoprene is not an eye or skin irritant, and it is not a skin sensitiser . The inhalation LC50 for methoprene in rats is greater than 210 mg/L . No overt signs of poisoning have been reported in incidents involving accidental human exposure to methoprene . **Chronic toxicity:** No methoprene-related effects were observed in 2-year feeding trials with rats given doses of 250 mg/kg/day, nor in mice given 30 mg/kg/day . Liver changes were observed in mice fed 50 to 250 mg/kg/day of methoprene during an 18-month study . Increased liver weights occurred in rats fed 250 mg/kg/day for 90 days, but not during a 24-month feeding study in which rats were fed 125 mg/kg/day . **Reproductive effects:** Experimental data indicate that no reproductive hazards are associated with methoprene . No methoprene-related effects were observed in three-generation reproduction studies in rats receiving dietary doses of 125 mg/kg/day . **Teratogenic effects:** There have been no teratogenic effects in animals dosed with methoprene; teratogenic effects were not seen in rats at doses of about 25 mg/kg/day, or in rabbits at doses of about 15 mg/kg/day . Methoprene does not appear to be teratogenic. **Mutagenic effects:** Methoprene does not appear to be mutagenic. No methoprene-related mutagenic effects were observed in rats following a single dose of 2000 mg/kg. **Carcinogenic effects:** No tumors were seen in an 18-month feeding study with mice, or in a 24-month oncogenicity study with rats. These data suggest that methoprene is not carcinogenic. **Organ toxicity:** The target organ primarily affected by methoprene after long-term exposure is the liver. **Fate in humans and animals:** In mammals, methoprene is rapidly and completely broken down and excreted, mostly in the urine and feces . Some evidence suggests that methoprene metabolites are incorporated into natural body components . Methoprene is excreted unchanged in cattle faeces in amounts that are sufficient to kill some larvae that breed in dung
FENITROTHION	NOTE: Maximum levels have been determined for manufacturing impurities, s-methyl fenitrothion and fenitrooxon. ADI: 0.003 mg/kg/day NOEL: 0.3 mg/kg/day
Titan Dual Grain Treatment (Titan Dual Grain Treatment) & FENITROTHION	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. PI3K inhibitors are often analogues of wortmannin, a steroid metabolite of the fungi Penicillium funicolosam. Severe hepatotoxicity and chemical instability make wortmannin an impractical pharmaceutical agent. Toxicities have included hyperglycaemia (likely a result of the role of PI3K in insulin signaling), skin toxicity, and gastrointestinal side effects. The pi3k/Akt/mtor (phosphatidylinositol 3 kinase/ Akt/mammalian target of rapamycin) signalling pathway is an established driver of oncogenic activity in human malignancies. Hyperglycaemia has been observed in clinical trials of all five classes of pi3k/Akt/mtor signalling pathway inhibitors, a finding that is not unexpected considering the role of the pi3k/Akt/mtor signalling pathway in regulating glucose metabolism. Rash has been reported in patients treated with pan-pi3k inhibitors, pan-Akt inhibitors, and mtor inhibitors; those events have been attributed to cytokine and chemokine deregulation resulting from pathway inhibition. Other adverse effects associated with administration of specific pi3k/Akt/mtor signalling pathway inhibitors include neutropenia, gastrointestinal toxicity, and mood disorders, which have so far been observed only in clinical trials of pan-pi3k inhibitors. Stomatitis and noninfectious pneumonitis have so far been reported only in patients treated with mtor inhibitors Differing toxicities are associated with the various classes of pi3k/Akt/mtor pathway inhibitors. The most common unique adverse events observed in everolimus clinical trials in breast cancer include stomatitis (all grades: approximately 60%), noninfectious pneumonitis (15%), rash (40%), hyperglycaemia (15%), and immunosuppression (40%). Stomatitis associated with mtor inhibitors is characterised by discrete, superficial, aphthous-like ulcers with a greyish-white pseudomembrane; it is clinically distinct from conventional chemotherapy-induced mucositis Stomatitis events typically occur within 2-8 weeks of the initiation of mtor inhibitor treatment; the incidence drops considerably after the first 6-8 weeks. Stomatitis was the most commonly reported all-grade adverse effects and it was among the most commonly reported grades 3 and 4 adverse effects. Importantly, most patients (>97%) can experience complete resolution of stomatitis (approximately 16-22 days from onset) with symptomatic interventions and dose modification. Everolimus-associated rash presents as an acneiform dermatitis that starts as an inflammatory lesion (papule or pustule), with the subsequent appearance of comedones (blackheads). The rash is widely distributed and is often found on the upper extremities and neck. Patients receiving everolimus can be predisposed to bacterial, fungal, viral, or protozoal infections, including pneumonia, sepsis, mycobacterial infections, aspergillosis, candidiasis, and reactivation of hepatitis B virus. In patients treated with everolimus, infections occurred in 44% of patients, but the incidence of grade 3 or 4 infections was low (grade 3, 4%; grade 4, 2%

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- CAUTION: May produce immunosuppression in individuals occupationally exposed to the material.

Exposure to immunosuppressives may aggravate infectious diseases.
Chronic exposure to therapeutic doses of compounds which produce immunosuppression has been associated with development of lymphomas (occasionally malignant) and mammary tumours. These may be secondary effects induced by activation of endogenous retroviruses.
Patients on immunosuppressive medications have a 10- to 100-fold increased risk of cancer compared to the general population. Furthermore, people who currently have or have already been treated for cancer have a higher rate of tumor progression and recurrence than patients with an intact immune system.
Patients receiving immunosuppressive regimens involving combinations of drugs, as part of an immunosuppressive regimen are at increased risk of developing lymphomas and other malignancies, particularly of the skin. The risk appears to be related to the intensity and duration of immunosuppression rather than to the use of any specific agent
Increased incidences of neoplasms, in mice and humans, have been reported after long-term immunosuppression by azathioprine and cyclosporin. Cyclosporin has been classified as a human carcinogen, by IARC, based on development of lymphomas after repeated and prolonged exposures to therapeutic doses.
For fenitrothion:
Acute Toxicity: The acute toxicity of fenitrothion to mammals is considered to be low . Typical symptoms of acute poisoning are observed in rats at doses considerably higher than those applied for parathion-methyl, a structural analogue of this substance . Studies reported primary dermal irritation; mild dermal irritation was reported in a rabbit study. Primary eye irritation was also reported; mild irritation was seen after a single application of 0.1 ml of fenitrothion into unwashed eyes of albino rabbits .
Chronic Toxicity: Chronic symptoms in humans include: general malaise, fatigue, headache, loss of memory and ability to concentrate, anorexia, nausea, thirst, loss of weight, cramps, muscular weakness and tremors. Fenitrothion at sufficient dosage produces typical cholinergic poisoning . In a study with rats, a dietary level of 500 ppm for 90 days was tolerated. They grew normally, and cholinesterase in plasma, red cells and tissues was decreased. A dietary level of 30 ppm for six months decreased the red cell and brain cholinesterase of female but not male rats; neither sex showed any sign of toxicity. A dietary level of 5 ppm for 92 weeks was a no-effect-level (NEL) . Mice that received fenitrothion at a dietary level of 1000 ppm developed symptoms within a week and at the end of a 20-day feeding period had cholinesterase activity in brain, red cells, and plasma reduced to 45, 26 and 5% of normal, respectively. Monkeys are more susceptible than dogs. A dosage of 2 mg/kg/day produced no effect on serum or erythrocyte cholinesterase in dogs but after 2 months of administration, did cause a reduction of erythrocyte enzyme activity in monkeys. A dietary concentration of 5 ppm was found to be a NEL in calves . Adverse effects and death were observed in rats given a diet containing 400 ppm for 63 weeks. Some of the animals survived, although at this level there was a 100% drop in erythrocyte cholinesterase. In 1.77-year feeding trials, the NEL for rats was 5 mg/kg diet . In dogs, doses of 0, 2, 9 and 40 mg/kg body weight/day of fenitrothion were administered for 98 days; at 40 mg/kg/day signs of poisoning and cholinergic stimulation were observed . Rats receiving a diet containing 10 ppm showed a slight drop in erythrocyte cholinesterase activity after 5 weeks of treatment; activity returned to normal 2 weeks after treatment stopped; with a 20 ppm level dose there was a reduction in erythrocyte and brain cholinesterase activity. No significant effect on cholinesterase activity was observed in plasma or erythrocytes at a dietary level of 20 ppm, and it was only with 100 ppm or more that effects were observed; enzyme activity returned to normal 30-40 days after the end of treatment . Rats fed on a diet containing 400 ppm for two years showed a 100% drop in erythrocyte cholinesterase activity. At 100 ppm, 10-30% depression of brain and 30-65% depression of erythrocyte and plasma cholinesterase activity occurred . Other studies indicated that there is a significant inhibition of growth and various cholinergic signs for 2 to 3 weeks following administration of 500 ppm fenitrothion in rats . The no-observable-effect-level (NOEL) for brain and red blood cell cholinesterase is 10 ppm, while the systemic NOEL for plasma inhibition in dogs is 5 ppm . Sumithion 50EC (a product containing fenitrothion) has been shown to cause delayed neurotoxicity in adult rats, as well as humans (143). **Reproductive Effects:** Damage to the nuclear membrane, decreases in staining capacity of cells, and an increase in anomalous mitoses were reported in monolayer cultures of fibroblasts taken from rats that received 0.1 or 0.2 of the LD50 level of fenitrothion daily during the first 15 days of pregnancy. However, the results were not dosage related, nor was there any change in rate of proliferation or mitotic phase distribution from that of the controls . Behavioral deficits have been noted in newborn mammals. Results from a study where pregnant rats were treated with 0, 5, 10 and 15 mg/kg of the product Sumithion 50EC daily through gestation days 7 to 15, showed the following results: There were no significant differences in number of pups born per litter, weight per litter or day of eye and ear opening. There was a significant difference in mortality up to day 16 postpartum: at the 15 mg/kg dose, 17.5% of the pups died; at the 10 and 5 mg/kg dose, 16.0% of the pups died; at the 0 mg/kg dose, 5% of the pups died. The remaining pups gained weight normally and showed no overt signs of intoxication. No significant behavioral effects could be measured at the lowest dose of 5 mg/kg/day. At the 10 and 15 mg/kg/day doses, while several of the behavioral outcomes were significantly different from controls, there seemed to be a difference between the "simple" behavioral measures such as motor activity and motor coordination and the more "complex" measures such as conditioned escape and social interactions. Behavioral measures showed significant alterations as long as 104 days following birth, indicating that prenatal intoxication with Sumithion had persistent effects that showed the offspring to be different from untreated animals. The lack of effect at the 5 mg/kg/day dose indicates that this chemical has a steep dose-response function and that exposure of agricultural workers should be carefully monitored .
Teratogenic Effects: No teratogenic effects were observed in albino rabbits dosed with 0, 0.3 or 1 mg fenitrothion/kg/day in gelatine capsules on gestation days 6 through 18 .
Mutagenic Effects: No mutagenic effects were seen in Drosophila melanogaster or mice.
Carcinogenic Effects: In a two-year feeding study in rats (50 males and 50 females), no dose-related increase in tumor incidence was found upon histopathological examinations of all groups . Fenitrothion was administered in the diet to groups of 50 male and 50 female ICR Swiss mice at dose levels of 0, 30, 100 and 200 ppm for 78 weeks. There was no evidence of compound-related effects on appearance and behavior, body weight or mortality. Gross necropsies revealed no consistent compound-related changes in any organs or tissues. The histopathological examinations revealed no consistent treatment-related increase in tumor incidences .
Organ Toxicity: One of the contaminants of fenitrothion, O,O,S-trimethyl phosphorothioate, has a distinct cytotoxic effect on the lungs of rats and is known to modulate immune responses in mice . Fenitrothion is an immunotoxin . In patients who died of pesticide poisoning, 240 ppm fenitrothion were found in the liver . Fenitrothion is considered a suspect viral enhancer, implicated in Reye's syndrome .
Fate in Humans and Animals: Fenitrothion is oxidised by mono-oxygenases in animals, insects and plants and is thereby changed to derivatives containing the P=O group, which are more powerful inhibitors of cholinesterase than was the original thiophosphate. After that, further degradation occurs by rupture of a P-O-CH3 linkage which is more quickly metabolised in the liver than the P-O phenyl linkage rupture occurring with parathion, which could contribute to fenitrothion's low mammalian toxicity . Studies in the mouse, rat and guinea pig have shown that fenitrothion is rapidly absorbed from the mammalian gastrointestinal tract. The presence of the oxygen analogue has been demonstrated in all tissues examined and this oxygen analogue has been detected in blood one minute after an intravenous injection of fenitrothion . Daily fenitrothion doses of 2.5 and 5 mg/man/day for 5 days were excreted within a 12 hour period and there was no indication of accumulation. Fenitrothion applied to the skin of rats disappeared most rapidly in the first hour, suggesting an absorption rate of slightly over 1%. After 31 hours, the highest concentration, other than on the skin, was found in the cartilaginous part of the bones . When volunteers were given single oral doses ranging from 2.5 to 20 mg/person, the maximal concentration of p-nitro-m-cresol in the urine was reached within 12 hours, and nearly the entire amount discharged was eliminated during the first 24 hours. Although the amount recovered was directly dosage-related, the proportion recovered was inversely dosage-related. With one exception, cholinesterase activity remained normal following these doses . The half-life of fenitrothion was noticeably longer after 10 doses of 30 mg/kg/day than after a single dose of 300 mg/kg. It was concluded that this effect was caused by suppression of the metabolism of the compound during its repeated administration and was associated with inhibition of demethylation and hydrolysis by microsomal enzymes . Fenitrothion is decomposed rapidly in tissues to desmethylsumition, dimethyl-phosphorothioic acid and phosphorothionic acid . The oxygen derivative of fenitrothion is formed in the microsomal fraction of the cell, the main metabolising organs being the liver and kidneys. The major excretion product is 3-methyl-4-nitrophenol, which can be further oxidised to 3-carboxy-4-nitrophenol. Another metabolite is the desmethyl-derivative . Eighteen people were subjected to clinical examination while spraying fenitrothion. The level of blood plasma cholinesterase was determined at regular intervals but no abnormalities were found. Blood cholinesterase was analysed in a large number of inhabitants of Nigeria where fenitrothion had been sprayed. After spraying for one week, a 50% reduction in blood cholinesterase in 20 spraymen was recorded. Rapid return to normal levels subsequently took place . Single oral doses of between 2.5 and 20 mg fenitrothion (approximately 0.042 to 0.33 mg/kg body weight) were administered orally to 24 human subjects. The urinary excretion of the metabolite 3-methyl-4-nitrophenol was almost complete in 24 hours, the excretion peak occurring after 12 hours. The plasma cholinesterase level did not decline, except in one of the subjects who had received 0.33 mg/kg fenitrothion
tyrosine kinase inhibitors (TKIs) have gained prominence as the most effective pathway-directed anti-cancer agents. TKIs have shown significant utility in the treatment of multiple hematological malignancies and solid tumors, including chronic myelogenous leukemia, non-small cell lung cancers, gastrointestinal stromal tumors, and HER2-positive breast cancers. Given their widespread applications, an

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increasing frequency of TKI-induced adverse eeffects has been reported. Although TKIs are known to affect multiple organs in the body including the lungs, liver, gastrointestinal tract, kidneys, thyroid, blood, and skin, cardiac involvement accounts for some of the most serious complications. The most frequently reported cardiovascular side effects range from hypertension, atrial fibrillation, reduced cardiac function, and heart failure to sudden death.
Alhough tyrosine kinases have been more successfully targeted pharmacologically . STKs are part of the cell machinery that is involved in signal transduction pathways controlling metabolism, cell division, angiogenesis and other functions . STKs are hyperactivated across multiple malignancies including ovarian cancer ,making them attractive therapeutic targets
yrosine kinase inhibitors treat a wide range of cancers and cause an equally wide range of side effects. For example, one group of tyrosine kinase inhibitors targets cancers such as non-small lung cancer, breast, colorectal and pancreatic cancer. Those treatment side effects may range from mild skin rashes to life-threatening skin issues like Steven-Johnson syndrome/toxic epidermal necrosis. Another group of TKIs that target non-small lung cancer and colorectal cancer may cause cardiovascular issues like high blood pressure (hypertension) and proteinuria.
Side effects vary based on the specific TKI and may include:
Fatigue.
Fever.
Weight loss/weight gain.mall molecule kinase inhibitors often block multiple enzymes meaning means there is some cross over in the actions and side effects of these drugs.
The most common side effects are:
Nonspecific rashes
Periocular oedema
Acral erythema
Splinter haemorrhage
Pigmentation changes
Photosensitivity
For dithiophosphate alkyl esters and their (zinc) salts:
Acute toxicity: Dithiophosphate alkyl esters consist of a phosphorodithioic acid structure with alkyl ester substituent groups. The alkyl groups are saturated hydrocarbon chains that vary in length and extent of branching. While corrosive to tissue the esters demonstrate a low concern for acute systemic toxicity. Data on acute mammalian toxicity of zinc dialkyldithiophosphates in highly refined lubricant base oil also indicate a low concern for acute toxicity. Commercial oil-based samples of the zinc dialkyldithiophosphate category have been tested for acute oral toxicity. The acute oral LD50 for these studies in rats ranged from 2000-3500 mg/kg. Clinical signs observed following treatment included diarrhea, lethargy, reduced food consumption, and staining about the nose and eye. Ptosis, piloerection, ataxia and salivation were occasionally observed. The incidence and severity of these symptoms were proportional to the dose. In many cases the effects were found to be reversible during observation week 2. Necropsy findings were few in number. Lung congestion, gastrointestinal irritation and a reduction in body fat were observed in some animals.
Acute dermal toxicity and irritation studies using the ester on experimental animals resulted in severe dermal irritation and corrosivity. There is minimal opportunity of human exposure to the chemicals in this category. Dithiophosphate alkyl esters exhibit extreme corrosive properties on skin.
Commercial oil-based samples of the zinc dialkyldithiophosphate category have been tested for acute dermal toxicity. The acute dermal LD50s for these studies in rabbits were greater than 2000 mg/kg (limit tests). No treatment-related mortality was observed at doses ranging from 2000-8000 mg/kg. Dermal application of the test materials to abraded skin for 24 hours typically produced moderate-to-severe erythema and edema, which in some cases persisted through the 14-day observation period. Clinical signs included varying degrees of reduced food consumption, weight loss, diarrhea, lethargy, ataxia, ptosis, motor incoordination and/or loss of righting reflex. There were no remarkable gross necropsy observations. Overall, the acute dermal LD50 for these substances were greater than 2000 mg/kg indicative of a relatively low order of lethal toxicity. Zinc dialkyldithiophosphates are high molecular weight components (average > 500 gm/mol), which generally accepted that the molecular weight limit for passive transport across biological membranes. Thus, upon exposure it is unlikely that significant amounts of these components will be absorbed for systemic distribution. In addition, these materials have a low water solubility that further inhibits absorption and distribution in the mammalian system.
The negligible vapor pressure and high viscosity at ambient temperature indicates that these materials are unlikely to represent an inhalation exposure under conditions of use
Repeat dose toxicity: Data from several repeated-dose toxicity studies using commercial samples of zinc dialkyldithiophosphates in highly refined lubricant base oil has been reviewed. Repeated dermal exposure to experimental animals resulted in moderate-to-severe dermal irritation, behavioral distress, body weight loss and emaciation, reduction in hematological parameters and adverse effects on male reproductive organs. These effects were observed across several members of the category with carbon chain lengths ranging from C4-8. There was no evidence that the incremental increase in carbon chain length or molecular weight could be correlated with significant changes in toxicity parameters.
Oral administration caused significant gastric irritation and related gastrointestinal disturbances, signs of distress but with no evidence of adverse effects on male reproductive organs.
Reproductive toxicity: An epidemiological study on workers exposed to oil-based zinc dialkyldithiophosphates (range C4-8) in an additive manufacturing plant revealed no adverse effects on worker reproductive health. Review of the available information underscores the similarity of clinical and pathological findings in repeated-dose dermal toxicity studies with C4-10 zinc dialkyldithiophosphates, as well as the absence of reproduction and developmental toxicity and the lack of untoward findings in a human epidemiological investigation.
Reproductive organ effects, following dermal application, have been observed in male rabbits; these are attributed to the stress associated with the severe dermal responses to the test material, rather than direct a systemic response to the test materials. Changes in male reproductive organs in the rabbit have been observed when other irritating substances are applied to the skin at dose levels that cause skin lesions. Thus, dermal irritation alone, or in combination with the accompanying weight loss and stress, is thought to play a role in the reproductive organ response to repeated cutaneous application of zinc dialkyldithiophosphates.
Mutagenicity: Findings indicate that commercial samples of zinc dialkyldithiophosphates in highly refined lubricant base oil have a small potential for inducing genetic toxicity. In vitro bacterial gene mutation assays, in vitro mammalian gene mutation assays, or in vivo chromosomal aberration assays have been conducted. Frequencies of reverse mutations in bacteria were not significantly changed after exposure to the zinc dialkyldithiophosphates. In vitro mutation studies in mammalian cells indicate that the zinc dialkyldithiophosphates do not consistently display mutagenic activity in the absence of metabolic activation, however, upon biotransformation, these materials showed mutagenic activity. The findings in bacterial and mammalian cells did not vary in proportion to the alkyl chain length or any other physicochemical parameter.
The results of the studies performed in the absence of hepatic microsome activation were inconsistent, but in general indicating that zinc dialkyldithiophosphates have mutagenic potential (3 studies negative, 3 studies positive in the absence of metabolic activation). However, the weight of evidence (2 studies positive, 1 study negative) indicates that metabolic activation of zinc dialkyldithiophosphates by induced hepatic microsomal enzymes results in a significant increase in the mutagenic potential of this class of chemical substances.
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Thiophosphates (or phosphorothioates, PS) are chemical compounds and anions with the general chemical formula PS
4-xO3-
x (x = 0, 1, 2, or 3) and related derivatives where organic groups are attached to one or more O or S. Thiophosphates feature tetrahedral phosphorus(V) centers.]
Organothiophosphates are a subclass of organophosphorus compounds that are structurally related to the inorganic thiophosphates. Common members have formulas of the type (RO)3-xRxPS and related compounds where RO is replaced by RS. Many of these compounds are used as insecticides, some have medical applications, and some have been used as oil additives.
number of phosphorothioates have been studied extensively for their safety profiles in a several species such as mice, rats, monkeys, and humans. The dose-dependent side effects in experimental rats and mice included thrombocytopenia, splenomegaly, and elevation of transaminases]. Histopathology changes included mononuclear cell infiltration in tissues such as liver, kidney, and spleen, and reticuloendothelial cell and lymphoid cell hyperplasia. The severity of side effects is dependent on the dose, frequency, and duration of the administration of oligonucleotides. In general, the toxicity profiles of phosphorothioate oligonucleotides are similar with various lengths and base compositions, with exceptions in the presence of certain sequence motifs such as CpG-dinucleotides] and poly-G , which contribute to the severity of toxicity

Continued...

	hosphates (P = O) are biologically active, whereas phosphorothioates (P = S) need bioactivation to the corresponding metabolite (oxon) before becoming so.
Titan Dual Grain Treatment (Titan Dual Grain Treatment) & METHOPRENE	Steroid Receptor Coactivator (SRC) modulators (like inhibitors or knockouts) affect hormone-driven processes, potentially causing reproductive issues (dwarfism, infertility), immune dysregulation (autoimmunity, T-cell changes), metabolic shifts, and influencing cancer growth (both promoting and inhibiting depending on the context). Side effects often relate to their fundamental role in regulating steroid hormones (estrogen, androgen, progesterone) in tissues like the brain, reproductive organs, and immune cells, leading to broad systemic impacts, though specific inhibitors (like SRC-3 inhibitors in Tregs) show promise with fewer general toxicities than broader therapies Key Adverse Effects & Considerations: Reproductive & Endocrine: Dysfunction: SRC knockout in mice causes reproductive failure, delayed puberty, and dwarfism. Behavioral: SRC-1 inhibition can alter sex behaviors and brain development in rodents. Immune System: Treg Modulation: Inhibiting SRCs (especially SRC-3) in Regulatory T cells (Tregs) can boost anti-cancer immunity but might also impact normal immune balance. Potential Autoimmunity: While SRC-3 KO Tregs seem benign, broader immune targeting can lead to severe autoimmunity, though SRC inhibitors aim to avoid this. Cancer: Dual Role: SRCs drive many cancers (breast, ovarian, prostate). Modulators can inhibit tumor growth, but some (like pan-SRC inhibitors) might impair anti-tumor immunity General Systemic Effects: Metabolic: SRC dysregulation is linked to Polycystic Ovary Syndrome (PCOS) and fibroids. Brain Function: SRCs are crucial for neuroendocrine regulation, affecting seasonal cycles and hormone responses in the brain Therapeutic Context Matters: Specific vs. Pan-Inhibitors: Targeting specific SRCs (like SRC-3 in Tregs) or using targeted inhibitors (like SI-2) aims for anti-cancer effects with fewer systemic side effects than broader immune checkpoint inhibitors, avoiding issues like cytokine release syndrome. Drug Development: Natural compounds (gossypol, bufalin) and synthetic molecules (SI-2) are being developed, showing potential for non-hormonal treatments in women's health, but clinical application needs careful study. In essence, SRC modulators are powerful tools affecting fundamental hormone signaling, meaning their "adverse effects" are often the flip side of their therapeutic action, necessitating precise targeting for safe use. The juvenile hormone mimics generally exhibit excellent acute and repeat dose hazard profiles in animals. Although they disrupt the normal endocrine function of insects, they do not do so in animals.

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

Legend: ✘ ***– Data either not available or does not fill the criteria for classification***
✔ ***– Data available to make classification***

SECTION 12 Ecological information

Toxicity

Titan Dual Grain Treatment (Titan Dual Grain Treatment)

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

methoprene

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	72h	Crustacea	0.001mg/L	4
EC50	48h	Crustacea	0.626-1.197mg/L	4
LC50	96h	Fish	1-10mg/l	4

fenitrothion

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.29-6.49mg/l	4
BCF	1344h	Fish	1.5-101.7	7
EC50	48h	Crustacea	0.001-0.005mg/L	4
EC50(ECx)	504h	Crustacea	<0.001mg/L	2
EC50	96h	Algae or other aquatic plants	0.72-0.92mg/l	4
LC50	96h	Fish	0.002mg/L	4

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

Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
Toxic to bees.
For fenitrothion:
log Kow : 3.4314
Half-life (hr) air : 408-15120
Half-life (hr) H2O surface water : 2-504
BCF : 9-157
Bioaccumulation : not sig

Continued...

Environmental fate:
Breakdown of Chemical in Soil and Groundwater: Preliminary data indicates fenitrothion degrades fairly rapidly in soil with a half-life of less than one week in non-sterile muck, sandy loam soils. The compound is intermediately mobile in a variety of soils ranging from sandy loam to clay .
Breakdown of Chemical in Surface Water: Surface foam on lakes acts as a scavenger and a trap for organic pollutants. Following aerial spraying of fenitrothion, 701 micrograms/l of fenitrothion was recorded in a surface slick formed by wind actions, compared to 9.5 micrograms/l in the subsurface water . Another study indicated the half-life for the disappearance of fenitrothion at 23 degrees C and pH 7.5 in buffered lake water and natural lake water in the dark (10 ppm sol.) was 21.6 and 49.5 days, respectively. In a field experiment (pH 7.0-7.5, 19-23 degrees C), the half-life of fenitrothion was 1.5-2 days upon spraying of a 10% fenitrothion EC-formulation at a rate of 4 oz/A to a model water system .
Breakdown of Chemical in Vegetation: Damage to cabbage and fruit is possible only if the application dose is exceeded. Fenitrothion has been know to be phytotoxic to cotton, Brassica crops, and certain fruit crops when high rates were applied. Certain apple varieties may be russeted . In a study conducted by FAO/WHO, about 50% of 32P-labelled fenitrothion sprayed on rice plants penetrated into the tissues in 24 hours. At the end of this period only 10% was left, indicating rapid decomposition. Some fenitrooxon was formed but it disappeared from the tissues more rapidly than fenitrothion. Rice grains harvested 46 days after treatment contained 0.0007 ppm fenitrothion and less than 1 ppm of p-nitrocresol and dimethyl phosphorothioic acid . Although the oxon may form in plants, it occurs only during the first few days after treatment and in proportions (ca 1%) smaller than those in animals. Desmethyl compounds occur only in minor amounts in plants. The half-life of fenitrothion in green plants ranges between the values established for Parathion and Parathion-Methyl, i.e. between one and two days; the half-life of the oxon is estimated to be only a few hours (FAO/WHO) .
Ecotoxicity:
Fish LC50 (24 h):- rainbow trout 1.1 ppm; (4 days) 0.7 ppm
Fish LC50 (96 h): brook trout 1.7 ppm, bluegill sunfish 3.8 ppm, North American freshwater fish 2-12 micrograms/l
Bird LD50: bobwhite quail 23.6 mg/kg, mallard duck 1190 mg/kg
Oysters LC50 (4 days): 0.69 ppm
Crabs LC50 (24 h): 0.6 ppm
Earthworm LC50 (24 h): 3.6 ppm
Chickens LC50: 325 ppm
Fenitrothion is a toxic hazard to honeybees.
Fenitrothion was found to be highly toxic to upland gamebirds and slightly toxic to waterfowl . The LC50 for pheasants was 450 to 500 ppm in diets of 2-week-old birds when fed fenitrothion-treated feed for 5 days, followed by untreated feed for 3 days .
Effects on Aquatic Organisms: Fenitrothion is moderately toxic to both warmwater and coldwater fish The time for achieving the highest levels of uptake and the extent of retention of organophosphate residues by fish was directly related to the extent of persistence of a compound in water. Motsugo fish exposed to 0.6-1.2 mg/l of fenitrothion attained the highest body concentrations (162 mg/kg) after 3 days. Fenitrothion (4.9 mg/kg) persisted longer than 4 weeks in fish . Fenitrothion is considered somewhat toxic to fish. The chronic toxicity of fenitrothion to fish is considered low . The 48-hour LC50 values for carp ranged between 2.0 mg/l and 4.1 mg/l . One source stated that aerial spraying of fenitrothion at 2 or 3 oz/acre, on New Brunswick forests has been reported to have no deleterious effect on fish in streams in the treated area . In a study on the acute toxicity of fenitrothion to rainbow trout, embryos were found to be the least sensitive, the sac fry stage was intermediate, and fingerlings and adults were the most sensitive. The toxicity of fenitrothion to rainbow trout increased with increasing temperature. The sublethal effects of fenitrothion exposure on fish include:

- **Morpho Anatomical Changes:** Swelling of the abdomen of fathead minnows occurred. Young Atlantic salmon exposed to 1 mg/l swam with distended fins.
- **Behavioral Changes:** There was a pronounced decline in various agonistic behaviors (chasing, vacating, nipping, etc.) within 2 hours of exposure to several concentrations of fenitrothion. Comfort behaviors (flicks, thrusts, etc.) increased with increasing concentration of toxicant, but declined at higher concentration. Altered station selection occurred. At higher concentrations, some fish were unable to maintain position and were swept downstream. After a 5-hour exposure, fish swam near the surface with bloated stomachs and heads pointing downward. Movement was slowed so much that Atlantic salmon did not attempt to avoid capture with a dipnet. Salmon parr exposed to 1 mg/l fenitrothion were more vulnerable to predation by brook trout.
- **Biochemical Changes:** Acetylcholinesterase activity was inhibited 13% to 25% after various sublethal concentrations of fenitrothion. Cholinesterase activity in the erythrocytes, gills, heart, and serum of rainbow trout was reduced within 1 hour after exposure to fenitrothion.
- **Respiratory Effects:** Oxygen consumption of Labeo rohita exposed to fenitrothion progressively decreased with increasing concentrations of insecticide. Exposure caused increased ventilation rate and buccal amplitude at concentrations slightly higher than the 48-hour LC50.
- **Effect on Growth:** Orally administrated fenitrothion had no effect on the growth of rainbow trout (122). The compound is considered very toxic to crustaceans and aquatic insects and has a medium toxicity to aquatic worms (115). A freshwater invertebrate toxicity (48-hour or 96-hour EC50) reported fenitrothion to be very highly toxic to aquatic invertebrates (3 ppb for Gammarus fasciatus)

For dithiophosphate alkyl esters and their (zinc) salts:
The physicochemical properties of dithiophosphate alkyl esters parallel their structural similarity. All members of this category are within a narrow molecular weight range (256-354 daltons) and are highly acidic. In addition, modeling data indicate they have similar melting and boiling points, low water solubility, low vapor pressure, and are lipophilic in nature.
Modeling data indicates that the vapor pressure of these substances range from 3.46 x 10-3 to 1.9 x 10-5 mm Hg at 25 C and generally follow a pattern based upon their molecular weight and the extent of branching of the alkyl side chain.
Modeling data indicates that these substances have low water solubility and that the log of the octanol-water partition coefficient (log Kow) of these substances range from 4.48-7.99. The low water solubility is consistent with the high lipophilic nature of these substances.
Members of the zinc dialkyldithiophosphate category, described here, contain alkyl chain lengths that range from C3-10, or tetrapropenylphenol (range = C10-15, C12 enriched). It is common for zinc dialkyldithiophosphates to contain mixed alkyl esters (e.g., C4, C5), although derivatives with single chain lengths (e.g., C8) are included in the category . As a result of this diversity in alkyl side chain length, the molecular weight distribution for the members of the category is broad, 578 to 1303 gm/mol. Due to the predominant influence of carbon chain length on molecular weight.
Vapour pressures for the zinc dialkyldithiophosphates are thought to be less than 0.5 mm Hg.
Unpublished data for a commercial zinc dialkyldithiophosphate with an alkyl group less than C8 indicates a water solubility of 1.6 mg/L. The zinc dialkyldithiophosphates are generally regarded to be poorly soluble in water.
Unpublished company data on a commercial zinc dialkyldithiophosphate with a carbon chain length of less than eight yielded a log Kow value of 2.49. Longer chain materials are likely to have higher octanol/water partition coefficients. The log Kow is a measure of the lipophilicity of a substance and is used as a surrogate indicator of the potential of a chemical substance to bioaccumulate in aquatic organisms. While Log Kow is a good predictor of bioaccumulation for nonpolar organic compounds, the mechanisms for uptake and depuration of metals and metal compounds are very complex and variable. For metal compounds, the Log Kow data are not indicative of the bioaccumulation potential .
Fate and Transport Characteristics. Members of this category are expected to be poorly biodegradable.
The members of the category are resistant to hydrolysis at room temperature because they lack readily hydrolysable moieties. When heated hydrolytic degradation results in the formation of the phosphorothioic acid ester and hydrogen sulfide. Continued heating at high temperatures results in the formation of the mono-ester and eventually, phosphorothioic acid itself.
These materials are known to be thermally labile at temperatures >120 C. This decomposition mechanism is key to how the zinc salts provide anti-wear and anti-oxidation performance enhancements in engine oils.
Photodegradation is not expected to cause significant physical degradation of dithiophosphate alkyl esters. Category members do not contain bonds that have a high potential to absorb UV light above 290 nm. These substances have low vapor pressure, which indicates that they have a low potential to partition into the air to a significant extent where they would be subject to indirect photodegradation.
These substances are not expected to partition to water or air if released into the environment due to their low water solubility and low vapor pressure. They are also hydrophobic in nature, which suggests that any which reaches the water compartment will be immobilized through binding to the organic component of soils and sediments.
A Japanese MITI publication cited a bioaccumulation factor of less than 100 for a C4-5 ester zinc dithiophosphate indicating a low potential for bioconcentration or cumulative effects. .
The hydrocarbon portion of these compounds that is susceptible to biodegradation is present in both the zinc dialkyldithiophosphates and the dithiophosphate alkyl esters. Therefore, it is expected that the dithiophosphate alkyl esters will behave similarly. The zinc salts are poorly biodegradable.
Ecotoxicity:
The low water solubility suggests that the acute aquatic toxicity of these substances should be low due to limited bioavailability to aquatic organisms. However, the length of the alkyl side chains on these substances will influence their relative water solubility, and, hence, their relative toxicity. Diethyl dithiophosphate for example is highly toxic to Daphnids Zinc O,O-bis(isooctyl)dithiophosphate (CAS RN 28629-66-5) also appears to be harmful to aquatic organisms such as fish and Daphnids.
Insect growth regulating compounds such as juvenile hormone mimics (JHM) have been observed to have adverse effects in arthropods, such as crustaceans, including disruption of normal molting processes.
For antineoplastics:
Ecotoxicity:
Because antineoplastics are genotoxic, mutagenic and carcinogenic concerns are warranted for their potential effect in the environment. There are a number of known mammalian toxic and nausea effects associated with antineoplastic treatment, which could indicate that similar effects, might be expected in non-target mammals, and possibly also in non-target species other than mammals. Total dosage over a whole therapy protocol is approximately 150 mg /kg body weight. Approximately 14-53% of the administered pharmaceutical is excreted unmetabolised into urine.
Antineoplastics as a class of drugs are of potential concern for environmental impacts, not just for their acute toxicity but perhaps more for their ability to effect subtle genetic changes, the cumulative impact of which over time can lead to more profound ecologic change. Hospitals are the major source of genotoxic drugs. publicly-owned waste-water

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treatment works (POTWs) that service hospitals, especially multiple hospitals, are likely candidates for releasing these chemicals into surface waters.
Antineoplastics are highly [geno]toxic compounds, primarily from hospitals, with poor removal from sewage treatment plants (STWs). Antineoplastic agents, antitumour agents primarily used only within hospitals for chemotherapy, are found sporadically and in a range of concentrations, probably because only small amounts are introduced to STWs via domestic sewage because of their long-lived physiologic retention.
These compounds act as nonspecific alkylating agents (i.e., specific receptors are not involved) and therefore have the potential to act as either acute or long-felt stressors (mutagens carcinogens/ teratogens/ embryotoxins) in any organism.
Using well-established QSAR modelling techniques almost 1/5 of the commonly used antineoplastics were predicted to be very toxic to algae, and close to 1/3 were predicted to be non-toxic to plants. A third of the compounds were predicted to be very toxic to daphnids, and almost half were predicted to be non-toxic to daphnids. Slightly more than 1/5 were predicted to be very toxic to fish, and 47% were predicted to be non-toxic to fish.
For alkenes (olefins)
Environmental fate:
Ecotoxicity studies conducted with a wide range of products have shown little potential for toxicity to aquatic organisms under expected conditions of use or in the event of an accidental release. Not all alpha olefins are readily biodegradable; however, they will ultimately biodegrade. While the octanol/water partition coefficients of alpha olefins suggest a potential for bioaccumulation of these materials in aquatic organisms, the volatility of these materials (especially for the liquid alpha olefins) and the low-water solubility (indicative of limited bioavailability), would indicate that bioaccumulation will not occur. Under most environmental scenarios, extensive evaporation and subsequent degradation in the atmosphere would preclude bioaccumulation. Therefore, alpha olefins are not expected to be toxic to aquatic organisms, will biodegrade, and will not bioaccumulate
The potential for exposure of aquatic organisms to members of the higher olefins will be influenced by their physico-chemical properties. The predicted or measured water solubilities of these olefins range from 50 mg/L at 20 C for hexene to 0.00015 mg/L at 25 C for 1-octadecene, and to 6.33 [E-23] mg/L at 25 C for C54 alpha olefin, which suggests there is a lower potential for the larger olefins to be bioavailable to aquatic organisms due to their low solubilities. Their vapor pressures range from 230.6 hPa at 25 C for hexene to 0.00009 hPa at 25 C for 1-octadecene, and to 1.13 [E-16] hPa at 25 C for C54 alpha olefin, which suggests the shorter chain olefins will tend to partition to the air at a significant rate and not remain in the other environmental compartments for long periods of time; while the longer chain olefins will tend to partition primarily to water, soil or sediment, depending on water solubility and sorption behavior. The predicted soil adsorption coefficients (Koc) range from 149 for C6 to 230,800 for C18 and to 1.0 [E10] for C54, indicating increasing partitioning to soil/sediment with increasing carbon number. Level I fugacity modelling predicts that the C6-13 olefins would partition primarily to air, while the C16 and longer chain olefins would partition primarily to soil. Results of Level III fugacity modelling suggest that the C6 -8 olefins will partition primarily to the water compartment; and, as the chain length increases beyond C10, soil and sediment become the primary compartments. These chemicals have a very low potential to hydrolyse and do not photodegrade directly. However, in the air, all members of the category are subject to atmospheric oxidation from hydroxyl radical attack, with calculated degradation half-lives of 1.8 to 4.8 hours. C6 -30 olefins have been shown to degrade to an extent of approximately 8-92% in standard 28 day biodegradation tests. These results were not clearly correlated with carbon number or any other identifiable parameter; however, the weight of evidence shows that the members of the higher olefins have potential for degradation in the environment. Volatilisation from water is predicted to occur rapidly (hours to days), with Henry s Law Constants (bond method) ranging from 0.423 (C6) to 10.7 (C18), and to 2.89 [E5] (C54) atm- m3/mol. Consideration of these degradation processes supports the assessment that these substances will degrade relatively rapidly in the environment and not persist. Based on calculated bioconcentration factors, the C6, C7, and C16 and longer chain length category members are not expected to bioaccumulate (BCF: C6 = 44-46, C7 = 236, C16 = 71-92 and >= C18 = 3.2-4.6). Although the C8 - 15 olefins have BCFs ranging from 313 to 2030, and Kow values ranging from 4.13 to 7.49, and thus are considered to have the potential for bioaccumulation, their physico-chemical properties and fate indicate that there would be limited environmental exposure because of volatility, biodegradability and limited solubility.
Ecotoxicity:
Data indicate that acute aquatic toxicity can be observed for C6 through the C10 olefins (C6: EC/LC50 range of 1-10 mg/L; C7-C10: EC/LC50 range of 0.1-1.0 mg/L), and that toxicity increases with increasing carbon number within that range, which is consistent with increasing Kow values (3.07 -5.12). Above a chain length of 10, toxicity is not observed within the limits of solubility. However, data indicate that chronic aquatic toxicity can be observed in the C10 olefins (EC10 = 20.0 ug/L, EC50= 28.1 ug/L, NOEC = 19.04 ug/L). Data also suggest that aquatic toxicity does not differ with bond location or presence of branching.
Studies on various thiophosphates indicated complete mineralization within three weeks by acclimation. A water stability study demonstrated the nature of hydrolysis involves the attack of water molecule on the phosphorus ester involving P-O bond fission. .
For organophosphorus compounds:
Environmental fate:
Organophosphorus compounds and pesticides are relatively non-persistent in the environment with half-lives ranging from hours to several weeks or months. Only rarely are pesticides found in crops beyond the growing season during which they are applied. Chemical or photochemical mechanisms may produce a leaving group which is easily degraded. As a rule these compounds do not represent a serious problem as contaminants of soil and water. Breakdown products are usually non-toxic being composed of low-molecular weight, volatile molecules that are easily degraded and utilised by micro-organisms.
Being esters they are also susceptible to hydrolysis. Most organophosphorus pesticides are stable to acid pHs but under alkaline conditions hydrolysis is rapid with the breakdown rate increasing 10-fold for each pH unit above 7. An increase of 10 deg. C of temperature will increase the hydrolysis rate approximately 4-fold. When these compounds are present in the soil their disappearance is affected by their interaction with the physical characteristics and water content of the soil, and the microflora present. In certain types of soil strong binding may make them unavailable for biological decomposition. In such soils even running water produces little movement and thus minimal contamination of water supplies. Less tightly bound substances are similarly unlikely to produce substantial contamination because of rapid breakdown. Metallic ions in the soil interact with organophosphorus esters through hydrogen linkage whilst increased organic matter facilitates further binding.
In general only minute amounts of residue and their breakdown products are found in natural water systems. In soil however there is a greater likelihood of the presence and buildup of toxic residues.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
methoprene	HIGH	HIGH
fenitrothion	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
methoprene	HIGH (LogKOW = 5.5)
fenitrothion	LOW (BCF = 101.7)

Mobility in soil

Ingredient	Mobility
methoprene	LOW (Log KOC = 2830)
fenitrothion	LOW (Log KOC = 864.6)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be

Continued...

applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.

- Antineoplastic (cytotoxic) wastes must be packed directly, ready for incineration, into colour-coded, secure, labelled, leak-proof containers sufficiently robust to withstand handling without breaking, bursting or leaking.
- Containers of special design are available for particular needs (such as disposal of sharps) and should be used.
- Once filled and closed, such containers must never be re-opened.
- Immediate containers must bear a nationally accepted symbol or device depicting cytotoxic substances and be labelled with the words: CYTOTOXIC WASTE - INCINERATE in a style of lettering approved by the national/ state authority.
- Where policies and procedures permit the merging of cytotoxic wastes with medical waste in an outer container used for medical waste, cytotoxic waste must first be placed in identifiable colour-coded/ labelled cytotoxic containers prior to merging.
- Management procedures must ensure that merged medical and cytotoxic waste is subjected to the incineration requirements appropriate for the total destruction of the cytotoxic waste.

WASTE STORAGE OF CYTOTOXIC WASTES

For the storage of cytotoxic waste, segregated or merged with medical waste, provide:

- special storage areas with adequate lighting.
- waste security and restriction of access to authorised persons.
- storage areas designed to facilitate easy routine cleaning and maintenance to hygienic standards, or post-spill decontamination.
- storage of cytotoxic waste in standard, identifying bins or other appropriate containers.

COLLECTION OF CYTOTOXIC WASTES

- Procedures for the collection of cytotoxic wastes, which are compatible with existing operational needs, and which protect workers, other people and the environment, must be developed.
- Waste must be removed from the site by contractors whose workers have been instructed in the protective methods to be used against the hazards involved, and who comply with the safe work practices established by internal and/or national/ state policies. Contractors must instruct, train and direct their personnel in the safe and legal handling of cytotoxic wastes. Contractor's personnel should observe the operating procedures of the waste-generator.
- Transport of cytotoxic wastes, through the community, must comply with the appropriate national/ state codes.

DESTRUCTION OF CYTOTOXIC WASTES

- Destruction of cytotoxic wastes should be carried out in multi-chambered incinerators, licensed for this purpose, operating at 1100 deg. C. or more, with a residence time of at least 1 second.
- Operators must be trained in handling procedures and hazards involved with handling the waste.
- Waste which arrives at the incinerator inappropriately packaged should **NOT** be returned to the waste generator. An authorised representative of the waste generator must attend the incinerator site to rectify the situation.
- **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.

SECTION 14 Transport information

Labels Required

Marine Pollutant

HAZCHEM 2X

Land transport (ADG)

14.1. **UN number or ID number**	3018
14.2. **UN proper shipping name**	ORGANOPHOSPHORUS PESTICIDE, LIQUID, TOXIC (contains fenitrothion)
14.3. **Transport hazard class(es)**	Class: 6.1 Subsidiary Hazard: Not Applicable
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: 61 223 274 Limited quantity: 5 L

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	3018
14.2. **UN proper shipping name**	Organophosphorus pesticide, liquid, toxic * (contains fenitrothion)
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 6.1 ICAO / IATA Subsidiary Hazard: Not Applicable ERG Code: 6L
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: A3 A4 Cargo Only Packing Instructions: 663

Continued...

Cargo Only Maximum Qty / Pack	220 L
Passenger and Cargo Packing Instructions	655
Passenger and Cargo Maximum Qty / Pack	60 L
Passenger and Cargo Limited Quantity Packing Instructions	Y642
Passenger and Cargo Limited Maximum Qty / Pack	2 L

Sea transport (IMDG-Code / GGVSee)

14.1. **UN number**	3018
14.2. **UN proper shipping name**	ORGANOPHOSPHORUS PESTICIDE, LIQUID, TOXIC (contains fenitrothion)
14.3. **Transport hazard class(es)**	IMDG Class: 6.1 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-A Special provisions: 61 223 274 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
methoprene	Not Applicable
fenitrothion	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
methoprene	Not Applicable
fenitrothion	Not Applicable

SECTION 15 Regulatory information

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

methoprene is found on the following regulatory lists

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

fenitrothion is found on the following regulatory lists

Australia Chemicals with non-industrial uses removed from the Australian Inventory of Chemical Substances (old Inventory)
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Model Work Health and Safety Regulations - Schedule 10 - Table 14.1 Hazardous chemicals (other than lead) requiring health monitoring
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (methoprene; fenitrothion)
Canada - NDSL	No (methoprene; fenitrothion)
China - IECSC	No (methoprene)
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (methoprene)
Korea - KECI	No (methoprene)
New Zealand - NZIoC	Yes
Philippines - PICCS	No (methoprene)
USA - TSCA	No (methoprene; fenitrothion)
Taiwan - TCSI	Yes
Mexico - INSQ	Yes

Continued...

National Inventory	Status
Vietnam - NCI	Yes
Russia - FBEPH	No (methoprene; fenitrothion)
UAE - Control List (Banned/Restricted Substances)	Yes
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	05/05/2026
Initial Date	19/03/2026

SDS Version Summary

Version	Date of Update	Sections Updated
3.1	05/05/2026	Toxicological information - Acute Health (eye), Toxicological information - Acute Health (inhaled), Toxicological information - Acute Health (skin), Toxicological information - Acute Health (swallowed), First Aid measures - Advice to Doctor, Toxicological information - Chronic Health, Hazards identification - Classification, Ecological Information - Environmental, Firefighting measures - Fire Fighter (fire/explosion hazard), Firefighting measures - Fire Fighter (fire fighting), First Aid measures - First Aid (eye), First Aid measures - First Aid (inhaled), First Aid measures - First Aid (skin), First Aid measures - First Aid (swallowed), Handling and storage - Handling Procedure, Composition / information on ingredients - Ingredients, Exposure controls / personal protection - Personal Protection (other), Exposure controls / personal protection - Personal Protection (hands/feet), Accidental release measures - Spills (major), Accidental release measures - Spills (minor), Handling and storage - Storage (storage incompatibility), Handling and storage - Storage (storage requirement), Handling and storage - Storage (suitable container), Toxicological information - Toxicity and Irritation (Other), Transport information - Transport, Transport Information

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

- PC—TWA: Permissible Concentration-Time Weighted Average
- PC—STEL: Permissible Concentration-Short Term Exposure Limit
- IARC: International Agency for Research on Cancer
- ACGIH: American Conference of Governmental Industrial Hygienists
- STEL: Short Term Exposure Limit
- TEEL: Temporary Emergency Exposure Limit。
- IDLH: Immediately Dangerous to Life or Health Concentrations
- ES: Exposure Standard
- OSF: Odour Safety Factor
- NOAEL: No Observed Adverse Effect Level
- LOAEL: Lowest Observed Adverse Effect Level
- TLV: Threshold Limit Value
- LOD: Limit Of Detection
- OTV: Odour Threshold Value
- BCF: BioConcentration Factors
- BEI: Biological Exposure Index
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration
- MARPOL: International Convention for the Prevention of Pollution from Ships
- IMSBC: International Maritime Solid Bulk Cargoes Code
- IGC: International Gas Carrier Code
- IBC: International Bulk Chemical Code

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