

Titan Flumioxazin 500 WG (TITAN FLUMIOXAZIN 500 WDG HERBICIDE)

Titan Ag

Chemwatch: 7643-239

Version No: 2.1

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

Chemwatch Hazard Alert Code: 4

Initial Date: 01/06/2026

Revision Date: 01/06/2026

Print Date: 09/06/2026

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

Product Identifier

Product name	Titan Flumioxazin 500 WG (TITAN FLUMIOXAZIN 500 WDG HERBICIDE)
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains flumioxazin)
Chemical formula	Not Applicable
Other means of identification	Not Available

Relevant identified uses of the substance or mixture and uses advised against

Relevant identified uses	Agricultural herbicide. Use according to manufacturer's directions.
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Details of the manufacturer or importer of the safety data sheet

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

Emergency telephone number

Association / Organisation	Titan Ag
Emergency telephone number(s)	1800 033 111 (24 hours)
Other emergency telephone number(s)	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	S6
Classification ^[1]	Serious Eye Damage/Eye Irritation Category 2B, Carcinogenicity Category 1A, Reproductive Toxicity Category 1B, 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)

H320	Causes eye irritation.
H350	May cause cancer.
H360D	May damage the unborn child.
H373	May cause damage to organs through prolonged or repeated exposure.
H410	Very toxic to aquatic life with long lasting effects.

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Precautionary statement(s) Prevention

P202	Do not handle until all safety precautions have been read and understood.
P260	Do not breathe dust/fume.
P280	Wear protective gloves and protective clothing.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P314	Get medical advice/attention if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.

Precautionary statement(s) Storage

P405	Store locked up.
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Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
103361-09-7	50	flumioxazin
1332-58-7	10-30	kaolin
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: - Wash out immediately with fresh running water. - Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. - Seek medical attention without delay; if pain persists or recurs seek medical attention. - Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs: - Flush skin and hair with running water (and soap if available). - Seek medical attention in event of irritation.
Inhalation	- If fumes or combustion products are inhaled remove from contaminated area. - Lay patient down. Keep warm and rested. - Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. - Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. - Transport to hospital, or doctor.
Ingestion	- Immediately give a glass of water. - First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

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
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Advice for firefighters

Fire Fighting	Alert Fire Brigade and tell them location and nature of hazard.
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	▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	▶ Combustible solid which burns but propagates flame with difficulty; it is estimated that most organic dusts are combustible (circa 70%) - according to the circumstances under which the combustion process occurs, such materials may cause fires and / or dust explosions. ▶ Organic powders when finely divided over a range of concentrations regardless of particulate size or shape and suspended in air or some other oxidizing medium may form explosive dust-air mixtures and result in a fire or dust explosion (including secondary explosions). ▶ Avoid generating dust, particularly clouds of dust in a confined or unventilated space as dusts may form an explosive mixture with air, and any source of ignition, i.e. flame or spark, will cause fire or explosion. Dust clouds generated by the fine grinding of the solid are a particular hazard; accumulations of fine dust (420 micron or less) may burn rapidly and fiercely if ignited - particles exceeding this limit will generally not form flammable dust clouds; once initiated, however, larger particles up to 1400 microns diameter will contribute to the propagation of an explosion. ▶ In the same way as gases and vapours, dusts in the form of a cloud are only ignitable over a range of concentrations; in principle, the concepts of lower explosive limit (LEL) and upper explosive limit (UEL) are applicable to dust clouds but only the LEL is of practical use; - this is because of the inherent difficulty of achieving homogeneous dust clouds at high temperatures (for dusts the LEL is often called the "Minimum Explosible Concentration", MEC). ▶ When processed with flammable liquids/vapors/mists, ignitable (hybrid) mixtures may be formed with combustible dusts. Ignitable mixtures will increase the rate of explosion pressure rise and the Minimum Ignition Energy (the minimum amount of energy required to ignite dust clouds - MIE) will be lower than the pure dust in air mixture. The Lower Explosive Limit (LEL) of the vapour/dust mixture will be lower than the individual LELs for the vapors/mists or dusts. ▶ A dust explosion may release of large quantities of gaseous products; this in turn creates a subsequent pressure rise of explosive force capable of damaging plant and buildings and injuring people. ▶ Usually the initial or primary explosion takes place in a confined space such as plant or machinery, and can be of sufficient force to damage or rupture the plant. If the shock wave from the primary explosion enters the surrounding area, it will disturb any settled dust layers, forming a second dust cloud, and often initiate a much larger secondary explosion. All large scale explosions have resulted from chain reactions of this type. ▶ Dry dust can be charged electrostatically by turbulence, pneumatic transport, pouring, in exhaust ducts and during transport. ▶ Build-up of electrostatic charge may be prevented by bonding and grounding. ▶ Powder handling equipment such as dust collectors, dryers and mills may require additional protection measures such as explosion venting. ▶ All movable parts coming in contact with this material should have a speed of less than 1-meter/sec. ▶ A sudden release of statically charged materials from storage or process equipment, particularly at elevated temperatures and/or pressure, may result in ignition especially in the absence of an apparent ignition source. ▶ One important effect of the particulate nature of powders is that the surface area and surface structure (and often moisture content) can vary widely from sample to sample, depending of how the powder was manufactured and handled; this means that it is virtually impossible to use flammability data published in the literature for dusts (in contrast to that published for gases and vapours). ▶ Autoignition temperatures are often quoted for dust clouds (minimum ignition temperature (MIT)) and dust layers (layer ignition temperature (LIT)); LIT generally falls as the thickness of the layer increases. Combustion products include: ▶ carbon monoxide (CO) ▶ carbon dioxide (CO₂) ▶ hydrogen fluoride ▶ nitrogen oxides (NO_x) ▶ other pyrolysis products typical of burning organic material.
HAZCHEM	2Z

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	▶ Remove all ignition sources. ▶ Clean up all spills immediately. ▶ Avoid contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Use dry clean up procedures and avoid generating dust. ▶ Place in a suitable, labelled container for waste disposal. Environmental hazard - contain spillage.
Major Spills	Environmental hazard - contain spillage. Moderate hazard. ▶ CAUTION: Advise personnel in area. ▶ Alert Emergency Services and tell them location and nature of hazard. ▶ Control personal contact by wearing protective clothing. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Recover product wherever possible. ▶ IF DRY: Use dry clean up procedures and avoid generating dust. Collect residues and place in sealed plastic bags or other containers for disposal. IF WET: Vacuum/shovel up and place in labelled containers for disposal. ▶ ALWAYS: Wash area down with large amounts of water and prevent runoff into drains. ▶ If contamination of drains or waterways occurs, advise Emergency Services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▶ Avoid skin contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area.
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	▶ 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. ▶ Organic powders when finely divided over a range of concentrations regardless of particulate size or shape and suspended in air or some other oxidizing medium may form explosive dust-air mixtures and result in a fire or dust explosion (including secondary explosions) ▶ Minimise airborne dust and eliminate all ignition sources. Keep away from heat, hot surfaces, sparks, and flame. ▶ Establish good housekeeping practices. ▶ Remove dust accumulations on a regular basis by vacuuming or gentle sweeping to avoid creating dust clouds. ▶ Use continuous suction at points of dust generation to capture and minimise the accumulation of dusts. Particular attention should be given to overhead and hidden horizontal surfaces to minimise the probability of a "secondary" explosion. According to NFPA Standard 654, dust layers 1/32 in.(0.8 mm) thick can be sufficient to warrant immediate cleaning of the area. ▶ Do not use air hoses for cleaning. ▶ Minimise dry sweeping to avoid generation of dust clouds. Vacuum dust-accumulating surfaces and remove to a chemical disposal area. Vacuums with explosion-proof motors should be used. ▶ Control sources of static electricity. Dusts or their packages may accumulate static charges, and static discharge can be a source of ignition. ▶ Solids handling systems must be designed in accordance with applicable standards (e.g. NFPA including 654 and 77) and other national guidance. ▶ Do not empty directly into flammable solvents or in the presence of flammable vapors. ▶ The operator, the packaging container and all equipment must be grounded with electrical bonding and grounding systems. Plastic bags and plastics cannot be grounded, and antistatic bags do not completely protect against development of static charges. Empty containers may contain residual dust which has the potential to accumulate following settling. Such dusts may explode in the presence of an appropriate ignition source. ▶ Do NOT cut, drill, grind or weld such containers. ▶ In addition ensure such activity is not performed near full, partially empty or empty containers without appropriate workplace safety authorisation or permit.
Other information	▶ Store in original containers. ▶ Keep containers securely sealed. ▶ Store in a cool, dry area protected from environmental extremes. ▶ 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. For major quantities: ▶ Consider storage in bunded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams). ▶ Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities.

Conditions for safe storage, including any incompatibilities

Suitable container	▶ Polyethylene or polypropylene container. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▶ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	kaolin	Kaolin	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits	kaolin	Kaolin	10 mg/m3	Not Available	Not Available	Containing no asbestos and < 1% crystalline silica.

MATERIAL DATA

For kaolin:
Kaolin dust appears to have fibrogenic potential even in the absence of crystalline silica. Kaolinosis can exist as simple and complicated forms with the latter often associated with respiratory symptoms. Crystalline silica enhances the severity of the pneumoconiosis.

The concentration of respirable dust for application of this limit is to be determined from the fraction that penetrates a separator whose size collection efficiency is described by a cumulative lognormal function with a median aerodynamic volume of 4.0 µm (+-) 0.3 µm and with a geometric standard deviation of 1.5 µm (+-) 0.1 µm, i.e.. less than 5 µm.

The TLV-TWA is thought to be sufficiently low to prevent changes in pre- employment chest X-ray findings in exposed employees, in some cases following decades of exposure. The limit is thought to be protective against disabling pneumoconiosis.

WARNING: For inhalation exposure ONLY: This substance has been classified by the IARC as Group 1: **CARCINOGENIC TO HUMANS**

The International Agency for Research on Cancer (IARC) has classified occupational exposures to **respirable** (<5 µm) crystalline silica as being carcinogenic to humans . This classification is based on what IARC considered sufficient evidence from epidemiological studies of humans for the carcinogenicity of inhaled silica in the forms of quartz and cristobalite. Crystalline silica is also known to cause silicosis, a non-cancerous lung disease. Intermittent exposure produces; focal fibrosis, (pneumoconiosis), cough, dyspnoea, liver tumours.

* Millions of particles per cubic foot (based on impinger samples counted by light field techniques).

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NOTE : the physical nature of quartz in the product determines whether it is likely to present a chronic health problem. To be a hazard the material must enter the breathing zone as respirable particles.

Animals exposed by inhalation to 10 mg/m3 titanium dioxide show no significant fibrosis, possibly reversible tissue reaction. The architecture of lung air spaces remains intact.

· The label on a package containing 1% or more of titanium oxide with aerodynamic diameter equal or below 10 microns shall bear the following statement: EUH211 "Warning! Hazardous respirable droplets may be formed when sprayed. Do NOT breathe spray or mist

· The label on the packaging of solid mixtures containing 1% or more of titanium dioxide shall bear the following statement: EUH212 "Warning! Hazardous respirable dust may be formed when used. Do not breathe dust".

In addition, the label on the packaging of liquid and solid mixtures not intended for the general public and not classified as hazardous which are labelled EUH211 or EU212 shall bear statement EUH210: "Safety data sheet available on request."

The concentration of dust, for application of respirable dust limits, is to be determined from the fraction that penetrates a separator whose size collection efficiency is described by a cumulative log-normal function with a median aerodynamic diameter of 4.0 µm (+-) 0.3 µm and with a geometric standard deviation of 1.5 µm (+-) 0.1 µm, i.e..generally less than 5 µm.

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. - Local exhaust ventilation is required where solids are handled as powders or crystals; even when particulates are relatively large, a certain proportion will be powdered by mutual friction. - Exhaust ventilation should be designed to prevent accumulation and recirculation of particulates in the workplace. - If in spite of local exhaust an adverse concentration of the substance in air could occur, respiratory protection should be considered. Such protection might consist of: (a): particle dust respirators, if necessary, combined with an absorption cartridge; (b): filter respirators with absorption cartridge or canister of the right type; (c): fresh-air hoods or masks - Build-up of electrostatic charge on the dust particle, may be prevented by bonding and grounding. - Powder handling equipment such as dust collectors, dryers and mills may require additional protection measures such as explosion venting. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to efficiently remove the contaminant.																
	<table border="1"> <thead> <tr> <th>Type of Contaminant:</th><th>Air Speed:</th></tr> </thead> <tbody> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td><td>1-2.5 m/s (200-500 ft/min)</td></tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td><td>2.5-10 m/s (500-2000 ft/min)</td></tr> </tbody> </table> Within each range the appropriate value depends on: <table border="1"> <thead> <tr> <th>Lower end of the range</th><th>Upper end of the range</th></tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td><td>1: Disturbing room air currents</td></tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only</td><td>2: Contaminants of high toxicity</td></tr> <tr> <td>3: Intermittent, low production.</td><td>3: High production, heavy use</td></tr> <tr> <td>4: Large hood or large air mass in motion</td><td>4: Small hood-local control only</td></tr> </tbody> </table> Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 4-10 m/s (800-2000 ft/min) for extraction of crusher dusts generated 2 metres distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.	Type of Contaminant:	Air Speed:	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 ft/min)	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 ft/min)	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use	4: Large hood or large air mass in motion	4: Small hood-local control only
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Individual protection measures, such as personal protective equipment																	
Eye and face protection	- Safety glasses with side shields. - Chemical goggles.[AS/NZS 1337.1, EN166 or national equivalent] - Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].																
Skin protection	See Hand protection below																
Hands/feet protection	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.																

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· 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.

Experience indicates that the following polymers are suitable as glove materials for protection against undissolved, dry solids, where abrasive particles are not present.

- ▶ polychloroprene.
- ▶ nitrile rubber.
- ▶ butyl rubber.
- ▶ fluorocarbon.
- ▶ polyvinyl chloride.

Gloves should be examined for wear and/ or degradation constantly.

Body protection See Other protection below

Other protection

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

Respiratory protection

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	P1 Air-line*	-	PAPR-P1
up to 50 x ES	Air-line**	P2	PAPR-P2
up to 100 x ES	-	P3	-
		Air-line*	-
100+ x ES	-	Air-line**	PAPR-P3

* - Negative pressure demand ** - Continuous flow

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)

- Respirators may be necessary when engineering and administrative controls do not adequately prevent exposures.
- The decision to use respiratory protection should be based on professional judgment that takes into account toxicity information, exposure measurement data, and frequency and likelihood of the worker's exposure - ensure users are not subject to high thermal loads which may result in heat stress or distress due to personal protective equipment (powered, positive flow, full face apparatus may be an option).
- Published occupational exposure limits, where they exist, will assist in determining the adequacy of the selected respiratory protection. These may be government mandated or vendor recommended.
- Certified respirators will be useful for protecting workers from inhalation of particulates when properly selected and fit tested as part of a complete respiratory protection program.
- Where protection from nuisance levels of dusts are desired, use type N95 (US) or type P1 (EN143) dust masks. Use respirators and components tested and approved under appropriate government standards such as NIOSH (US) or CEN (EU)
- Use approved positive flow mask if significant quantities of dust becomes airborne.
- Try to avoid creating dust conditions.

Where significant concentrations of the material are likely to enter the breathing zone, a Class P3 respirator may be required.

Class P3 particulate filters are used for protection against highly toxic or highly irritant particulates.

Filtration rate: Filters at least 99.95% of airborne particles

Suitable for:

- Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.
- Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.
- Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS
- Highly toxic particles e.g. Organophosphate Insecticides, Radionuclides, Asbestos

Note: P3 Rating can only be achieved when used with a Full Face Respirator or Powered Air-Purifying Respirator (PAPR). If used with any other respirator, it will only provide filtration protection up to a P2 rating.

SECTION 9 Physical and chemical properties**Information on basic physical and chemical properties**

Appearance	Beige granulate.		
Physical state	Divided Solid	Relative density (Water = 1)	0.51
Odour	No Odour	Partition coefficient n-octanol / water	Not Available

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Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	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 Applicable
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Not Available	pH as a solution (1%)	5.4
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	Based on available data, the classification criteria are not met.
b) Skin Irritation/Corrosion	Based on available data, the classification criteria are not met.
c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	Based on available data, the classification criteria are not met.
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	There is sufficient evidence to classify this material as carcinogenic
g) Reproductivity	There is sufficient evidence to classify this material as toxic to reproductivity
h) STOT - Single Exposure	Based on available data, the classification criteria are not met.
i) STOT - Repeated Exposure	There is sufficient evidence to classify this material as toxic to specific organs through repeated exposure
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	The material is not thought to produce respiratory irritation (as classified by EC Directives using animal models). Nevertheless inhalation of dusts, or fumes, especially for prolonged periods, may produce respiratory discomfort and occasionally, distress. Inhalation of dusts, generated by the material during the course of normal handling, may be damaging to the health of the individual. Persons with impaired respiratory function, airway diseases and conditions such as emphysema or chronic bronchitis, may incur further disability if excessive concentrations of particulate are inhaled. If prior damage to the circulatory or nervous systems has occurred or if kidney damage has been sustained, proper screenings should be conducted on individuals who may be exposed to further risk if handling and use of the material result in excessive exposures. Effects on lungs are significantly enhanced in the presence of respirable particles. Overexposure to respirable dust may produce wheezing, coughing and breathing difficulties leading to or symptomatic of impaired respiratory function.
Ingestion	The material has NOT been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.
Skin Contact	The material is not thought to produce adverse health effects or skin irritation following contact (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting. 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	Limited evidence exists, or practical experience suggests, that the material may cause eye irritation in a substantial number of individuals and/or is expected to produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.

Continued...

Chronic	On the basis of epidemiological data, it has been concluded that prolonged inhalation of the material, in an occupational setting, may produce cancer in humans. Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of: - clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. The health hazards associated with bentonite, kaolin, and common clay, which are commercially important clay products, as well as the related phyllosilicate minerals montmorillonite, kaolinite, and illite, have an extensive literature. Fibrous clay minerals, such as sepiolite, attapulgite, and zeolites, have a separate literature. The biological effects of clay minerals are influenced by their mineral composition and particle size. The decreasing rank order of the potencies of quartz, kaolinite, and montmorillonite to produce lung damage is consistent with their known relative active surface areas and surface chemistry. Clays are chemically all described as aluminosilicates; these are further classified as bentonite, kaolin and common clays. Bentonite is a rock formed of highly colloidal and plastic clays composed mainly of montmorillonite, a clay mineral of the smectite group. Kaolin or china clay is a mixture of different minerals. Its main component is kaolinite; in addition, it frequently contains quartz, mica, feldspar, illite, and montmorillonite. The main components of common clay and shale are illite and chlorite. Illite is also a component of ball clays. Illite closely resembles micas, From the limited data available from studies on bentonite-exposed persons, retained montmorillonite appears to effect only mild nonspecific tissue changes, which are similar to those that have been described in the spectrum of changes of the "small airways mineral dust disease" (nodular peribronchiolar dust accumulations containing refractile material [montmorillonite] in association with limited interstitial fibrosis). In some of the studies, radiological abnormalities have also been reported Long-term occupational exposures to bentonite dust may cause structural and functional damage to the lungs. However, available data are inadequate to conclusively establish a dose-response relationship or even a cause-and-effect relationship due to limited information on period and intensity of exposure and to confounding factors, such as exposure to silica and tobacco smoke. Long-term exposure to kaolin may lead to a relatively benign pneumoconiosis, in an exposure-related fashion, known as kaolinosis. Deterioration of lung function has been observed only in cases with prominent radiological alterations. Based on data from china clay workers in the United Kingdom, it can be very roughly estimated that kaolin is at least an order of magnitude less potent than quartz.. Clearcut deterioration of respiratory function and related symptoms have been reported only in cases with prominent radiological findings. The composition of the clay - i.e., quantity and quality of minerals other than kaolinite — is an important determinant of the effects. Bentonite, kaolin, and other clays often contain quartz, and exposure to quartz is causally related to silicosis and lung cancer. Statistically significant increases in the incidence of or mortality from chronic bronchitis and pulmonary emphysema have been reported after exposure to quartz. The removal of clay particles from the lungs takes place by solubilisation in situ and by physical clearance. In humans, there was a rapid initial clearance of 8% and 40% of aluminosilicate particles that were, respectively, 1.9 and 6.1 µm in aerodynamic diameter from the lung region over 6 days. Thereafter, 4% and 11% of the two particle sizes were removed following a halftime of 20 days, and the rest with half-times of 330 and 420 days. Ultrafine particles (<100 nm) have a high deposition in the nasal area; they can penetrate the alveolar/capillary barrier. Epidemiological studies have indicated an increase in morbidity and mortality associated with an increase in airborne particulate matter, particularly in the ultrafine size range An important determinant of the toxicity of clays is the content of quartz. The presence of quartz in the clays studied hampers reliable independent estimation of the fibrogenicity of other components of clays. Single intratracheal injection into rodents of bentonite and montmorillonite with low content of quartz produced dose- and particle size-dependent cytotoxic effects, as well as transient local inflammation, the signs of which included oedema and, consequently, increased lung weight. After high doses of intratracheal kaolin (containing 8-65% quartz), fibrosis has been described in some studies, whereas at lower kaolin doses, no fibrosis has been observed in the few available studies. There are limited data on the effects of multiple exposures of experimental animals to montmorillonite or bentonite. Mice maintained on diets containing 10% or 25% bentonite but otherwise adequate to support normal growth displayed slightly reduced growth rates, whereas mice maintained on a similar diet with 50% bentonite showed minimal growth and developed fatty livers and eventually fibrosis of the liver and benign hepatomas. In vitro studies of the effects of bentonite on a variety of mammalian cell types usually indicated a high degree of cytotoxicity. Concentrations below 1.0 mg/ml of bentonite and montmorillonite particles less than 5 µm in diameter caused membrane damage and even cell lysis, as well as functional changes in several types of cells. No adequate studies are available on the carcinogenicity of bentonite. In an inhalation study and in a study using intrapleural injection, kaolin did not induce tumours in rats. No studies are available on the genotoxicity of clays. Single, very limited studies did not demonstrate developmental toxicity in rats after oral exposure to bentonite or kaolin. Chronic dust inhalation of kaolin, as experienced in mineral extraction, has caused kaolinosis with heavy lung marking, emphysema, and nodular pneumoconiosis. Evidence of kaolinosis (pneumoconiosis) was found in 9% of 553 Cornish china clay workers who had been exposed to kaolin dust for periods exceeding 5 years, whereas no kaolinosis was observed in workers exposed for less than 5 years. Workers in more heavily exposed jobs of milling, bagging and loading showed a prevalence of kaolinosis rising from 6% in those within between 5 and 15 years exposure to 23% in those exposed for more than 15 years. Workers intermittently and less heavily exposed in the older, outdated drying plants required 25 years of massive exposure before reaching the highest prevalence of 17%. Massive fibrosis was seen in four workers, and six workers needed antituberculosis chemotherapy. Preventative measures instituted include preemployment chest examination and approaches to the problem of dust control. Sheer, G.; Brit. Jnl. Ind. Med. 21, pp 218-225, 1964 Long term exposure to high dust concentrations may cause changes in lung function (i.e. pneumoconiosis) caused by particles less than 0.5 micron penetrating and remaining in the lung. A prime symptom is breathlessness. Lung shadows show on X-ray. Overexposure to the breathable dust may cause coughing, wheezing, difficulty in breathing and impaired lung function. Chronic symptoms may include decreased vital lung capacity and chest infections. Repeated exposures in the workplace to high levels of fine-divided dusts may produce a condition known as pneumoconiosis, which is the lodgement of any inhaled dusts in the lung, irrespective of the effect. This is particularly true when a significant number of particles less than 0.5 microns (1/50000 inch) are present. Lung shadows are seen in the X-ray. Symptoms of pneumoconiosis may include a progressive dry cough, shortness of breath on exertion, increased chest expansion, weakness and weight loss. As the disease progresses, the cough produces stringy phlegm, vital capacity decreases further, and shortness of breath becomes more severe. Other signs or symptoms include changed breath sounds, reduced oxygen uptake during exercise, emphysema and rarely, pneumothorax (air in the lung cavity). Removing workers from the possibility of further exposure to dust generally stops the progress of lung abnormalities. When there is high potential for worker exposure, examinations at regular period with emphasis on lung function should be performed. Inhaling dust over an extended number of years may cause pneumoconiosis, which is the accumulation of dusts in the lungs and the subsequent tissue reaction. This may or may not be reversible.					
	<table><tr><th>Titan Flumioxazin 500 WG (TITAN FLUMIOXAZIN 500 WDG HERBICIDE)</th><th>TOXICITY</th><th>IRRITATION</th></tr><tr><td></td><td>Not Available</td><td>Not Available</td></tr></table>	Titan Flumioxazin 500 WG (TITAN FLUMIOXAZIN 500 WDG HERBICIDE)	TOXICITY	IRRITATION		Not Available
Titan Flumioxazin 500 WG (TITAN FLUMIOXAZIN 500 WDG HERBICIDE)	TOXICITY	IRRITATION				
	Not Available	Not Available				

Titan Flumioxazin 500 WG (TITAN FLUMIOXAZIN 500 WDG HERBICIDE)

flumioxazin	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[2]	Not Available
	Inhalation (Rat) LC50: >3.93 mg/L4h ^[2]	
	Oral (Rat) LD50: >5000 mg/kg ^[2]	
kaolin	TOXICITY	IRRITATION
	Not Available	Not Available

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

Titan Flumioxazin 500 WG (TITAN FLUMIOXAZIN 500 WDG HERBICIDE)	For titanium dioxide: Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin. Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica. No data were available on genotoxic effects in titanium dioxide-exposed humans. Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts. Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium. Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light. Animal carcinogenicity data Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats. In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative. Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice. In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative.
	FLUMIOXAZIN Following ingestion, flumioxazin is rapidly absorbed, extensively metabolized and eliminated. Dermal absorption is slow and limited in extent. It has low acute oral, dermal and inhalation toxicity. It is a slight eye irritant, but not a skin irritant or sensitiser. In repeated dose studies in mice, rats and dogs, flumioxazin caused liver toxicity characterised by alterations in liver function and enzyme activities. Oral and dermal exposure induced anaemia and other haematological disturbances in rats. Flumioxazin was negative in studies designed to detect its potential to damage genetic material (DNA), and it did not induce cancers in life-time exposure studies in animals. In developmental studies, an increased incidence of foetal death, impaired foetal development and growth retardation was observed in rats, following oral or dermal exposure at levels which were not toxic to the dams. This has necessitated a label warning statement to alert women of child-bearing age to avoid mixing, loading or spraying any product containing flumioxazin. Female rats received 0, 3000 or 10000 ppm flumioxazin in the diet for up to 5 weeks. The majority of the changes were observed in both treated groups. Anaemia was indicated by significantly reduced RBC, haemoglobin, haematocrit, MCV and MCH, and myeloid/erythroid ratio in the bone marrow. The erythroblast count and the incidence of siderocytes in peripheral blood were increased from day 2. The neutrophil count was initially transiently decreased followed by an increase. Clinical chemistry revealed elevated serum iron levels and lower AST activity. Reduced triglycerides and a slightly increased sodium level were detected at 10,000 ppm only. Spleen and liver weights were increased at necropsy. Elevated urinary coproporphyrin and free erythrocyte protoporphyrin were detected at 3000 ppm. Pregnant rats were given a single oral dose of 0 or 400 mg/kg bw flumioxazin on gestation day-12 and dams were killed on various gestation days thereafter. For the treated group, embryo death was increased from gestation day-15. A high incidence of enlarged foetal heart was observed on days 14 and 15, with a lower incidence from day-16, and no increased incidence by days 17 to 20. The incidence of foetal oedema was increased from day-14, and peaked on days 15-16, but gradually recovered by day-20. Closure of the interventricular foremen was delayed by treatment. Foetal RBC count and the haemoglobin level were reduced during days 13-16, and serum protein level was lower during days 15-16, and all recovered by gestation day-20 Flumioxazin was not mutagenic or genotoxic in an Ames test using S. typhimurium strains TA98, TA100, TA1535, TA1537 and TA1538, and E. coli WP2uvrA, a chromosomal aberration assay in rat bone marrow cells in vivo, and an unscheduled DNA synthesis study in rat hepatocytes in vivo. The ADI for flumioxazin was established at 0.003 mg/kg bw/day based on a NOEL of 3 mg/kg bw/day in an oral developmental study in rats and using a 1,000-fold safety factor in view of the nature and irreversibility of the effect. Titan Flumioxazin 500 WG (TITAN FLUMIOXAZIN 500 WDG HERBICIDE) & FLUMIOXAZIN N-phenylphthalimides exhibit irreversible developmental/reproductive effects at non-maternotoxic acute doses, chronic hepatotoxicity and induction of porphyria and the potential for photodermatitis. For Protoporphyrinogen Oxidase (PPO) Inhibitors: PPO inhibition at high doses resulted in a range of observations in mammalian toxicology studies. As oxidised porphyrin is a key component of mammalian haemoglobin, a common finding at comparatively high doses in toxicology studies was a slight reduction in haemoglobin levels and related blood parameters. Inhibition of porphyrin synthesis results in precursor porphyrins accumulating in the liver where they are

Titan Flumioxazin 500 WG (TITAN FLUMIOXAZIN 500 WDG HERBICIDE)

excreted in the bile coupled with cholesterol. This process results in deposition of pigment in the liver and other tissues, as well as alterations in cholesterol levels due to increased production to compensate for that lost with the porphyrin excretion.

The developmental toxicity studies conducted on rats and rabbits indicate that the majority of the compounds did not show any reproductive, developmental, or teratogenic abnormalities, except at very high doses that elicit maternal toxicity. The developmental toxicity correlates with PPO herbicide accumulation.

The PPO inhibitor herbicides are either not readily absorbed and/or are rapidly degraded by metabolism and/or excreted. The mammalian metabolites are similar to photochemical degradation products. In mammals, there are remarkable species differences in the levels of porphyrin accumulation resulting from exposure to PPO inhibitors. There is no bioaccumulation risk in animals. Metabolism of PPO inhibitors has been studied in a number of species, including rats, rabbits, goats, sheep, cattle, and chicken. In general, the metabolic degradation of these compounds by animals includes nitroreduction, deesterification, and conjugation to GSH, cysteine, and carbohydrates. Most of the metabolites are excreted in urine, with small amounts excreted in faeces and milk. In chickens, 95% of the metabolites are eliminated in excreta, with small amounts (0.09%) eliminated in the eggs.

PPO inhibition in mammals may disrupt heme synthesis, which in turn causes anemia. In the submitted studies, decreased hematological parameters [decreased red blood cells (RBC), decreased hematocrit (Ht), decreased mean corpuscular haemoglobin concentration (MCHC), and mean corpuscular volume (MCV)] were observed at about the same dose level across species, with the exception of the dog, where effects were observed at a slightly higher dose. These effects occurred around the same dose level from short- through long-term exposures, without increasing in severity. Effects were also seen in the liver (increased weight, centrilobular fatty change, lymphoid infiltrate) in mice, the spleen (increased spleen weight and extramedullary hematopoiesis) in rats, and in both these organs (increased iron storage in the liver and extramedullary hematopoiesis in the spleen) in dogs. These effects also occurred around the same dose level from short- through long-term exposures, without increasing in severity. No dermal toxicity was seen at the limit dose in a 28-day dermal toxicity study in rats.

Toxicology studies with PPO inhibitors have shown that certain chemicals cause embryo lethality, teratogenicity and growth retardation in rats but not in other mammals such as rabbits. In these studies it was shown that the effect of 30 mg/kg of S-52482, a phenylimide PPO inhibitor, on embryo development in rats was correlated with the accumulation of protoporphyrin IX (Proto IX) in the embryo with a concomitant loss of haeme. However, 3000 mg/kg of S-52482 caused no accumulation of Proto IX in rabbit embryos and there was no adverse effect on the embryos. The authors concluded that this difference was due to the relative sensitivity of PPO in rats versus rabbits. Thus, the effects of PPO-inhibiting herbicides on mammals is species-dependent. The mammalian toxicity of these herbicides appears to be minimal at the rates they are used.

Protoporphyrinogen oxidase (PPO, E.C.1.3.3.4) catalyzes the oxygen-dependent oxidation of protoporphyrinogen IX to protoporphyrin IX (Proto IX).

In the presence of light, accumulated protoporphyrin can generate highly reactive oxygen species and induce membrane lipid peroxidation. The peroxidation of the lipid can result in a chain reaction and cause fragmentation and destruction of the lipid. The consequence of lipid peroxidation for a cell is loss of the membrane function.

Protoporphyrin IX (Proto IX) is an important precursor to biologically essential prosthetic groups such as heme, cytochrome c, and chlorophylls. As a result, a number of organisms are able to synthesize this tetrapyrrole from basic precursors such as glycine and succinyl CoA, or glutamate. Despite the wide range of organisms that synthesize protoporphyrin IX the process is largely conserved from bacteria to mammals with a few distinct exceptions in higher plants.

The inhibition or functional loss of PPO is more than merely blocking the production of heme and chlorophyll. When the enzyme is inhibited, the substrate protoporphyrinogen-IX will accumulate in the cytoplasm and will be slowly oxidized by O₂ in the mitochondrion and chloroplast to produce protoporphyrin-IX. This spontaneous production can have dire consequences: In the presence of light, the photosensitive protoporphyrin-IX generates singlet oxygen that causes lipid peroxidation and cell death.

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Variegate porphyria (VP) is also an autosomal dominant disorder caused by the deficiency of protoporphyrinogen oxidase.

Symptoms may be cutaneous or neurovisceral with similar inciting factors as acute intermittent porphyria (AIP), but the cutaneous symptoms are more difficult to treat and persist longer. Like hereditary coproporphyria, may be associated with acute episodes (as seen in acute intermittent porphyria) and with photodermatous manifestations (as seen in porphyria cutanea tarda). Protoporphyrin can lead to reactive singlet oxygen formation in the presence of light, and photodermatitis within variegate porphyria (VP) patients is thought to be caused by photooxidation of protoporphyrinogen and increased production of reactive oxygen species within skin fibroblasts. The symptom of VP and its highly variable penetrance of infected individuals make the study of the nature of PPO causing the disease of great interest. Besides, protoporphyrin-IX is an extremely effective photosensitizer, but it is not useful before activation. PPO inhibitors could activate the photosensitizer protoporphyrin-IX and cause its accumulation within tumor cells. Hence, an important medical application of PPO inhibitors is associated with photodynamic therapy (PDT), which has been used in the detection and treatment of cancer. Currently PDT is performed by administering photosensitizers to patients and attempting to establish high concentrations in the tumors.

These tumors are then exposed to irradiation with light with the appropriate wavelength to activate the photosensitizers and destroy the cells. Proto IX is an extremely effective photosensitizer, but it cannot be used since it does not accumulate within tumors after parenteral administration. PPO inhibitors could cause the accumulation of Proto IX within tumor cells. The levels reached after treatment with certain PPO analogs was tenfold higher than the critical levels needed for effective PDT. The use of PPO inhibitors for PDT is being further explored.

No significant acute toxicological data identified in literature search.

for bentonite clays:

Bentonite (CAS No. 1302-78-9) consists of a group of clays formed by crystallisation of vitreous volcanic ashes that were deposited in water. The expected acute oral toxicity of bentonite in humans is very low (LD₅₀>15 g/kg). However, severe anterior segment inflammation, uveitis and retrocorneal abscess from eye exposure were reported when bentonite had been used as a prophylactic.

In a 33 day dietary (2 and 6%) and a 90 day dietary (1, 3 and 5%) studies in chickens, no changes in behaviour, overall state, clinical and biochemical parameters and electrolytic composition of the blood. Repeat dietary administration of bentonite did not affect calcium or phosphorus metabolism. However, larger amounts caused decreased growth, muscle weakness, and death with marked changes in both calcium and phosphorus metabolism.

Bentonite did not cause fibrosis after 1 year exposure of 60 mg dust (<5 µm) in a rat study. However, in a second rat study, where 5 µm particles were intratracheally instilled at 5, 15 and 45 mg/rat, dose-related fibrosis was observed. Bentonite clay dust is believed to be responsible for bronchial asthma in workers at a processing plant in USA.

Ingestion of bentonite without adequate liquids may result in intestinal obstruction in humans.

Hypokalaemia and microcytic iron-deficiency anaemia may occur in patients after repeat doses of clay. Chronic ingestion has been reported to cause myositis.

Titan Flumioxazin 500 WG (TITAN FLUMIOXAZIN 500 WDG HERBICIDE) & KAOLIN

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

Toxicity

Titan Flumioxazin 500 WG (TITAN FLUMIOXAZIN 500 WDG HERBICIDE)	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
flumioxazin	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	96h	Crustacea	0.23mg/l	Not Available
	EC50	48h	Crustacea	5.9mg/l	Not Available
	LC50	96h	Fish	2.3mg/l	Not Available
kaolin	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
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.
Bentonite and kaolin have low toxicity to aquatic species, a large number of which have been tested
DO NOT discharge into sewer or waterways.

Persistence and degradability

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

Bioaccumulative potential

Ingredient	Bioaccumulation
flumioxazin	LOW (LogKOW = 2.55)

Mobility in soil

Ingredient	Mobility
	No Data available for all ingredients

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	- Containers may still present a chemical hazard/ danger when empty. - Return to supplier for reuse/ recycling if possible. Otherwise: - If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. - Where possible retain label warnings and SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: - Reduction - Reuse - Recycling - Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. In most instances the supplier of the material should be consulted. 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.
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SECTION 14 Transport information

Labels Required

	
Marine Pollutant	

HAZCHEM	2Z
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Land transport (ADG)

14.1. UN number or ID number	3077	
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains flumioxazin)	
14.3. Transport hazard class(es)	Class	9
	Subsidiary Hazard	Not Applicable
14.4. Packing group	III	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Special provisions	274 331 335 375 AU01
	Limited quantity	5 kg

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082 are not subject to this Code when transported by road or rail in;

(a) packagings;

(b) IBCs; or

(c) any other receptacle not exceeding 500 kg(L).

- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

Air transport (ICAO-IATA / DGR)

14.1. UN number	3077		
14.2. UN proper shipping name	Environmentally hazardous substance, solid, n.o.s. (contains flumioxazin)		
14.3. Transport hazard class(es)	ICAO/IATA Class	9	
	ICAO / IATA Subsidiary Hazard	Not Applicable	
	ERG Code	9L	
14.4. Packing group	III		
14.5. Environmental hazard	Environmentally hazardous		
14.6. Special precautions for user	Special provisions	A97 A158 A179 A197 A215	
	Cargo Only Packing Instructions	956	
	Cargo Only Maximum Qty / Pack	400 kg	
	Passenger and Cargo Packing Instructions	956	
	Passenger and Cargo Maximum Qty / Pack	400 kg	
	Passenger and Cargo Limited Quantity Packing Instructions	Y956	
	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G	

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	3077		
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains flumioxazin)		
14.3. Transport hazard class(es)	IMDG Class	9	
	IMDG Subsidiary Hazard	Not Applicable	
14.4. Packing group	III		
14.5. Environmental hazard	Marine Pollutant		
14.6. Special precautions for user	EMS Number	F-A, S-F	
	Special provisions	274 335 375 966 967 969	
	Limited Quantities	5 kg	

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
flumioxazin	Not Applicable
kaolin	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
flumioxazin	Not Applicable
kaolin	Not Applicable

SECTION 15 Regulatory information

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

flumioxazin is found on the following regulatory lists
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7
Chemical Footprint Project - Chemicals of High Concern List
kaolin is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (flumioxazin)
Canada - DSL	No (flumioxazin)
Canada - NDSL	No (flumioxazin; kaolin)
China - IECSC	No (flumioxazin)
Europe - EINEC / ELINCS / NLP	No (flumioxazin)
Japan - ENCS	No (flumioxazin; kaolin)
Korea - KECI	No (flumioxazin)
New Zealand - NZIoC	Yes
Philippines - PICCS	No (flumioxazin)
USA - TSCA	TSCA Inventory 'Active' substance(s) (kaolin); No (flumioxazin)
Taiwan - TCSI	Yes
Mexico - INSQ	No (flumioxazin)
Vietnam - NCI	Yes
Russia - FBEPH	No (flumioxazin)
UAE - Control List (Banned/Restricted Substances)	No (flumioxazin; kaolin)
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	01/06/2026
Initial Date	01/06/2026

Other information

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

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

Definitions and abbreviations

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

Titan Flumioxazin 500 WG (TITAN FLUMIOXAZIN 500 WDG HERBICIDE)

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

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