AC RAY 675 WG HERBICIDE

AXICHEM Pty Ltd

Chemwatch Hazard Alert Code: **3**

Chemwatch: **5517-41**
Version No: **2.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **13/01/2022**
Print Date: **13/01/2022**
L.GHS.AUS.EN

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

Product Identifier

Product name	AC RAY 675 WG HERBICIDE
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains metsulfuron methyl and aminopyralid-potassium)
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	For the control of weeds in pastures, non-agricultural areas, commercial and industrial areas and rights-of-way as specified in the Directions for Use. Use according to manufacturer's directions.

Details of the supplier of the safety data sheet

Registered company name	**AXICHEM Pty Ltd**
Address	18 Conquest Way Wangara WA 6065 Australia
Telephone	+61 8 9302 4666
Fax	Not Available
Website	www.axichem.com.au
Email	msds@axichem.com.au

Emergency telephone number

Association / Organisation	**CHEMWATCH EMERGENCY RESPONSE**
Emergency telephone numbers	+61 2 9186 1132
Other emergency telephone numbers	+61 1800 951 288

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SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Carcinogenicity Category 1A, Reproductive Toxicity 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)	

Page 1 continued...

Signal word	**Danger**

Hazard statement(s)

H315	Causes skin irritation.
H318	Causes serious eye damage.
H350	May cause cancer.
H361fd	Suspected of damaging fertility. Suspected of damaging the unborn child.
H410	Very toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.

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.
P391	Collect spillage.
P302+P352	IF ON SKIN: Wash with plenty of water.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

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.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
566191-87-5	30-60	aminopyralid-potassium
74223-64-6	10-30	metsulfuron methyl
1332-58-7	<10	kaolin
497-19-8	<5	sodium carbonate
68512-34-5	<5	sodium lignosulfonate, sulfomethylated
26264-58-4	<5	sodium methyl naphthalenesulfonate
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: ▸ 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

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	lifting the upper and lower lids. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary.
Ingestion	▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.
For triazines:
Clinical effects:
Nausea, vomiting, diarrhoea, abdominal pain and a burning sensation in the mouth.However, due to the lack of clinical data serious effects cannot be excluded from large dose deliberate ingestions.
In the case of products with organic solvents, aspiration can develop. Ataxia, anorexia, dyspnoea and muscle spasms have all been reported in animal studies but have not been seen in humans.
Management principles:
Ingestion:
- In most cases there is probably no need for anything other than oral fluids and reassurance. If a very large amount has been ingested then consider: adult: gastric lavage (with a cuffed endotracheal tube if an organic solvent is involved) followed by 50 g activated charcoal, child: 1 g/kg activated charcoal.
- Do not induce vomiting if product contains an organic solvent.
- Observe the patient if a large dose has been ingested.
- Symptomatic and supportive care.

Inhalation:
- Remove to fresh air. Give oxygen if necessary.
- Bronchodilators may be given if indicated. Otherwise treat for the particular solvent involved.

Skin:
- Wash with copious amounts of water and prevent drying/cracking (due to solvent) with an emollient

Eye:
- Irrigate for 15 to 20 minutes with running water or saline.
- Refer to an ophthalmologist.

IPCS InChem Series

SECTION 5 Firefighting measures

Extinguishing media

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

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 breathing apparatus plus protective gloves in the event of a fire. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ 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.

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Fire/Explosion Hazard	▸ Solid which exhibits difficult combustion or is difficult to ignite. ▸ 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; once initiated larger particles up to 1400 microns diameter will contribute to the propagation of an explosion. ▸ A dust explosion may release 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 also 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-metre/sec. Combustion products include: carbon monoxide (CO) carbon dioxide (CO2) hydrogen chloride phosgene nitrogen oxides (NOx) sulfur oxides (SOx) 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 all personal 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 contact humans, exposed food or food utensils.

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	‣ 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. ‣ No smoking, naked lights or ignition sources. ‣ Store in a cool, dry, well-ventilated area. ‣ Store away from incompatible materials and foodstuff containers. ‣ Protect containers against physical damage and check regularly for leaks. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	‣ Polyethylene or polypropylene container. ‣ Packing as recommended by manufacturer. ‣ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Contains a six-membered heterocyclic ring. Six-membered heterocycles can be described as pi--deficient. Substitution by electronegative groups or additional nitrogen atoms in the ring significantly increase the pi-deficiency. These effects also decrease the basicity. Electrophilic aromatic substitution is more difficult while nucleophilic aromatic substitution is facilitated. for pyridines: · Because of the electronegative nitrogen in the pyridine ring, the molecule is relatively electron deficient. It, therefore, enters less readily electrophilic aromatic substitution reactions, which are characteristic of benzene derivatives; even more so if the reaction mix doesn't scavenge protons released by the reaction (protonated pyridine is even more electron-deficient). However, unlike benzene and its derivatives, pyridine is more prone to nucleophilic substitution and metalation of the ring by strong organometallic bases. · The nitrogen center of pyridine features a basic lone pair of electrons. Because this lone pair is not part of the aromatic ring, pyridine is a base, having chemical properties similar to those of tertiary amines. Pyridine can act as Lewis base, donating its pair of electrons to a Lewis acid. · Pyridine is protonated by reaction with acids and forms a positively charged aromatic polyatomic ion called pyridinium The reactivity of pyridine can be distinguished for three chemical groups. · With electrophiles, electrophilic substitution takes place where pyridine expresses aromatic properties. · With nucleophiles, pyridine reacts at positions 2 and 4 and thus behaves similar to imines and carbonyls. · The reaction with many Lewis acids results in the addition to the nitrogen atom of pyridine, which is similar to the reactivity of tertiary amines. The ability of pyridine and its derivatives to oxidize, forming amine oxides (N-oxides), is also a feature of tertiary amines Secondary amines form salts with strong acids and can be oxidized to the corresponding nitrone using hydrogen peroxide, catalyzed by selenium dioxide

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	Although triazines are aromatic compounds, their resonance energy is much lower than in benzene. Electrophilic aromatic substitution is difficult - nucleophilic aromatic substitution is easier. The 1,2,4-triazines can react with electron-rich dienophiles in an inverse electron demand Diels-Alder reaction. This forms a bicyclic intermediate which normally then extrudes a molecule of nitrogen gas to form an aromatic ring again. In this way the 1,2,4-triazines can be reacted with alkynes to form pyridine rings. Triazine-based ligands have been used to bind three dinuclear arene ruthenium (or osmium) compounds to form metallaprisms. ▸ Avoid oxidising agents, acids, acid chlorides, acid anhydrides, chloroformates. ▸ Avoid reaction with oxidising agents, bases and strong reducing agents. ▸ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

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.

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
sodium carbonate	7.6 mg/m3	83 mg/m3	500 mg/m3

Ingredient	Original IDLH	Revised IDLH
aminopyralid-potassium	Not Available	Not Available
metsulfuron methyl	Not Available	Not Available
kaolin	Not Available	Not Available
sodium carbonate	Not Available	Not Available
sodium lignosulfonate, sulfomethylated	Not Available	Not Available
sodium methyl naphthalenesulfonate	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
aminopyralid-potassium	E	≤ 0.01 mg/m³
sodium carbonate	E	≤ 0.01 mg/m³
sodium methyl naphthalenesulfonate	E	≤ 0.01 mg/m³
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. An approved self contained breathing apparatus (SCBA) may be required in some situations. Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying

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"escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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

Within each range the appropriate value depends on:

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

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

Personal protection

Eye and face protection

- Safety glasses with side shields.
- Chemical goggles.
- 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], [AS/NZS 1336 or national equivalent]

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

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	· 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. ▸ fluorocaoutchouc. ▸ 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.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
AC RAY 675 WG HERBICIDE

Material	CPI
NATURAL RUBBER	A
NITRILE	A

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

Respiratory protection

Particulate. (AS/NZS 1716 & 1715, EN 143:2000 & 149:001, 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

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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	Brown granules with a mild odour.

Physical state	Solid	**Relative density (Water = 1)**	0.52
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Applicable
pH (as supplied)	Not Available	**Decomposition temperature**	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 Applicable	**Taste**	Not Available
Evaporation rate	Not Applicable	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Applicable	**Surface Tension (dyn/cm or mN/m)**	Not Applicable
Lower Explosive Limit (%)	Not Applicable	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Not Available	**pH as a solution (%)**	7.34
Vapour density (Air = 1)	Not Applicable	**VOC g/L**	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

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Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	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. Inhalation overexposure to picolines produces include central nervous system depression and narcosis. Poisoning in a 32 year-old male exposed to industrial vapours of 3-picoline was characterised by unique autonomic disturbances against asthenic background (angiodystonia, tendency towards hypotonia and bradycardia, increase of pilomotor reflex and disturbance of thermoregulation) and by polyneuritic phenomena Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Short-term administration of derivatives of s-triazines cause structural damage to the liver of test animals. The significance of these results (if any) for human exposure cannot, as yet, be determined. [Foltinova etal - Folia Histochemica 1971]. The s-triazines appear to act at the level of carbohydrate metabolism. The chlorinated, methoxy and methylthio derivatives inhibit starch accumulation by blocking sugar production. The s-triazines also cause the disappearance of sucrose and glyceric acid with the formation of aspartic and malic acids. Clinical signs of intoxication by picolines (syn: methylpyridines) include weight loss, diarrhoea, weakness, ataxia and unconsciousness as well as narcosis headache, nausea, giddiness and vomiting. A series of neurophysiological tests performed on Long-Evans rats treated with various isomeric picolines (i.p.) produced central nervous system depression. Oral doses of the picolines are rapidly absorbed and penetrate the liver, heart, spleen, lungs and muscle. Percentage uptake increases with dosage and elimination occurs in two dose-dependent phases. Of the isomers, 4-picoline has the shortest residence time in the liver, brain and kidney whilst 3-picoline has the longest biological half-life.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. 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. Drop application of 2-picoline to rabbit eyes produced moderate injury (8 on a scale of 1 to 10). Severe irritation, severe conjunctival and corneal injury capable of resulting in permanent impairment of vision, were noted
Chronic	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. 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. 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

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consequence of other toxic effects.
Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population.
Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.
There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals.

Sulfonylureas, imidazolinones, sulfonoanilides and triazolo-pyrimidines, as herbicides, are used extensively because of their wide-spectrum effects on weeds and their low toxicity to mammals. The effects of these herbicides on plants, micro-algae and bacteria are due to the inhibition of acetolactate synthase (ALS) involved in the synthesis of acetolactic and butyric acids, which are the precursors of the branched-chain amino acids: isoleucine, leucine and valine.
Mammals also produce these precursor amino-acids using ALS so the potential for toxic effects is apparent though not evident from many studies.

It is reported that an agricultural worker developed contact eczema following several years of exposure to feed pellets containing a calcium lignosulfonate binder. The authors of this report concluded that the contact allergy was due to calcium lignosulfonate alone. Andersson, R., and K. Goransson: NIOSHTIC Record No.: 68738
Epidemiological studies have associated long-term exposures to triazine herbicides with increase risk of ovarian cancer in female farm workers in Italy and of breast cancer in the general population of Kentucky in the United States. In experiments with female F344 rats, atrazine induced tumours of the mammary gland and reproductive organs. Atrazine also caused lengthening of the oestrus cycle, a dose-dependent increase in the plasma levels of 17beta-oestradiol and early onset of mammary and pituitary tumours in female Prague-Dawley rats.
Investigations into the mechanism of these apparent oestrogenic effects have not been able to demonstrate any consistent interactions with triazine herbicides with the oestrogen receptor or effects on receptor-mediated responses. Atrazine, simazine and propazine have been shown to induce aromatase activity in a human adrenocortical carcinoma cell line. This response was observed at concentrations in the submicromolar range. Aromatase is a circulating enzyme which converts androstenedione (generated in the adrenals) to oestrone in peripheral tissues such as adipose tissues. Oestrone subsequently undergoes conversion to oestradiol which binds to oestrogen receptors in many tissues with induction of tumours. In addition, many human breast cancers contain aromatase. (Breast cancer therapies, based on aromatase inhibitors, are now available.)
The effects of triazine herbicides and some of their metabolites on aromatase activity may provide a partial explanation for the observed increase in plasma oestradiol in rats, together with the observed oestrogen-mediated toxicities *in vivo. [1]*
[1] Sanderson et al: Environmental Health Perspectives, 109, pp 1027-1031, 2001
Suggestive evidence between atrazine (or triazines) exposure and an increased risk of prostate cancer, breast cancer, and ovarian cancer have been reported. Although these data provide a suspicion of carcinogenicity, the limited number of investigations and study limitations preclude drawing conclusions regarding these cancer types.
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 um 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.

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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 um 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
Clinical symptoms and signs of intoxication following occupational exposure to pyridine, its homologues and derivatives include gastrointestinal disturbance with diarrhoea, abdominal pain and nausea, weakness, headache, insomnia and nervousness..Data indicate that piperidine, pyridine, methyl and alkyl derivatives of pyridine (picolines, lutidines collidines), nicotinonitrile and picolinonitrile are slightly to moderately toxic following acute exposures
The available data support the conclusion that the pyridines possess similar human health-related data, and in particular, target organs appear to be the liver and the male reproductive tract.,
The weight-of-evidence suggests that Pyridine and Pyridine Derivatives Category chemicals are not mutagenic. This conclusion is supported by a number of in vivo mutagenicity assays and carcinogenicity studies with negative results for pyridine.
Reproductive screening evaluations using several repeated dose toxicity studies indicates that piperidine, pyridine and nicotinonitrile may be male reproductive toxicants.
Exposures less than those which produce overt clinical signs may produce varying levels of liver damage with central lobular fatty degeneration, congestion and cellular infiltration; repeated low level exposures may produce cirrhosis. The kidney is less sensitive to pyridine-induced damage than is the liver. Pyridine, like primidone, phenobarbitol and oxazepam induces liver neoplasms in mice, but not in rats, even though in rats these chemicals cause a spectrum of toxic liver lesions. The mouse, an animal with a high background rate of liver neoplasms, is particularly sensitive to the development of malignant liver neoplasms following chemical exposure. There is equivocal evidence (1) that pyridine is carcinogenic in male F344/N rats (based on an increased incidence of renal tubule neoplasms), in female rats of the same species (based on increases in mononuclear cell leukaemia), in male Wistar rats (based on an increased incidence of mono- nuclear cell leukaemia), and clear evidence of carcinogenicity (1) in male and female B6C3F1 mice (based on increased incidences of malignant hepatocellular neoplasms). 1: National Toxicology Program Technical Report Series No. 470, March 2000
Prolonged or repeated skin contact may cause degreasing with drying, cracking and dermatitis following.
Chronic exposure to picolines (methylpyridines) results in anaemia, ocular and facial paralysis as well as symptoms experienced in acute intoxications.
The structure and composition of the liver and the structure and growth patterns of the skin was altered in the off-spring of female rats given 157 mg/kg/d of 2-picoline throughout pregnancy.
Daily administration of 2-picoline to rats for 4 months at doses of 50 mg/kg decreased hepatic glycogen levels and increased glucose and lactic acids. 300 mg/kg for 1 days initially stimulated energy generation
but subsequently inhibited it.
A 58-year old man occupationally exposed to 3-picoline for 11 years showed an increase in liver glutamic pyruvic transaminase and glutamic oxaloacetic transaminase.
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.

AC RAY 675 WG HERBICIDE	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

aminopyralid-potassium	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

metsulfuron methyl	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye: moderate, reversible *

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	Inhalation(Rat) LC50; >5 mg/L4h[2]	Skin (g.pig): mild *
	Oral (Rat) LD50; >5000 mg/kg[2]	Skin (rabbit): moderate

kaolin	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

sodium carbonate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[2]	Eye (rabbit): 100 mg/24h moderate
	Oral (Rat) LD50; 2800 mg/kg[2]	Eye (rabbit): 100 mg/30s mild
		Eye (rabbit): 50 mg SEVERE
		Eye: adverse effect observed (irritating)[1]
		Skin (rabbit): 500 mg/24h mild
		Skin: no adverse effect observed (not irritating)[1]

sodium lignosulfonate, sulfomethylated	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

sodium methyl naphthalenesulfonate	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; 1350 mg/kg[2]	Skin (rabbit): SEVERE *

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*

AMINOPYRALID-POTASSIUM	Acute toxicity data indicate that aminopyralid has low toxicity via oral, dermal and inhalation routes of exposure. The technical aminopyralid product is classified in toxicity category I [DANGER] based on an acute eye irritation study conducted with the free acid. In a rat developmental study the NOAEL for maternal and developmental toxicity was equal to or greater than 1,000 mg/kg/day [HDT]. In a developmental toxicity study in rabbits with aminopyralid, the NOAEL for maternal toxicity was 250 mg/kg/day and the developmental NOAEL was equal or greater than 500 mg/kg/day. Maternal toxicity was observed at 500 and 750 mg/kg/day [HDT] in the form of decreased body weights and clinical observations of uncoordinated gait. Ulcers and erosions of the glandular mucosa of the stomach were observed in the 500 and 750 mg/kg/day dose groups. Similar toxic effects were also observed in a developmental study in rabbits with Milestone, the formulated triisopropanolamine (TIPA) salt of aminopyralid. Developmental toxicity could not be determined in aminopyralid rabbit study since the 750 mg/kg/day group was removed from the study due to the severity of the clinical signs (body weight changes, decreased food consumption and a decreased amount of feces). However, in the rabbit developmental study with Milestone, developmental toxicity was demonstrated by a decrease in foetal body weights at 520 mg acid equivalents (ae)/kg/day. In a 2-generation reproduction study in rats, there was no evidence of parental, reproductive, or offspring toxicity observed after exposure to aminopyralid up to 1000 mg/kg/day [HDT]. The developmental toxicity studies and the 2-generation reproduction study did not exhibit quantitative or qualitative susceptibility. There were no systemic toxic effects observed at 1000 mg/kg/day [HDT] in a 28-day dermal toxicity study in rats with aminopyralid. However, dermal toxicity was indicated by slight epidermal hyperplasia in males at the HDT. The database on aminopyralid indicates that the stomach, ileum and cecum are targets for this compound. In a 90-day toxicity study in dogs the NOAEL was 282 mg/kg/day for males and 232 mg/kg/day for females based on slight diffuse hyperplasia and hypertrophy of the mucosal epithelium of the stomach at 1070 mg/kg/day in males and 929 mg/kg/day in females. In the 1-year chronic toxicity study in dogs, the NOAEL was 99 mg/kg/day for males and 93 mg/kg/day for females based on thickening of the stomach, slight lymphoid hyperplasia of the gastric mucosa, and slight chronic mucosal inflammation at the HDT. In a 90-day mouse dietary study, no toxicity was observed at 1000 mg/kg/day [HDT]. In a 90-day rat feeding study the NOAEL was 1000 mg/kg/day [HDT] for females and 500 mg/kg/day for males based on hyperplasia of the mucosal epithelium of the ileum and the cecum at 1000 mg/kg/day [HDT]. In the mouse chronic feeding study the NOAEL was 1000 mg/kg/day [HDT] for males and 250 mg/kg/day for females. In the rat chronic feeding study the NOAEL was 50 mg/kg/day based on cecal enlargement, slight mucosal hyperplasia (males) and slightly decreased body weights at 500 mg/kg/day. Aminopyralid has been classified as "not likely" to be carcinogenic to humans. No increases in any tumors were found in carcinogenicity studies in rats and mice. In a metabolism study in rats, aminopyralid was rapidly absorbed, distributed, and excreted following oral administration. Tissue distribution and bioaccumulation were minimal; 0.73% of administered dose [AD] was recovered in tissues after 7 days for all dosing groups. The highest levels of radioactivity were found in the skin and carcass. Aminopyralid was excreted unchanged, indicating an absence of metabolism. The AD was recovered as parent compound in 100% of the feces and = 96% of the urine. Three unknown components found in urine (= 4 %) were also detected in similar quantities in dose formulations, suggesting that they were trace impurities. Based on aminopyralids low toxicity, an acute Reference Dose (RfD) for the general population is not required. The chronic RfD for aminopyralid is 0.5 mg/kg/day. This value is based on the NOAEL of 50 mg/kg/day in the rat combined chronic toxicity/carcinogenicity study with a 100-fold uncertainty factor to account for interspecies extrapolation (10X) and intraspecies variability (10X). An additional safety factor to protect infants and children is not required, due to the toxicity properties of the material and the conservative nature of the exposure estimates USA EPA Pesticide Fact Sheet, 2005
METSULFURON METHYL	ADI: 0.01 mg/kg/day ADI: 0.25 mg/kg * NOEL: 1 mg/kg/day NOEL (2 y) for rats 50, dogs 200 mg/kg diet * Toxicity Class WHO III; EPA IV * The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. For metsulfuron methyl:

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	‣ **Acute Toxicity:** This chemical has very low toxicity in mammals. Based on laboratory tests, the oral dose of metsulfuron-methyl that causes mortality in half of the test animals (LD50) is > 5,000 mg/kg in rats. It has low dermal toxicity in tests with rabbits, with an LD50 > 2,000 mg/kg, and low inhalation toxicity in rats, with a median lethal concentration in air of greater than 5 mg/liter air. Moderate but reversible eye irritation has been seen in rabbits, and mild skin irritation has been observed in guinea pigs. No skin sensitization has been observed in guinea pigs. ‣ **Signs and Symptoms of Poisoning:** Systemic poisoning by sulfonylurea based compounds is unlikely, unless large quantities have been ingested. No accounts of poisoning by metsulfuron-methyl are currently available. ‣ **Chronic Toxicity:** A 2-year feeding study in rats resulted in a No Observable Effects Level (NOEL) of 25.0 mg/kg/day (or 500 ppm in feed), based on decreased body weights seen at 250 mg/kg/day (5,000 ppm) which was the highest dose tested. EPA has based its reference dose (0.25 mg/kg/day) on this study. ‣ **Reproductive Effects:** Multigeneration studies in rats did not result in any reproductive effects at the highest doses tested of 250 mg/kg/day. ‣ **Teratogenic Effects:** Metsulfuron-methyl did not cause developmental abnormalities to offspring of rats and rabbits fed 1000 mg/kg/day and 700 mg/kg/day respectively during gestation. These doses represent the highest dose tested for each experiment. ‣ **Mutagenic Effects:** The weight of evidence presented by a battery of tests to measure mutagenicity and other adverse effects on DNA indicates that metsulfuron-methyl is neither mutagenic nor genotoxic. ‣ **Carcinogenic Effects:** Negative for rats and mice in laboratory tests, but studies may not have been at maximum tolerated dose. ‣ **Organ Toxicity:** Metsulfuron-methyl is a moderate eye irritant. ‣ **Fate in Humans and Other Animals:** The chemical is broken down quickly and eliminated from the body. In tests with radiolabeled metsulfuron-methyl in rats, the excretion half-lives ranged from 9 to 16 hours and 23 to 29 hours for rats administered low and high doses, respectively. It did not bioaccumulate in fish ***[* The Pesticides Manual, Incorporating The Agrochemicals Handbook, 10th Edition, Editor Clive Tomlin, 1994, British Crop Protection Council]***
KAOLIN	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 prophypaste. 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 um) in a rat study. However, in a second rat study, where 5 um 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.
SODIUM CARBONATE	for sodium carbonate: Sodium carbonate has no or a low skin irritation potential but it is considered irritating to the eyes. Due to the alkaline properties an irritation of the respiratory tract is also possible. No valid animal data are available on repeated dose toxicity studies by oral, dermal, inhalation or by other routes for sodium carbonate. A repeated dose inhalation study, which was not reported in sufficient detail, revealed local effects on the lungs which could be expected based on the alkaline nature of the compound. Under normal handling and use conditions neither the concentration of sodium in the blood nor the pH of the blood will be increased and therefore sodium carbonate is not expected to be systemically available in the body. It can be stated that the substance will neither reach the foetus nor reach male and female reproductive organs, which shows that there is no risk for developmental toxicity and no risk for toxicity to reproduction. This was confirmed by a developmental study with rabbits, rats and mice. An *in vitro* mutagenicity test with bacteria was negative and based on the structure of sodium carbonate no genotoxic effects are expected. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
SODIUM METHYL NAPHTHALENESULFONATE	for similar material: CAS RN: 27185-34-6 Dermal (rabbit): 4200 mg/kg * Eye (rabbit): moderate *
AMINOPYRALID-POTASSIUM & KAOLIN	No significant acute toxicological data identified in literature search.
AMINOPYRALID-POTASSIUM & SODIUM CARBONATE	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. 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. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea,

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	cough and mucus production.

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

AC RAY 675 WG HERBICIDE

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

aminopyralid-potassium

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

metsulfuron methyl

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	336h	Algae or other aquatic plants	0.001mg/L	4
LC50	96h	Fish	>0.2mg/l	4
EC50	48h	Crustacea	>220.5mg/L	4
EC50	96h	Algae or other aquatic plants	0.129-0.309mg/L	4

kaolin

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

sodium carbonate

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	Not Available	Algae or other aquatic plants	1-10mg/l	2
LC50	96h	Fish	300mg/l	2
EC50	48h	Crustacea	156.6-298.9mg/l	4

sodium lignosulfonate, sulfomethylated

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

sodium methyl naphthalenesulfonate

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. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. 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.
for alkyl sulfates; alkane sulfonates and alpha-olefin sulfonates:

Environmental fate:
The close structural similarities result in physico-chemical properties and environmental fate characteristic which follow a regular pattern.
The most important common structural feature of the category members is the presence of a predominantly linear aliphatic hydrocarbon chain with a polar sulfate or sulfonate group, neutralised with a counter ion (i.e., Na+, K+, NH4+, or an alkanolamine cation).

Continued...

The hydrophobic hydrocarbon chain (with a length typically between C8 and C18) and the polar sulfate or sulfonate groups confer surfactant properties and enable the commercial use of these substances as anionic surfactants
The structural similarities result in the same mode of ecotoxic action. Within each subcategory the most important parameter influencing ecotoxicity is the varying length of the alkyl chain. Although the counter ion may also influence the physico-chemical behaviour of these chemicals, the chemical reactivity and classification for the purpose of this assessment is not expected to be affected by the difference in counter ion.
As ionic substances, all members of this category have extremely low vapor pressures. Calculated values are in the ranges 10-11 to 10-15 hPa (C8-18 alkyl sulfates), 4.3.10-11 to 9.10-15 hPa (C8-18 alkane sulfonates), 2.1.10-13 to 6.9.10-15 hPa (C14-18 alkene sulfonates) and 3.3.10-17 to 5.8.10-19 hPa (C14-18 hydroxy alkane sulfonates). Therefore, they decompose before reaching their theoretical boiling points.
Measured water solubilities are available only for alkyl sulfates; they are in the range 196 000 mg/l (C12) to 300 mg/l (C16) and by factors of 50 to 300 higher than calculated values (C12: 617 mg/l, C16: 5 mg/l).
As surfactants have a tendency to concentrate at hydrophilic/hydrophobic boundaries rather than to equilibrate between phases log Kow is not a good descriptor of surfactant hydrophobicity and only of limited predictive value for the partitioning of these compounds in the environment.
All calculated physico-chemical properties of surfactants should be treated with caution, because the estimation models do not take into account surfactant properties. In addition, the results are doubtful for ionic substances.
Deduced from physico-chemical and surfactancy properties the target compartment for the substances of this category is the hydrosphere. Based on the ionic structure partitioning into the atmosphere can be excluded. In water, the compounds are stable to hydrolysis under environmental conditions.
Taking into account the low BCF factors (<73) that were determined for (up to) C16-alkyl sulfates, any significant bioaccumulation is not expected.
Soil sorption increases with chain length. Strong sorption on soils would be expected for chain length C14 upwards. Sediment concentrations were between 0.0035 and 0.021 mg/kg dw indicating that accumulation in sediments is low. Under certain conditions of reduced moisture in soil, i.e. in arid or semi-arid regions, accumulation in soil cannot be excluded.
The substances of this category are readily biodegradable. Significant biodegradation of alkyl sulfates in the raw sewage, i.e. in the sewer system before reaching the (waste-water treatment plant (WWTPs) is very likely. The substances of this category are quantitatively removed in WWTPs, mainly by biodegradation.
Because of the anaerobic degradation of alkyl sulfates in sewage sludge, exposure of agricultural soils due to application of sludge as fertiliser is not expected.
However, for alkane sulfonates and alpha-olefin sulfonates this exposure pathway cannot be excluded due to their recalcitrant or limited anaerobic degradability.
For alkyl sulfates: The biological degradation of AS is initiated by a hydrolytic cleavage of the sulfate ester bond catalysed by alkylsulfatases. The cleavage leaves inorganic sulfate and fatty alcohol which undergo oxidation by dehydrogenases to produce fatty acids via fatty aldehydes. The fatty acids are degraded by beta-oxidation and finally totally mineralised or incorporated into biomass. The biodegradation pathway for secondary AS differs from that of the primary AS by the formation of a ketone instead of an aldehyde. The biological degradation of AS is initiated by a hydrolytic cleavage of the sulfate ester bond catalysed by alkylsulfatases. The cleavage leaves inorganic sulfate and fatty alcohol which undergo oxidation by dehydrogenases to produce fatty acids via fatty aldehydes. The fatty acids are degraded by beta-oxidation and finally totally mineralised or incorporated into biomass. The biodegradation pathway for secondary AS differs from that of the primary AS by the formation of a ketone instead of an aldehyde. Biodegradation under anoxic conditions is anticipated to follow the same pathway as for the aerobic degradation.
Primary and secondary AS generally undergo complete primary biodegradation within a few days followed by a rapid ultimate biodegradation. Branched AS are also degraded quite rapidly, but multiple branchings of the alkyl chain considerably reduce the rate and extent of primary biodegradation. There are numerous studies confirming the aerobic biodegradability of AS, and linear primary AS exceeds all other anionic surfactants in the rate of primary and ultimate biodegradation. Also secondary AS are normally readily biodegradable as, e.g., the oxygen uptake from biodegradation of a linear secondary C10-13 AS corresponded to 77% ThOD in 22 days. Some highly branched AS being poorly primary biodegradable may also resist ultimate biodegradation.
Both linear and 2-alkyl-branched primary AS are degraded to a high extent under anaerobic conditions.
AS are generally considered to have a low potential for bioconcentration in aquatic organisms
For alkane sulfonates: Alkane sulfonate anionics (SAS) undergo rapid primary biodegradation with Methylene Blue Active Substance (MBAS) removal higher than 90% within a few days. Removal of 96% were seen in the OECD screening test for primary biodegradation. In activated sludge simulation tests, 96% of C10-18 SAS was removed, while the parent C13-18 SAS was removed by 83-96%.
Alkyl sulfonates are not degraded under anoxic conditions
For alpha-olefin sulfonates: alpha-Olefine sulfonates (AOS) AOS undergo rapid primary biodegradability with methylene blue active substances (MBAS) removal between 95 and 100% in 2 to 8 days in river water and inoculated media. The ultimate biodegradability of AOS exceeds the pass requirements in OECD 301 tests for ready biodegradability. report 85% DOC removal in the modified OECD screening test, 85% ThOD in the closed bottle test, and 65-80% ThCO2 in the Sturm test. In activated sludge simulation tests, AOS was removed by 100% MBAS and 88% DOC. The alkene sulfonates and hydroxyalkane sulfonates in commercial AOS are both ultimately biodegraded as approximately 84% ThCO2 was obtained during degradation of C14, C16, and C18 within 27 days, whereas the corresponding 3-hydroxyalkane sulfonates were degraded by approximately 86% under the same conditions.
AOS are not readily degradable under anaerobic conditions Reports indicate a range of 31% to 43% MBAS removal under anoxic conditions indicating primary biodegradation
Ecotoxicity:
The aquatic toxicity is influenced by a number of parameters, the length of the alkyl chain being most important. The pH and temperature of water bodies can affect the EC/LC50 values for compounds that contain ammonium ions.
The most sensitive trophic level in tests on the toxicity of alkyl sulfates were invertebrates, followed by fish. Algae proved to be less sensitive. The key study for the aquatic hazard assessment is a chronic test on Ceriodaphnia dubia, which covers a range of the alkyl chain length from C12 to C18. A parabolic response was observed with the C14 chain length being the most toxic (NOEC = 0.045 mg/l).
For alkyl sulfates: Fish LC50 (96 h): fathead minnow - fry 10.2 mg/l; juvenile 17 mg/l; adult 22.5 mg/l; rainbow trout 4.6 mg/l (static)
The aquatic toxicity of AS seems to increase with increasing alkyl chain length. This has been shown for daphnids and for some fish species. An overall comparison of the acute toxicity between the primary and secondary AS shows only minor differences in the toxicity, although only a few studies for comparison are available.
The available data describing the toxicity of AS towards algae indicate that the lowest EC50 values range between 1 and 10 mg/l for C12 AS
The toxicity of AS towards invertebrates has mainly been examined in tests with Daphnia magna. The acute toxicity of AS to Daphnia magna increased with increasing alkyl chain length. It has been shown that during degradation of C12 AS, the toxicity first increased to a maximum after 30 hours and then fell to almost a negligible value. The increase in toxicity was explained by the formation of the more toxic dodecanoic acid which is rapidly transformed to other and less toxic metabolites.
Studies showed that the 24 h-LC50 values for killifish in distilled water decreased by a factor of about 10 when the alkyl chain was increased by two carbon atoms. C16 was 10 times more toxic than C14, which was about 10 times more toxic than C12 AS.
The toxicity of AS to fish has been demonstrated to increase with increasing alkyl chain length as also seen in studies with Daphnia magna. The acute toxicity on

Continued...

Daphnia magna has been determined for chain length C8-C14. Results were comparable to alkyl sulfates in the range between C8 and C10, while C12 and C14 are significantly less toxic. Chronic data obtained for C12 alkane sulfonate sodium and C12-alkyl sulfate sodium with the rotifer *Brachionus calicyflorus* similarly show that alkane sulfonates might be less toxic than alkyl sulfates. C16 and C18 alkane sulfonates are assumed to exhibit the same toxicity than alkyl sulfates of comparable chain lengths. No data are available concerning the toxicity of alkane sulfonates on fish and algae. However, a similar toxicity might be assumed because of structural and physico-chemical similarities between the three subcategories
Whereas most correlations between AS structure and toxicity show an increasing toxicity with increasing alkyl chain length, the budding in Hydra attenuata was apparently more affected by C10 AS than by C12, C14, and C16 AS. The authors suggested that the decrease in toxicity with increasing alkyl chain length was attributable to reduced solubility in water
Tests on the toxicity to microorganisms were only conducted with alkyl sulfates as test substances. A test on the inhibition of respiration of activated sludge resulted in an 3 h-EC50 of 135 mg/l (nominally). The lowest effect value for protozoa was obtained from a test on *Uronema parduczi* using C12-alkyl sulfate sodium - the 20 h-EC5 was 0.75 mg/l.
Experimental test results on benthic organisms in a water-sediment system are not available. However, due to sediment-water partitioning coefficients Kd < 350, no significant risk for organisms in this compartment is to be expected.
Data indicate that toxic effects on soil organisms might only be expected at high concentrations for alkyl sulfates. Toxicity of alkane sulfonates and alpha-olefin sulfonates can not be assessed because test results for terrestrial organisms are not available.
For alpha-olefin sulfonates, reliable short-term tests on fish, invertebrates and algae are available. The results indicate that toxicity is increasing as the alkyl chain length increases. The lowest available effect value is the 96 h-LC50 = 0.5 mg/l, determined in tests on *Oryzias latipes, Rasbora heteromorpha* and *Salmo trutta*
Algae show toxic effects to growth when exposed 10-100 mg/l for C14-18 AOS.
EC50 values for *Daphnia magna* have been determined within the range 5-50 mg/l for C14-18 AOS . Another study with *Daphnia magna*, showed EC50 values of 16.6 mg/l for C14-16 AOS and 7.7 mg/l for C16-18 AOS.
Studies performed with fish show that the higher homologues of AOS are more toxic than the lower ones. This has been illustrated for different fish species (LC50 (96 h) range 0.5-5.3 mg/l)
For alkane sulfonates: The toxicity of various SAS homologues was determined in tests with *Chlamydomonas variabilis.* After 24 hours of exposure at 20 C, there was a tendency to an increased toxicity with increasing chain length. The EC50 values were 125 mg/l for C10.3, 74.9 mg/l for C11.2, 32.4 mg/l for C14, 15.8 mg/l for C15, 9.42 mg/l for C16, 3.93 mg/l for C17, 3.71 mg/l for C18.9, and 8.47 mg/l for C20.7.

SIDS Initial Assessment Profile
Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Torben Madsen et al: Miljoministeriet (Danish Environmental Protection Agency
Bentonite and kaolin have low toxicity to aquatic species, a large number of which have been tested
A persistent herbicide (or contains the herbicide)
Certain herbicides can persist on vegetation and in the soil for months or years and these products are called "persistent herbicides". Persistent herbicides are a narrow range of herbicides used to kill broad leaf weeds and thistle that compete with grasses and grain crops. They are "persistent" because they will not be killed by the high temperatures in thermophilic composting and may take over 2 years or more to fully decay
The class of herbicides from the picolinic acid family is of the greatest concern. These chemicals are marketed for use in hayfields, horse pastures, golf courses, roadways, grain crops, and lawns, to kill unwanted broad-leaf weeds. These herbicides do not normally impact grasses and plants like corn, wheat, and oats.
Herbicides vary in their potential to persist in soil. Herbicide families that have persistent members include triazines, uracils, phenylureas, sulfonylureas, dinitroanilines, isoxazolidiones, imidazolinones, and certain plant growth regulators belonging to the pyridine family.
The length of time a herbicide remains active in soil is called "soil persistence," or "soil residual life". For some herbicides, there may be a fine line between controlling weeds for the entire growing season and then planting a sensitive rotation crop. Anything that affects the disappearance or breakdown of herbicides affects persistence.
Persistent herbicides are generally colourless and odorless. Scientific studies reveal that pass unaltered during animal digestion (including microbial digestion) when used at labelled rates. In fact, animal digestion tends to strengthen the impact of these chemicals through concentration because the animal processes the food but passes most of the chemical as a waste.
Persistent herbicide may will kill a tomato plant at a concentration of one part per billion, and impact many other garden plants as well. Plant families sensitive to persistent herbicides include:

- The nitrogen-fixing, legume family of plants such as peas, beans, lentils, and clover
- The nightshade, solanaceous family of plants such as tomatoes and potatoes
- The sunflower, composites family of plants such as sunflower, petunias, daisies, lettuce, and asters
- The cucumber, cucurbit family of plants such as cucumber, squash, pumpkin, and watermelon

Damaged plants may show on or more of the following symptoms:
Stunted growth: the main growth tip stops growing and the lateral buds begin to grow

- Reduced fruit set
- Cupping of leaves
- Failure of secondary leaves to grow after the seed leaves emerge
- In legumes, compound leaves stay single

Depending on the type of herbicide and the level of concentration in the soil, persistent herbicides can last anywhere from several months to three or more years before completely breaking down into inert compounds. The length of time depends upon a variety of factors, including the type and moisture content of the soil.
The most common feed stocks with persistent herbicide contamination are manures and bedding, but grass clippings and many food items can be contaminated.
Persistent pesticides do not break-down in the composting process. At its most fundamental level, composting is digestion by microorganisms, regardless of whether it is an aerated static pile, anaerobic digestion, etc. Simple composting math demonstrates that one ton of feedstock reduces to approximately half that amount as finished compost. Persistent herbicides break down much more slowly than the materials in compost, and therefore, most of the chemicals pass into the finished compost. Low toxicity to animals is one often cited benefit of persistent herbicides.
According to the USEPA, persistent herbicides are not harmful to people or animals when these products are used at labelled rates.
For metsulfuron methyl
Because it has residual activity in soils, it is necessary to allow ample time for metsulfuron-methyl to break down before planting certain crops (22 months for sunflowers, flax, corn, or safflower, and 10 months before planting sorghum). It should not be used on ryegrass or on pastures containing alfalfa or clovers.

Continued...

Kow 0.018 (pH 7)
log Kow : -1.854- 1
Koc: 35 (pH 7)
Half-life soil : 480-3600
Environmental fate:
Soil and water: broken down by chemical hydrolysis and microbial action.
DT50: 1-5 weeks. Breaks down more rapidly at lower pHs, higher temperatures and higher soil moisture levels.
Breakdown of Chemical in Soil and Groundwater: The breakdown of metsulfuron-methyl in soils is largely dependant on soil temperature, moisture content, and pH. The chemical will degrade faster under acidic conditions, and in soils with higher moisture content and higher temperature . Metsulfuron-methyl has a higher mobility potential in alkaline soils than in acidic soils, as it is more soluble under alkaline conditions. Metsulfuron-methyl is stable to photolysis, but will break down in ultraviolet light. Half-life estimates for metsulfuron-methyl in soil are wide ranging from 14 - 180 days, with an overall average of reported values of 30 days . Reported half-life values (in days) for soil include: clay - 178 ; sandy loam - 102 ; clay loam - 70 , 14-28 , 14-105 ; silty loam - 120-180 .
Breakdown of Chemical in Surface Water: The dissipation time for metsulfuron-methyl was investigated in a mixed wood/boreal forest lake. The DT50 or length of time required for half of the material to dissipate in water was >84 days when high concentrations of metsulfuron-methyl were applied, and 29.1 days at concentrations that might be expected if the chemical is applied for forestry uses . The chemical is stable to hydrolysis at neutral and alkaline pHs, and has a half-life of 3 weeks at pH 5.0, 25 degrees C and >30 days at 15 degrees C .
Breakdown of Chemical in Vegetation: Metsulfuron-methyl is rapidly taken up by plants at the roots and on foliage. The chemical is translocated throughout the plant, but is not persistent. It is broken down to non-herbicidal products in tolerant plants. It undergoes complete degradation within days by hydroxylation, conjugation. Metabolites include the hydroxymethyl analogue, methyl 2-(aminosulfonyl)benzoate and 2-(aminosulfonyl)benzoic acid. Cereal plants metabolise the material quickly.
Ecotoxicology:
Birds: Acute oral LD50 for mallard ducks >5000 mg/kg
Eight-day dietary LC50 for mallard ducks and bobwhite quail: >5620 mg/kg
Fish LC50 (96 h) for rainbow trout and bluegill sunfish >150 mg/l
Bees: Non-toxic to bees: LD50 >25 ug/bee
Daphnia EC50 (48 h): >150 mg/l
Extensive studies have been conducted to evaluate the effects of lignosulfonate on the environment. Results show that they are not harmful to plants, animals or aquatic life. Toxic levels of lignosulfonates in surface water have been established and confirm that concentrations must be relatively high before fish and other organisms are affected. Fish LC50s are calculated to be 5200-6400 ppm (practically non-toxic). Vegetation and growth of fir trees were not significantly affected at normal and above normal application rates of lignosulfonates.
There may be concerns associated with increased oxygen demand in waters where lignosulfonates are introduced directly into a stream from pulping plants. Receiving water near pulping plants experiences an increased oxygen demand and the water takes on a yellowish-brown tint . Effluent from pulping plants is often up to 55% lignosulfonates .
The the effect of lignosulfonates on bacterial reproduction rate in recirculated cooling water was studied by plate counting method. It was found that both wood-pulp and straw-pulp lignosulfonates hasten bacterial reproduction. Sugar in lignosulfonates was the main factor that hasten bacterial reproduction. However,modified lignosulfonates inhibit bacterial reproduction. Lignosulfonate oxidised by hydrogen peroxide also inhibited bacterial reproduction. It was determined that except under extreme soil pH conditions (<1), the release of sulfur dioxide from the application of lignosulfonates is essentially non existent. The odour associated with the application of lignosulfonates to roads (as dust suppressant) was found to be due to trace amounts of organic compounds mainly of the furfural variety.
There appears to be little if any research describing movement of lignosulfonates in the soil and whether they can leach into surface or ground water
Lignosulfonate is a complex mixture of small- to moderate-sized polymeric compounds with sulfonate groups attached to the molecule. The product commonly designated “lignosulfonate” is really a mixture of sulfonated lignin, sugars, sugar acids, resins and inorganic chemicals. This complex and variable mixture is water soluble.
Lignosulfonate is a highly anionic polymer used to deflocculate clay-based muds; in addition it is used as a cement additive where it binds to the surface of cement particles and delays absorption of water and this influence hardening rates. Lignosulfonate is a byproduct of the sulfite method for manufacturing paper from wood pulp. Lignosulfonates form colloidal solutions or dispersions with water but are not soluble in organic solvents. Lignosulfonates are biopolymers; they are salts of lignosulfonic acid that has been formed when pulp is manufactured by the sulphite method. The lignosulfonates are of varied composition because the woods are different, the extent of the lignin degradation can be different and a different number of sulfonic groups can have been added. Lignin is a polymer with a most varied length and composition, a fundamental structure being hydroxyphenyl propane. It contains many phenolic rings and methoxy groups.
Lignosulfonate is derived from wood and thus contain trace amounts of the metals that are naturally present in trees. The levels and types of metals vary depending upon the types of trees and the soil on which they were grown. The amounts of metals typically found in lignosulfonate are well below one part per million (ppm).
Pyridine and its derivatives:
Environmental fate:
The atmospheric photodegradation estimates for the Pyridine and Pyridine Derivatives Category chemicals indicate that piperidine which is the lower molecular weight, non-aromatic and unsubstituted chemical in the category, would be expected to degrade rapidly (t1/2 < 1 day) when exposed to UV light in the atmosphere. Pyridine and the three methyl derivatives of pyridine (picolines) which are the higher molecular weight, aromatic and substituted chemicals in the category, would be expected to photodegrade more slowly (t1/2 . 30 or 10 days, respectively). , Lutidines, and collidines are expected to photodegrade even more slowly. The nitriles derivatives of pyridine are also predicted to photodegrade more slowly (t1/2 . 164 days). However, the nitrile derivatives of pyridine were predicted to partition to air much less favorably than to soil and water. As molecular weight and substitution increase in the category, greater distribution to water and soil and less to air is predicted. This trend is consistent with the vapor pressure data.
Pyridines are not expected to hydrolysis in the environment because they lack a potentially hydrolysable functional group.
There are adequate measured data across the pyridine group to allow the conclusion that these chemicals are biodegradable in the presence of adequate oxygen and bacteria; however, they are relatively stable under anaerobic and/or sterile environments**.**
Depending upon the environmental conditions, different types of bacteria, fungi, and enzymes are involved in the degradation process of these compounds .
Different organisms are using different pathways to biotransform a substrate The transformation rate of the pyridine derivatives is dependent on the substituents .
For example, pyridine carboxylic acids have the highest transformation rate followed by mono-hydroxypyridines, methylpyridines, aminopyridines, and halogenated pyridines
Ecotoxicity:

Continued...

Measured values for acute aquatic toxicity indicate that the Pyridine and Pyridine Derivatives Category chemicals are slightly to moderately toxic to fish, invertebrates and algae. Modeled data for acute aquatic toxicity were generally consistent with the reliable measured values in cases for which both existed.
The triazine pesticides behave as weak bases in aqueous solution. Triazines are more soluble at low pHs. Adsorption of triazines through an exchange process to organic matter and clay minerals is dependent on the pH of the solution and the acidity of the absorbent surface. Hydrogen bonding and hydrophobic bonding also occur with soil organic matter at higher pHs. Hydrolysis and oxidation are general routes of soil metabolism whilst photodecomposition appears to be minimal.
Vapour transport losses are dependent on vapour pressure and the pH of the evaporating surface as ionised compounds are less volatile. Transport from soil to water occurs in solution and in sediments. Herbicide concentrations in excess of 5 ppb may play a part in the decline in submerged aquatic vegetation (SAV). However, recovery from exposure to these concentrations does occur as these herbicides degrade rapidly under estuarine conditions. Residues do not appear to build up in sediments.
Chelating agents might reduce the elimination of heavy metals by adsorption on activated sludge. A remobilisation of heavy metals out of river sediment might be expected.
Polyanionic monomers, such as ethylenediaminetetraacetic acid (EDTA), are toxic to green algae. Toxicity to algae is moderate and it appears that the mode of toxic action of these polyanionic monomers is overchelation of nutrient elements needed by algae for growth. Polyanionic monomers are assessed similarly to the polycarboxylic acid polymers.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
metsulfuron methyl	HIGH	HIGH
sodium carbonate	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
metsulfuron methyl	LOW (LogKOW = 1.7626)
sodium carbonate	LOW (LogKOW = -0.4605)

Mobility in soil

Ingredient	Mobility
metsulfuron methyl	LOW (KOC = 391.1)
sodium carbonate	HIGH (KOC = 1)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ **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	2Z

Land transport (ADG)

UN number	3077
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains metsulfuron methyl and aminopyralid-potassium)

Continued...

Transport hazard class(es)	Class: 9 Subrisk: Not Applicable
Packing group	III
Environmental hazard	Environmentally hazardous
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)

UN number	3077
UN proper shipping name	Environmentally hazardous substance, solid, n.o.s. * (contains metsulfuron methyl and aminopyralid-potassium)
Transport hazard class(es)	ICAO/IATA Class: 9 ICAO / IATA Subrisk: Not Applicable ERG Code: 9L
Packing group	III
Environmental hazard	Environmentally hazardous
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)

UN number	3077
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains metsulfuron methyl and aminopyralid-potassium)
Transport hazard class(es)	IMDG Class: 9 IMDG Subrisk: Not Applicable
Packing group	III
Environmental hazard	Marine Pollutant
Special precautions for user	EMS Number: F-A , S-F Special provisions: 274 335 966 967 969 Limited Quantities: 5 kg

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

Not Applicable

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

Product name	Group
aminopyralid-potassium	Not Available
metsulfuron methyl	Not Available
kaolin	Not Available
sodium carbonate	Not Available

Continued...

Product name	Group
sodium lignosulfonate, sulfomethylated	Not Available
sodium methyl naphthalenesulfonate	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
aminopyralid-potassium	Not Available
metsulfuron methyl	Not Available
kaolin	Not Available
sodium carbonate	Not Available
sodium lignosulfonate, sulfomethylated	Not Available
sodium methyl naphthalenesulfonate	Not Available

SECTION 15 Regulatory information

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

aminopyralid-potassium is found on the following regulatory lists

Not Applicable

metsulfuron methyl is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

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)

sodium carbonate 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 10 / Appendix C
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)

sodium lignosulfonate, sulfomethylated is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

sodium methyl naphthalenesulfonate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (aminopyralid-potassium; metsulfuron methyl)
Canada - DSL	No (aminopyralid-potassium; metsulfuron methyl)
Canada - NDSL	No (aminopyralid-potassium; metsulfuron methyl; kaolin; sodium carbonate; sodium lignosulfonate, sulfomethylated; sodium methyl naphthalenesulfonate)
China - IECSC	No (aminopyralid-potassium)
Europe - EINEC / ELINCS / NLP	No (aminopyralid-potassium; metsulfuron methyl; sodium lignosulfonate, sulfomethylated)
Japan - ENCS	No (aminopyralid-potassium; metsulfuron methyl; kaolin; sodium lignosulfonate, sulfomethylated)
Korea - KECI	No (aminopyralid-potassium; metsulfuron methyl)
New Zealand - NZIoC	Yes

Continued...

National Inventory	Status
Philippines - PICCS	No (aminopyralid-potassium; metsulfuron methyl; sodium methyl naphthalenesulfonate)
USA - TSCA	No (aminopyralid-potassium; metsulfuron methyl)
Taiwan - TCSI	No (aminopyralid-potassium)
Mexico - INSQ	No (aminopyralid-potassium; sodium methyl naphthalenesulfonate)
Vietnam - NCI	Yes
Russia - FBEPH	No (aminopyralid-potassium; metsulfuron methyl; sodium lignosulfonate, sulfomethylated; sodium methyl naphthalenesulfonate)
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	13/01/2022
Initial Date	13/01/2022

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
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