



YaraMila 12-10-10 Ballance Agri-Nutrients

Chemwatch Hazard Alert Code: 2

Chemwatch: 5174-85
Version No: 5.1.7.10
Safety Data Sheet according to the Health and Safety at Work (Hazardous Substances) Regulations 2017

Issue Date: 01/11/2019
Print Date: 10/09/2021
L.GHS.NZL.EN

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

Product Identifier

Product name	YaraMila 12-10-10
Chemical Name	Not Applicable
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	Fertiliser
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Details of the supplier of the safety data sheet

Registered company name	Ballance Agri-Nutrients
Address	161 Hewletts Rd Mount Maunganui New Zealand
Telephone	+64 800 222 090
Fax	Not Available
Website	Not Available
Email	customerservices-mount@ballance.co.nz

Emergency telephone number

Association / Organisation	CHEMCALL
Emergency telephone numbers	Freephone: 0800 CHEMCALL (0800 243 622) (24 Hours/ 7 Days)
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

Considered a Hazardous Substance according to the criteria of the New Zealand Hazardous Substances New Organisms legislation. Not regulated for transport of Dangerous Goods.

ChemWatch Hazard Ratings

	Min	Max	
Flammability	0		
Toxicity	1		
Body Contact	2		
Reactivity	0		
Chronic	0		

0 = Minimum
1 = Low
2 = Moderate
3 = High
4 = Extreme

Classification [1]	Skin Corrosion/Irritation Category 3, Serious Eye Damage/Eye Irritation Category 2, Hazardous to the Aquatic Environment Acute Hazard Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 4, Hazardous to Terrestrial Vertebrates, Acute Toxicity (Oral) Category 4
Legend:	1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI
Determined by Chemwatch using GHS/HSNO criteria	6.1D (oral), 6.3B, 6.4A, 9.1D, 9.3C

Label elements

Hazard pictogram(s)	
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Signal word	Warning
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Hazard statement(s)

H316	Causes mild skin irritation.
H319	Causes serious eye irritation.
H401	Toxic to aquatic life.
H413	May cause long lasting harmful effects to aquatic life.
H433	Hazardous to terrestrial vertebrates.
H302	Harmful if swallowed.

Precautionary statement(s) Prevention

P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P280	Wear protective gloves, protective clothing, eye protection and face protection.

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.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P330	Rinse mouth.

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
7722-76-1	25-<30	<u>ammonium phosphate, monobasic</u>
7447-40-7	15-<20	<u>potassium chloride</u>
6484-52-2	15-<20	<u>ammonium nitrate</u>
7783-28-0	7-<10	<u>diammonium phosphate</u>
7783-20-2	7-<10	<u>ammonium sulfate</u>
7757-93-9	3-<5	<u>calcium phosphate, dibasic</u>
7778-77-0	3-<5	<u>potassium phosphate, monobasic</u>
7778-18-9	1-<2	<u>calcium sulfate</u>
12125-02-9	1-<2	<u>ammonium chloride</u>

Legend: 1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 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	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay.
Ingestion	<ul style="list-style-type: none"> ▶ 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 phosphate salts intoxication:

- ▶ All treatments should be based on observed signs and symptoms of distress in the patient. Consideration should be given to the possibility that overexposure to materials other than this product may have occurred.
- ▶ Ingestion of large quantities of phosphate salts (over 1.0 grams for an adult) may cause an osmotic catharsis resulting in diarrhoea and probable abdominal cramps. Larger doses such as 4-8 grams will almost certainly cause these effects in everyone. In healthy individuals most of the ingested salt will be excreted in the faeces with the diarrhoea and, thus, not cause any systemic toxicity. Doses greater than 10 grams hypothetically may cause systemic toxicity.
- ▶ Treatment should take into consideration both anionic and cation portion of the molecule.
- ▶ All phosphate salts, except calcium salts, have a hypothetical risk of hypocalcaemia, so calcium levels should be monitored.

The toxicity of nitrates and nitrites result from their vasodilating properties and their propensity to form methaemoglobin.

- ▶ Most produce a peak effect within 30 minutes.
- ▶ Clinical signs of cyanosis appear before other symptoms because of the dark pigmentation of methaemoglobin.
- ▶ Initial attention should be directed towards improving oxygen delivery, with assisted ventilation, if necessary. Hyperbaric oxygen has not demonstrated conclusive benefits.
- ▶ Institute cardiac monitoring, especially in patients with coronary artery or pulmonary disease.
- ▶ Hypotension should respond to Trendelenburg's position and intravenous fluids; otherwise dopamine may be needed.
- ▶ Naloxone, glucose and thiamine should be given if a multiple ingestion is suspected.
- ▶ Decontaminate using Ipecac Syrup for alert patients or lavage for obtunded patients who present within 2-4 hours of ingestion.
- ▶ Symptomatic patients with methaemoglobin levels over 30% should receive methylene blue. (Cyanosis alone, is not an indication for treatment). The usual dose is 1-2 mg/kg of a 1% solution (10 mg/ml) IV over 5 minutes; repeat, using the same dose if symptoms of hypoxia fail to subside within 1 hour.

[Ellenhorn and Barceloux: Medical Toxicology]

BIOLOGICAL EXPOSURE INDEX - BEI

These represent the determinants observed in specimens collected from a healthy worker who has been exposed at the Exposure Standard (ES or TLV):

Determinant	Index	Sampling Time	Comments
1. Methaemoglobin in blood	1.5% of haemoglobin	During or end of shift	B,NS,SQ

B: Background levels occur in specimens collected from subjects **NOT** exposed

NS: Non-specific determinant; also observed after exposure to other materials

SQ: Semi-quantitative determinant - Interpretation may be ambiguous; should be used as a screening test or confirmatory test.

SECTION 5 Firefighting measures

Extinguishing media

- ▶ There is no restriction on the type of extinguisher which may be used.
- ▶ Use extinguishing media suitable for surrounding area.

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ 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.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Non combustible. ▶ Not considered a significant fire risk, however containers may burn. <p>Decomposition may produce toxic fumes of:</p> <p>hydrogen chloride nitrogen oxides (NOx) phosphorus oxides (POx) sulfur oxides (SOx) metal oxides</p> <p>May emit poisonous fumes. May emit corrosive fumes.</p>

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	<ul style="list-style-type: none"> ▶ 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.
Major Spills	<p>Moderate hazard.</p> <ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ 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. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	<ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ Store in a cool, dry area protected from environmental extremes. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. <p>For major quantities:</p> <ul style="list-style-type: none"> ▶ Consider storage in banded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams). ▶ Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Glass container is suitable for laboratory quantities ▶ Polyethylene or polypropylene container. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Avoid strong bases. <p>Ammonium nitrate:</p> <ul style="list-style-type: none"> ▶ is a strong oxidiser ▶ reacts violently and/ or forms explosive mixtures with hot water, reducing agents, combustible materials, flammable liquids, organic materials, ammonium dichromate, barium chloride, barium nitrate, charcoal, cyanoguanidine, oils, phosphorus, potassium chromate, potassium dichromate, potassium nitrate, potassium permanganate, sodium chloride, finely divided metals ▶ forms explosive and/ or heat- and shock- sensitive compounds with acetic acid, alkali metals (potassium, sodium etc.), ammonia, nitric acid, sodium hypochlorite, sulfur, urea ▶ may explode violently when heated and contained or confined <p>NOTE:- Explosive detonations can occur when material is mixed with organic material, oils or charcoals and when heated or subjected to shock.</p> <p>Diammonium phosphate (syn: ammonium phosphate, dibasic)</p> <ul style="list-style-type: none"> ▶ slowly forms anhydrous ammonia on contact with air ▶ forms anhydrous ammonia gas on contact with caustics ▶ reacts violently with strong oxidisers, magnesium, potassium chlorate, strong bases ▶ reacts with antimony(V) pentafluoride, lead diacetate, magnesium, silver nitrate, zinc acetate <p>Ammonium sulfate:</p> <ul style="list-style-type: none"> ▶ is strongly acid in aqueous solution ▶ reacts with caustics forming ammonia ▶ reacts violently with potassium chlorate

- ▶ when hot reacts with nitrates, nitrites, chlorates
- ▶ attacks metals
- ▶ is incompatible with sulfuric acid, aliphatic amines, alkanolamines, amides, organic anhydrides, isocyanates, vinyl acetate, alkylene oxides, epichlorohydrin, potassium plus ammonium nitrate, sodium-potassium powder plus ammonium nitrate
- ▶ mixtures with sodium hypochlorite form unstable, explosive nitrogen trichloride
- ▶ Phosphates are incompatible with oxidising and reducing agents.
- ▶ Phosphates are susceptible to formation of highly toxic and flammable phosphine gas in the presence of strong reducing agents such as hydrides.
- ▶ Partial oxidation of phosphates by oxidizing agents may result in the release of toxic phosphorus oxides.



X — Must not be stored together

O — May be stored together with specific preventions

+ — May be stored together

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
New Zealand Workplace Exposure Standards (WES)	calcium sulfate	Calcium sulphate (Gypsum, Plaster of Paris)	10 mg/m ³	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	calcium sulfate	Plaster of Paris (Calcium sulphate)	10 mg/m ³	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	ammonium chloride	Ammonium chloride fume	10 mg/m ³	20 mg/m ³	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
ammonium phosphate, monobasic	17 mg/m ³	190 mg/m ³	1,100 mg/m ³
ammonium nitrate	6.7 mg/m ³	73 mg/m ³	440 mg/m ³
diammonium phosphate	20 mg/m ³	210 mg/m ³	1,300 mg/m ³
ammonium sulfate	13 mg/m ³	140 mg/m ³	840 mg/m ³
potassium phosphate, monobasic	9.6 mg/m ³	110 mg/m ³	630 mg/m ³
ammonium chloride	20 mg/m ³	54 mg/m ³	330 mg/m ³

Ingredient	Original IDLH	Revised IDLH
ammonium phosphate, monobasic	Not Available	Not Available
potassium chloride	Not Available	Not Available
ammonium nitrate	Not Available	Not Available
diammonium phosphate	Not Available	Not Available
ammonium sulfate	Not Available	Not Available
calcium phosphate, dibasic	Not Available	Not Available
potassium phosphate, monobasic	Not Available	Not Available
calcium sulfate	Not Available	Not Available
ammonium chloride	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
ammonium phosphate, monobasic	E	≤ 0.01 mg/m ³
ammonium nitrate	E	≤ 0.01 mg/m ³
diammonium phosphate	E	≤ 0.01 mg/m ³
ammonium sulfate	E	≤ 0.01 mg/m ³
calcium phosphate, dibasic	E	≤ 0.01 mg/m ³

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

For ammonium chloride:

Based on moderate inhalation the TLV-TWA is thought to be protective against irritation of the respiratory tract for calcium sulfate:

The TLV-TWA is thought to be protective against the significant risks of eye, skin and other physical irritation.

Exposure controls

<p>Appropriate engineering controls</p>	<p>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.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>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.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <ul style="list-style-type: none"> ▶ Local exhaust ventilation is required where solids are handled as powders or crystals; even when particulates are relatively large, a certain proportion will be powdered by mutual friction. ▶ If in spite of local exhaust an adverse concentration of the substance in air could occur, respiratory protection should be considered. <p>Such protection might consist of:</p> <p>(a): particle dust respirators, if necessary, combined with an absorption cartridge;</p> <p>(b): filter respirators with absorption cartridge or canister of the right type;</p> <p>(c): fresh-air hoods or masks.</p> <p>Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p> <table border="1" data-bbox="383 795 1484 940"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="383 974 1117 1142"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 4-10 m/s (800-2000 f/min) for extraction of crusher dusts generated 2 metres distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	Type of Contaminant:	Air Speed:	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use	4: Large hood or large air mass in motion	4: Small hood-local control only
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<p>Personal protection</p>																	
<p>Eye and face protection</p>	<ul style="list-style-type: none"> ▶ 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] 																
<p>Skin protection</p>	<p>See Hand protection below</p>																
<p>Hands/feet protection</p>	<p>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.</p> <p>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.</p> <p>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.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> - frequency and duration of contact, - chemical resistance of glove material, - glove thickness and - dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> - 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. <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p>																

	<ul style="list-style-type: none"> Excellent when breakthrough time > 480 min Good when breakthrough time > 20 min Fair when breakthrough time < 20 min Poor when glove material degrades <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>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.</p> <p>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.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> 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 <p>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.</p> <p>Experience indicates that the following polymers are suitable as glove materials for protection against undissolved, dry solids, where abrasive particles are not present.</p> <ul style="list-style-type: none"> polychloroprene. nitrile rubber. butyl rubber. fluorocacoutchouc. polyvinyl chloride. <p>Gloves should be examined for wear and/ or degradation constantly.</p>
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> Overalls. P.V.C apron. Barrier cream. Skin cleansing cream. Eye wash unit.

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 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.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Beige/ grey/ white granules with no odour; soluble in water.		
Physical state	Divided Solid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature	>210
Melting point / freezing point (°C)	>210	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 Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available

YaraMila 12-10-10

Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Applicable
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (%)	>4.5 (10.5%)
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	<p>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.</p> <p>Levels above 10 ug/m3 of suspended inorganic sulfates in the air may cause an excess risk of asthmatic attacks in susceptible persons. 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.</p> <p>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.</p>
Ingestion	<p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>The principal concern with exposure to inorganic nitrate is its biological reduction to reactive and toxic nitrite. Nitrate itself is relatively harmless. Where bacteria are present and the environment is anaerobic, nitrate can be reduced to nitrite. The main site for this reaction is mouth and stomach, but nitrite formation in the lower intestine and in the bladder (urinary infection) may also be of some toxicological importance.</p> <p>Adults have tolerated large doses of nitrate as sodium and ammonium salt (> 100 mg NO₃-/kg) in some cases repeated for several days for medical or experimental purposes with only minor effects in some subjects (light methaemoglobinaemia, diarrhoea, vomiting). Death and severe effects of nitrate ingestion are generally associated with doses above 10 g NO₃-. Doses between 2 and 9 g NO₃- have been reported to cause methaemoglobinaemia. These values correspond to 33 to 150 mg NO₃-/kg</p> <p>The half-life in the body for an oral dose of nitrate to be approximately 5 hours. As blood absorption depends on food matrix and route of exposure, and as larger doses may increase the urinary excretion rate, the biological half-life for both nitrate and nitrite should be expected to be 3 to 8 hours. Nitrate does not accumulate in the body.</p> <p>The major acute toxic effect of nitrate and nitrite poisoning is methaemoglobinaemia.</p> <p>Swallowing large doses of ammonium nitrate may cause dilation of blood vessels by direct smooth muscle relaxation and methaemoglobinaemia. Symptoms include dizziness, abdominal pain, vomiting, bloody diarrhoea, weakness, narcosis, convulsions and collapse. Other effects of exposure include headache, warm flushed skin, nausea, vomiting, diuresis and fatigue. Both tachycardia and bradycardia, atrial fibrillation, cardiac ischaemia, frequent ventricular premature beats and bigeminy have been reported. Severe poisonings have produced hypotension, decreased peripheral vascular resistance, cardiovascular collapse, convulsions and coma</p> <p>Human metabolism allows detoxification of ammonia, however toxic effects appear if this mechanism is overwhelmed by other than small doses. Ingestion of ammonium salts may produce local irritation, nausea, vomiting and diarrhoea. Very large doses of ammonium salts may produce a drop in blood pressure, collapse, central nervous system disorders, spasms, narcosis, respiratory paralysis and haemolysis.</p> <p>Large doses of ammonium salts may be sufficiently absorbed to produce diuresis and systemic ammonia poisoning. Such poisonings have been described after parenteral administration of the salts and produce flaccidity of facial muscles, tremor, generalised discomfort, anxiety and impairment of motor performance, recognition and of critical flicker fusion. Such a clinical picture resembles that found in terminal liver failure - elevated levels of ammonia are found regularly in advanced liver disease.</p>
Skin Contact	<p>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.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</p> <p>Irritation and skin reactions are possible with sensitive skin</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>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.</p>
Eye	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals.</p> <p>Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>

Chronic	<p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>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.</p> <p>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.</p> <p>Chronic exposure to ammonium nitrate may produce hypotension and fatigue. Chronic ingestion of 6-12 grams per day has produced gastritis, acidosis, isosmotic diuresis and nitrite toxicity manifested by methaemoglobinaemia or vasodilation</p>
	<p>Long term exposure to high dust concentrations may cause changes in lung function (i.e. pneumoconiosis) caused by particles less than 0.5 micron penetrating and remaining in the lung. A prime symptom is breathlessness. Lung shadows show on X-ray.</p> <p>The major concern of possible long-term effects of exposure to nitrate and nitrite is associated with formation of nitroso compounds, many of which are carcinogenic. This formation may take place wherever nitrite and nitrosable compounds are present, but it is favoured by acidic conditions or the presence of some bacteria. The gastrointestinal tract and especially the stomach is regarded as the main formation site, but nitrosation reactions can also take place in an infected urinary bladder.</p> <p>Nitrite is mutagenic in a number of in vitro assays against microorganisms or cultured mammalian cells. Nitrates show no mutagenic activity in microbial tests under aerobic conditions. Activity has been reported under anaerobic conditions, probably due to reduction of nitrate into nitrite</p> <p>The mutagenic effects of nitrites were observed in an in vivo and in vitro experiment using Syrian hamsters. In vivo assays have been equivocal, both positive and negative results having been reported</p> <p>Exposure to sodium nitrite in drinking water resulted in an increased incidence of epithelial hyperplasia in the forestomach of male and female rats and in the glandular stomach of male mice.</p> <p>There was equivocal evidence of carcinogenic activity of sodium nitrite in female B6C3F1 mice based on the positive trend in the incidences of squamous cell papillomas or carcinomas (combined) of the forestomach. There was no evidence of carcinogenic activity in male and female F344/N rats or B6C3F1 male mice exposed to 750, 1500 or 3000 ppm.</p> <p>NTP Technical Report Series No. 495, May 2001</p> <p>Under certain conditions, nitrites can react with secondary amines, either alone or in biological systems, to form carcinogenic nitrosamines.</p> <p>Sodium nitrite (60 mg/kg) administered in drinking water to pregnant guinea pigs produced maternal anaemia and increased the incidences of abortion and foetal mortality. Administration of 2000-3000 mg/l sodium nitrite in drinking water, to pregnant rats, produced 30-53% foetal mortality. In rat dams given 0.025-0.5% in feed, sodium nitrite caused an increase in foetal and pup mortality and decreases in pre-weanling body weights.</p> <p>Dogs given daily doses of sodium phosphate dibasic for 9-22 weeks showed calcium deposits in the kidneys (nephrocalcinosis) with disseminated atrophy of the proximal tubule. Animals fed on sodium phosphate dibasic and potassium dihydrogen phosphate, in both short- and long-term studies, showed increased bone porosity; hyperparathyroidism and soft tissue calcification were also evident.</p>

YaraMila 12-10-10	TOXICITY	IRRITATION
	Not Available	Not Available
ammonium phosphate, monobasic	TOXICITY	IRRITATION
	dermal (rat) LD50: >5000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation(Rat) LC50; >5 mg/l4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral(Rat) LD50; >2000 mg/kg ^[1]	
potassium chloride	TOXICITY	IRRITATION
	Oral(Mouse) LD50; ~117 mg/kg ^[1]	Eye (rabbit): 500 mg/24h - mild
ammonium nitrate	TOXICITY	IRRITATION
	dermal (rat) LD50: >5000 mg/kg ^[1]	Not Available
	Inhalation(Rat) LC50; >88.8 mg/l4h ^[2]	
	Oral(Rat) LD50; 2462 mg/kg ^[2]	
diammonium phosphate	TOXICITY	IRRITATION
	dermal (rat) LD50: >5000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation(Rat) LC50; >5 mg/l4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral(Rat) LD50; >2000 mg/kg ^[1]	
ammonium sulfate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral(Mouse) LD50; 610 mg/kg ^[2]	Skin: no adverse effect observed (not irritating) ^[1]
calcium phosphate, dibasic	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: <7940 mg/kg ^[2]	Eye (rabbit): 8 on a scale of 110
	Inhalation(Rat) LC50; >2.6 mg/l4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral(Rat) LD50; ~7940 mg/kg ^[1]	Skin (rabbit): 0 on a scale of 8
		Skin: no adverse effect observed (not irritating) ^[1]
potassium phosphate, monobasic	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >300 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation(Rat) LC50; >0.83 mg/l4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral(Rat) LD50; >500 mg/kg ^[1]	

calcium sulfate	TOXICITY	IRRITATION
	Inhalation(Rat) LC50; >3.26 mg/4h ^[1] Oral(Rat) LD50; >1581 mg/kg ^[1]	Not Available
ammonium chloride	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1] Oral(Mouse) LD50; 1300 mg/kg ^[2]	Eye (rabbit): 100 mg SEVERE Eye (rabbit): 500 mg/24h 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	

POTASSIUM CHLORIDE	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
AMMONIUM SULFATE	<p>for ammonium sulfate</p> <p>As ammonium sulfate dissociates in biological systems studies with other ammonium and sulfate salts can be used to cover these endpoints: A screening study according to OECD TG 422 with ammonium phosphate as analogue substance, which forms ammonium ions in aqueous solutions is available. Fully valid fertility studies with analogue compounds containing sulfate ions are however lacking. Two limited studies with sodium sulfate can be used for assessment of fertility and developmental toxicity, however, in none of these studies have the foetuses been examined histologically. There are no in vivo data on genotoxicity for ammonium sulfate. To bridge the data gap, data for ammonium chloride, which dissociates in aqueous media to form ammonium ions, as does ammonium sulfate, will be used.</p> <p>In aqueous media, ammonium sulfate dissociates in the ammonium and sulfate ions (NH₄⁺, SO₄²⁻). These can be taken up into the body by the oral and respiratory routes. Absorbed ammonium is transported to the liver and there metabolised to urea and excreted via the kidneys. Ammonium is also an endogenous substance that serves a major role in the maintenance of the acid-base balance. Minor amounts of ammonium nitrogen are incorporated in the physiological N-pool. Sulfate is a normal intermediate in the metabolism of endogenous sulfur compounds, and is excreted unchanged or in conjugated form in urine.</p> <p>Acute toxicity: Ammonium sulfate is of relatively low acute toxicity (LD50, oral, rat: 2000 - 4250 mg/kg bw; LD50 dermal, rat/mouse > 2000 mg/kg bw; 8-h LC50, inhalation, rat > 1000 mg/m³). Clinical signs after oral exposure included staggering, prostration, apathy, and laboured and irregular breathing immediately after dosing at doses near to or exceeding the LD50 value. In humans, inhalation exposure to 0.1-0.5 mg ammonium sulfate/m³ aerosol for two to four hours produced no pulmonary effects. At 1 mg ammonium sulfate/m³ very slight pulmonary effects in the form of a decrease in expiratory flow, in pulmonary flow resistance and dynamic lung compliance were found in healthy volunteers after acute exposure.</p> <p>Neat ammonium sulfate was not irritating to the skin and eyes of rabbits. There is no data on sensitisation available.</p> <p>Repeat dose toxicity: A 14-day inhalation study on rats exposed to 300 mg/m³, the only tested dose, did not report histopathological changes in the lower respiratory tract. As the respiratory tract is the target organ for inhalation exposure, the NOEL for toxicity to the lower respiratory tract is 300 mg/m³.</p> <p>The NOAEL after feeding diets containing ammonium sulfate for 13 weeks to rats was 886 mg/kg bw/day. The only toxicity sign found was diarrhea in male animals of the high-dose group (LOAEL: 1792 mg/kg bw/day).</p> <p>Reproductive toxicity: There are no valid studies available on the effects of ammonium sulfate on fertility and development. Based on data from a similar ammonium compound (diammonium phosphate), which has been tested up to 1500 mg/kg bw in a screening study according to OECD TG 422 in rats it can be concluded that ammonium ions up to the dose tested have no negative effects on fertility. In the 13-week feeding study of ammonium sulfate with rats, no histological changes of testes were observed up to 1792 mg/kg bw. The ovaries were not examined. Fully valid studies with sulfate on fertility are not available.</p> <p>In a limited study (pretreatment time short, low number of animals, no fertility indices measured) where female mice were treated with up to ca. 6550 mg sulfate/kg bw (as sodium sulfate) no effects on litter size were found.</p> <p>Developmental toxicity: Studies of developmental toxicity for ammonium sulfate are not available. In the screening study according to OECD TG 422 with up to 1500 mg diammonium phosphate/kg bw no effects on development have been detected in rats. In another limited screening study with exposure of mice to a single dose of 2800 mg sodium sulfate/kg bw no macroscopic effects or adverse effects on body weight gain have been detected in the pups. In both studies foetuses were not examined histopathologically</p> <p>Genotoxicity: Ammonium sulfate was not mutagenic in bacteria (Ames test) and yeasts with and without metabolic activation systems. It did not induce chromosomal aberrations in mammalian or human cell cultures. No in vivo genotoxicity tests are available. Based on the negative results from in vitro studies and the negative results in the micronucleus test in vivo with ammonium chloride a mutagenic activity of ammonium sulfate in vivo is unlikely.</p> <p>Similarly to other salts, high doses of ammonium sulfate may have the capability of tumour promotion in the rat stomach; it is, however, much less potent than sodium chloride when tested under identical conditions.</p>
CALCIUM PHOSPHATE, DIBASIC	<p>for calcium:</p> <p>Toxicity from calcium is not common because the gastrointestinal tract normally limits the amount of calcium absorbed. Therefore, short-term intake of large amounts of calcium does not generally produce any ill effects aside from constipation and an increased risk of kidney stones. However, more severe toxicity can occur when excess calcium is ingested over long periods, or when calcium is combined with increased amounts of vitamin D, which increases calcium absorption. Calcium toxicity is also sometimes found after excessive intravenous administration of calcium. Toxicity is manifested by abnormal deposition of calcium in tissues and by elevated blood calcium levels (hypercalcaemia). However, hypercalcaemia is often due to other causes, such as abnormally high amounts of parathyroid hormone (PTH). Usually, under these circumstances, bone density is lost and the resulting hypercalcaemia can cause kidney stones and abdominal pain. Some cancers can also cause hypercalcaemia, either by secreting abnormal proteins that act like PTH or by invading and killing bone cells causing them to release calcium. Very high levels of calcium can result in appetite loss, nausea, vomiting, abdominal pain, confusion, seizures, and even coma.</p> <p>for calcium chloride:</p> <p>Acute toxicity: The acute oral toxicity of calcium chloride is low: LD50 in mice is 1940-2045 mg/kg bw, 3798-4179 mg/kg bw in rats, and 500-1000 mg/kg bw in rabbits. The acute oral toxicity is attributed to the severe irritating property of the original substance or its high-concentration solutions to the gastrointestinal tract. In humans, however, acute oral toxicity is rare because large single doses induce nausea and vomiting. The dermal acute toxicity is negligible: LD50 in rabbits >5000 mg/kg bw. No significant change was found by gross necropsy examination except skin lesions at or near the site of administration. Hypercalcaemia may occur only when there exists other factors that alter calcium homeostasis, such as renal inefficiency and primary hyperthyroidism.</p> <p>Irritation/corrosiveness studies conducted under OECD test guidelines indicate that calcium chloride is not/slightly irritating to skin but severely irritating to eyes of rabbits. Prolonged exposure and application of moistened material or concentrated solutions resulted in considerable skin irritation, however. Irritating effect of the substance was observed in human skin injuries caused by incidental contact with the substance or its high-concentration solutions.</p> <p>Repeat dose toxicity: A limited oral repeated dose toxicity study shows no adverse effect of calcium chloride on rats fed on 1000-2000 mg/kg bw/day for 12 months. Calcium and chloride are both essential nutrients for humans and a daily intake of more than 1000 mg each of the ions is recommended. The establishment of the ADI for calcium chloride has not been deemed necessary by JECFA (Joint FAO/WHO Expert Committee on Food Additives)</p> <p>Genotoxicity: Genetic toxicity of calcium chloride was negative in the bacterial mutation tests and the mammalian chromosome aberration test.</p> <p>Reproductive and developmental toxicity: No reproductive toxicity study has been reported. A developmental toxicity study equivalent to an</p>

	OECD Guideline study, on the other hand, reveals no toxic effects on dams or foetuses at doses up to 189 mg/kg bw/day (mice), 176 mg/kg bw/day (rats) and 169 mg/kg bw/day (rabbits).
POTASSIUM PHOSPHATE, MONOBASIC	No data of toxicological significance identified in literature search.
CALCIUM SULFATE	<p>Gypsum (calcium sulfate dihydrate) is a skin, eye, mucous membrane, and respiratory system irritant. Early studies of gypsum miners did not relate pneumoconiosis with chronic exposure to gypsum. Other studies in humans (as well as animals) showed no lung fibrosis produced by natural dusts of calcium sulfate except in the presence of silica. However, a series of studies reported chronic nonspecific respiratory diseases in gypsum industry workers in Gacki, Poland.</p> <p>Unlike other fibers, gypsum is very soluble in the body; its half-life in the lungs has been estimated as minutes. In four healthy men receiving calcium supplementation with calcium sulfate (CaSO₄·1/2H₂O) (200 or 220 mg) for 22 days, an average absorption of 28.3% was reported. Several feeding studies in pigs on the bioavailability of calcium in calcium supplements, including gypsum, have been conducted. The bioavailability of calcium in gypsum was similar to that for calcitic limestone, oyster shell flour, marble dust, and aragonite, ranging from 85 to 102%. In mice, the i.p. and intragastric LD₅₀ values were 6200 and 4704 mg/kg, respectively, for phosphogypsum (98% CaSO₄·H₂O). For Plaster of Paris, the values were 4415 and 5824, respectively. In rats, an intragastric LD₅₀ of 9934 mg/kg was reported for phosphogypsum</p> <p>Repeat dose toxicity: In a study of 241 underground male workers employed in four gypsum mines in Nottinghamshire and Sussex for a year (November 1976-December 1977), results of chest X-rays, lung function tests, and respiratory systems suggested an association of the observed lung shadows with the higher quartz content in dust rather than to gypsum; the small round opacities in the lungs were characteristic of silica exposure.</p> <p>Prophylactic examinations of workers in a gypsum extraction and production plant (dust concentration exceeded TLV 2.5- to 10-fold) reported no risk of pneumoconiosis due to gypsum exposure, while another study of gypsum manufacturing plant workers reported that chronic occupational exposure to gypsum dust had resulted in pulmonary ventilatory defect of the restrictive form.</p> <p>Three cases of idiopathic interstitial pneumonia with multiple bullae throughout the lungs were seen in Japanese schoolteachers (lifetime occupation) exposed to chalk; 2/3 of the chalk was made from gypsum and small amounts of silica and other minerals.</p> <p>In rats exposed to an aerosol of anhydrous calcium sulfate fibers (15 mg/m³) or a combination of milled and fibrous calcium sulfate (60 mg/m³) six hours per day, five days per week for three weeks, gypsum dust was quickly cleared from the lungs of via dissolution and mechanisms of particle clearance.</p> <p>In guinea pigs given intraperitoneal (i.p.) injections of gypsum (doses not provided), gypsum was absorbed followed by the dissolution of gypsum in surrounding tissues. In another study, after i.p. injection of gypsum (2 cm³ of a 5 or 10% suspension in saline) into guinea pigs, which were sacrificed at intervals up to 180 days, most of the dust was found distributed in the peritoneum of the anterior abdominal wall. Gypsum dust produced irregular and clustered nodules, which decreased in size over time.</p> <p>Direct administration of WTC PM2.5 [mostly composed of calcium-based compounds, including calcium sulfate (gypsum) and calcium carbonate (calcite)] (10, 32, or 100 µg) into the airways of mice produced mild to moderate lung inflammation and airway hyperresponsiveness at the high dose. [It was noted that WTC PM2.5 is composed of many chemical species and that their interactions may be related with development of airway hyperresponsiveness.] In female SPF Wistar rats intratracheally (i.t.) instilled with anhydrite dust (35 mg) and sacrificed three months later, an increase in total lipid or hydroxyproline content in the lungs was not observed compared to controls.</p> <p>In inhalation (nose-only) experiments in which male F344 rats were exposed to calcium sulfate fiber aerosols (100 mg/m³) for six hours per day, five days per week for three weeks, there were no effects on the number of macrophages per alveolus, bronchoalveolar lavage fluid (BALF) protein concentration, or BALF g-glutamyl transpeptidase activity (g-GT). Following three weeks of recovery, nonprotein thiol levels (NPSH), mainly glutathione, were increased in animals. In follow-up experiments, rats were exposed to an aerosol of anhydrous calcium sulfate fibers (15 mg/m³) or a combination of milled and fibrous calcium sulfate (60 mg/m³) for the same duration. Calcium levels in the lungs were similar to those of controls; however, gypsum fibers were detected in the lungs of treated animals. Significant increases in NSPH levels in BALF were observed in rats killed immediately after exposure at both doses and in recovery group animals at the higher dose. At 15 mg/m³, almost all NPSH was lost in macrophages from all treated animals (including those in recovery), but a significant decrease in extracellular g-GT activity was seen only in recovery group animals. Overall, the findings were "considered to be non-pathological local effects due to physical factors related to the shape of the gypsum fibers and not to calcium sulphate per se."</p> <p>Intratracheal administration of man-made calcium sulfate fiber (2.0 mg) once per week for five weeks resulted in no deaths or significant body weight changes in female Syrian hamsters compared to controls.</p> <p>Inflammation (specifically, chronic alveolitis with macrophage and neutrophil aggregation) was observed in the lung.</p> <p>In guinea pigs, inhalation of calcined gypsum dust (1.6 x 10⁴ particles/mL) for 44 hours per week in 5.5 days for two years, followed with or without a recovery period of up to 22 months, produced only minor effects in the lungs. There were 12 of 21 deaths over the entire experimental period. These were due to pneumonia or other pulmonary lesions; however, no significant gross signs of pulmonary disease or nodular or diffuse pneumoconiosis became significant. Beginning near 11 months, pigmentation and atelectasis were seen. During the recovery period, four of ten guinea pigs died; two died of pneumonia. Pigmentation continued in most animals but not atelectasis. Low-grade chronic inflammation, occurring in the first two months, also disappeared.</p> <p>Mercury emissions controls on coal-fired power plants have increased the likelihood of the presence of mercury in synthetic gypsum formed in wet flue gas desulfurisation (FGD) systems and the finished wallboard produced from the FGD gypsum. In a study at a commercial wallboard plant, the raw FGD gypsum, the product stucco (beta form of CaSO₄·1/2H₂O), and the finished dry wallboard each contained about 1 µg Hg/g dry weight. Total mercury loss from the original FGD gypsum content was about 0.045 g Hg/ton dry gypsum processed</p> <p>Synergistic/Antagonistic Effects: In rats, i.t. administration of anhydrite (5-35 mg) successively and simultaneously with quartz reduced the toxic effect of quartz in lung tissue. This protective effect on quartz toxicity was also seen in guinea pigs; calcined gypsum dust prevented or hindered the development of fibrosis. Natural anhydrite, however, increased the fibrogenic effect of cadmium sulfide in rats. Additionally, calcined gypsum dust had a stimulatory effect on experimental tuberculosis in guinea pigs.</p> <p>Cytotoxicity: In Syrian hamster embryo cells, gypsum (up to 10 µg/cm²) did not induce apoptosis. Negative results were also found in mouse peritoneal macrophages (tested at 150 µg/mL gypsum dust) and in Chinese hamster lung V79-4 cells (tested up to 100 µg/mL).</p> <p>Carcinogenicity: In female Sprague-Dawley rats, i.p. injection of natural anhydrite dusts from German coal mines (doses not provided) induced granulomas; whether gypsum was the causal factor was not established. In Wistar rats, four i.p. injections of gypsum (25 mg each) induced abdominal cavity tumours, mostly sarcomatous mesothelioma, in 5% of animals; first tumour was seen at 546 days. In a subsequent experiment using the same procedure, female Wistar rats exhibited the first tumour at 579 days after the last injection. Mean survival of the tumour-bearing rats (5.7% of test group) was 583 days, while mean survival of the test group was 587 days. Tumour types seen were a sarcoma having cellular polymorphism, a carcinoma, and a reticulosarcoma.</p> <p>Intratracheal administration of man-made calcium sulfate fiber (2.0 mg) once per week for five weeks produced tumours in three of 20 female Syrian hamsters observed two years later. An anaplastic carcinoma was found in the heart, and one dark cell carcinoma was seen in the kidney. Two tumours of unspecified types were observed in the rib.</p> <p>In guinea pigs, inhalation of gypsum (doses not provided) for 24 months produced no lung tumours.</p> <p>In rats, i.t. administration of gypsum (doses not provided in abstract) from FGD for up to 18 months produced no arterial blood gas changes or indications of secondary heart damage as compared to controls.</p> <p>In another study, a single i.t. dose (25 mg) of flue gas gypsum dust did not produce a pathological reaction when observed for up to 18 months. There were also no signs of developing granuloma or fibrosis of the lungs. Lead quickly accumulated in the femur after injection but was eliminated during the observation period. In the Ames test, the flue gas gypsum dust was negative.</p> <p>Genotoxicity: Calcium sulfate (up to 2.5%) was negative in Salmonella typhimurium strains TA1535, TA1537, and TA1538 and in Saccharomyces cerevisiae strain D4 with and without metabolic activation.</p> <p>Developmental toxicity: In pregnant mice, rats, and rabbits, daily oral administration of calcium sulfate (16-1600 mg/kg bw) beginning on gestation day 6 up to 18 produced no effects on maternal body weights, maternal or foetal survival, or nidation; developmental effects were also not seen.</p>

AMMONIUM CHLORIDE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
YaraMila 12-10-10 & AMMONIUM PHOSPHATE, MONOBASIC & DIAMMONIUM PHOSPHATE & AMMONIUM SULFATE & CALCIUM PHOSPHATE, DIBASIC & CALCIUM SULFATE	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, cough and mucus production.
YaraMila 12-10-10 & AMMONIUM PHOSPHATE, MONOBASIC & DIAMMONIUM PHOSPHATE	No significant acute toxicological data identified in literature search.

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

Legend: ✗ – Data either not available or does not fill the criteria for classification
 ✓ – Data available to make classification

SECTION 12 Ecological information

Toxicity

	Endpoint	Test Duration (hr)	Species	Value	Source
YaraMila 12-10-10	Not Available	Not Available	Not Available	Not Available	Not Available
ammonium phosphate, monobasic	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	72h	Algae or other aquatic plants	>100mg/l	2
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	LC50	96h	Fish	>100mg/l	2
potassium chloride	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	25h	Fish	9.319mg/L	4
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	LC50	96h	Fish	750-1020mg/l	4
	EC50	48h	Crustacea	95.3-170.7mg/l	4
ammonium nitrate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	96h	Algae or other aquatic plants	894.6mg/L	4
	NOEC(ECx)	480h	Fish	0.003mg/l	4
	LC50	96h	Fish	48.184-59.63mg/L	4
	EC50	48h	Crustacea	490mg/l	2
diammonium phosphate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	72h	Algae or other aquatic plants	>100mg/l	2
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	LC50	96h	Fish	>100mg/l	2
ammonium sulfate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	>100mg/l	2
	EC50(ECx)	24h	Fish	0.068mg/L	5
	EC50	72h	Algae or other aquatic plants	190mg/l	2
calcium phosphate, dibasic	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	34.6mg/l	2
	EC50	48h	Crustacea	60mg/l	2
	EC50(ECx)	48h	Crustacea	>2.9mg/l	2

	EC50	72h	Algae or other aquatic plants	>4.4mg/l	2
	LC50	96h	Fish	>13.5mg/l	2
	EC50	48h	Crustacea	>2.9mg/l	2
potassium phosphate, monobasic	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	72h	Algae or other aquatic plants	>100mg/l	2
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	LC50	96h	Fish	>100mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
calcium sulfate	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	0.25h	Fish	75mg/l	4
	EC50	72h	Algae or other aquatic plants	>79mg/l	2
	LC50	96h	Fish	>79mg/l	2
ammonium chloride	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>76.6mg/l	4
	LC50	96h	Fish	0.14mg/l	4
	EC50	48h	Crustacea	0.075-0.126mg/l	4
	NOEC(ECx)	Not Available	Fish	0.002mg/L	5
	EC50	96h	Algae or other aquatic plants	58.476-59.706mg/L	4
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				

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 ammonium sulfate

Environmental fate:

Based on the physico-chemical properties of ammonium sulfate, water is expected to be the main target compartment. Although ammonium sulfate can be created in the atmosphere from ammonia and sulfur dioxide, this process is limited by atmospheric sulfur dioxide, not by ammonia, which has many natural sources. Particulate ammonium sulfate is removed from air by wet and dry deposition. There is no evidence for photodegradation of ammonium sulfate.

In unsterilised soil, ammonium sulfate is mineralised fairly rapidly, and subsequently nitrified. Nitrification and denitrification processes also occur naturally in streams and rivers, as well as in many secondary sewage treatment processes.

Based on the high water solubility and the ionic nature, ammonium sulfate is not expected to adsorb or bioaccumulate to a significant extent. However, mobility in soil may be reduced through ion-ion interactions.

Ecotoxicity:

Fish LC50 (96 h): *Salmo gairdneri* (juvenile) 173 mg/l

Fish NOEC (61 d): *Oncorhynchus gorboscha* (alevins) 11 mg/l

Freshwater snail LC50 (24 h): *Helisoma trivolvis* (juvenile) 393 mg/l

Daphnia magna EC50 (96 h): >100 mg/l

Algae EC50 (18 d): *Chlorella vulgaris* 2700 mg/l (cell count)

The PNEC for the freshwater aquatic environment is based upon the lowest observed chronic toxicity result, the NOEC value of 11 mg/l ammonium sulfate for alevins of *Oncorhynchus gorboscha*. An assessment factor of 100 is appropriate, leading to a freshwater aquatic PNEC of 0.11 mg/l. Supporting information is also available for three juvenile amphibian species. The most sensitive amphibians were 6 week-old *Pseudacris regilla* tadpoles, with a NOEC (10 d) of 82 mg/l ammonium sulfate.

Marine Environment

Marine acute data are available for fish, invertebrates and for phytoplankton, the latter being most sensitive. For *Gymnodinium splendens* and *Gonyaulax polyedra*, growth reduction was found at concentrations of 0.7 mg/l and above. No EC50 can be derived. For seawater invertebrates the lowest effect value was obtained for green mussel *Perna viridis* (96h-LC50 = 47.7 mg/l). For marine fish the lowest effect value was found for larvae of *Sciaenops ocellatus* with a LC50 (10 d) of 27 mg/l.

Microorganisms in sewage treatment:

Nitrification during sewage treatment plant operation involves both sensitive (no growth at 4700 but growth at 94 mg/l ammonium sulfate) and insensitive (growth at 4700 mg/l ammonium sulfate) strains of *Nitrobacter* spp. These results indicate that a NOEC for specific nitrifying bacteria will be greater than 94 mg/l.

In the terrestrial environment, the major effect of repeated ammonium sulfate application is a reduction in soil pH. The most toxic results for specific soil bacteria, for cyanobacteria in rice fields, show less than 50% reduction in nitrogen fixation at 330 kg/ha/yr in the absence of liming. Similar results are seen for plants, with 471 kg/ha/y for 6 years affecting drought resistance in *Picea abies*. The soil fauna is less sensitive, with both *Collembolla* and *Cryptostigmata* numbers increasing under 708 kg /ha/year ammonium sulfate application

For ammonium nitrate

Fertility of *Daphnia magna* decreased at 50 g/l.

Post embryonic growth of crustacea was impaired at >10mg/l.

(*Aspergillus niger*) 40 hr. LC50: 15mg/l (36 C)

[CROPCARE]

Ammonium nitrate is soluble in water and acts as a source of nutrition for many microorganisms. Spills may produce massive eutrophication in static waters effecting the local aquatic community.

Ammonium nitrate penetrates soil at a rate which depends on soil type and water content. It is not expected to accumulate in the food chain.

for inorganic sulfates:

Environmental fate:

Data from tap water studies with human volunteers indicate that sulfates produce a laxative effect at concentrations of 1000 - 1200 mg/litre, but no increase in diarrhoea, dehydration or weight loss. The presence of sulfate in drinking-water can also result in a noticeable taste; the lowest taste threshold concentration for sulfate is approximately 250 mg/litre as the sodium salt. Sulfate may also contribute to the corrosion of distribution systems. No health-based guideline value for sulfate in drinking water is proposed. However, there is an increasing likelihood of complaints arising from a noticeable taste as concentrations in water increase above 500 mg/litre.

Sulfates are removed from the air by both dry and wet deposition processes. Wet deposition processes including rain-out (a process that occurs within the clouds) and washout (removal by precipitation below the clouds) contribute to the removal of sulfate from the atmosphere.

In soil, the inorganic sulfates can adsorb to soil particles or leach into surface water and groundwater. Sulfates can be taken up by plants and be incorporated into the parenchyma of the plant.

Sulfate in water can also be reduced by sulfate bacteria (*Thiobacilli*) which use them as a source of energy.

In anaerobic environments sulfate is biologically reduced to (hydrogen) sulfide by sulfate reducing bacteria, or incorporated into living organisms as source of sulfur, and thereby included in the sulfur cycle. Sodium sulfate is not reactive in aqueous solution at room temperature. Sodium sulfate will completely dissolve, ionise and distribute across the entire planetary "aquasphere". Some sulfates may eventually be deposited, the majority of sulfates participate in the sulfur cycle in which natural and industrial sodium sulfate are not

Continued...

distinguishable

The BCF of sodium sulfate is very low and therefore significant bioconcentration is not expected. Sodium and sulfate ions are essential to all living organisms and their intracellular and extracellular concentrations are actively regulated. However some plants (e.g. corn and *Kochia Scoparia*), are capable of accumulating sulfate to concentrations that are potentially toxic to ruminants.

Ecotoxicity:

For sulfate in general:

Fish LC50: toxic from 7000 mg/l

Bacteria: toxic from 2500 mg/l

Algae were shown to be the most sensitive to sodium sulfate; EC50 120 h = 1,900 mg/l. For invertebrates (*Daphnia magna*) the EC50 48 h = 4,580 mg/l and fish appeared to be the least sensitive with a LC50 96h = 7,960 mg/l for *Pimephales promelas*. Activated sludge showed a very low sensitivity to sodium sulfate. There was no effect up to 8 g/l. Sodium sulfate is not very toxic to terrestrial plants. *Picea banksiana* was the most sensitive species, an effect was seen at 1.4 g/l. Sediment dwelling organisms were not very sensitive either, with an LC50 96h = 660 mg/l for *Trycorythus sp.* Overall it can be concluded that sodium sulfate has no acute adverse effect on aquatic and sediment dwelling organisms. Toxicity to terrestrial plants is also low.

No data were found for long term toxicity. The acute studies all show a toxicity of sodium sulfate higher than 100 mg/l, no bioaccumulation is expected.

Although inorganic chloride ions are not normally considered toxic they can exist in effluents at acutely toxic levels (chloride >3000 mg/l). The resulting salinity can exceed the tolerances of most freshwater organisms.

Inorganic chlorine eventually finds its way into the aqueous compartment and as such is bioavailable. Incidental exposure to inorganic chloride may occur in occupational settings where chemicals management policies are improperly applied. The toxicity of chloride salts depends on the counter-ion (cation) present; that of chloride itself is unknown. Chloride toxicity has not been observed in humans except in the special case of impaired sodium chloride metabolism, e.g. in congestive heart failure. Healthy individuals can tolerate the intake of large quantities of chloride provided that there is a concomitant intake of fresh water.

Although excessive intake of drinking-water containing sodium chloride at concentrations above 2.5 g/litre has been reported to produce hypertension, this effect is believed to be related to the sodium ion concentration.

Chloride concentrations in excess of about 250 mg/litre can give rise to detectable taste in water, but the threshold depends upon the associated cations. Consumers can, however, become accustomed to concentrations in excess of 250 mg/litre. No health-based guideline value is proposed for chloride in drinking-water.

In humans, 88% of chloride is extracellular and contributes to the osmotic activity of body fluids. The electrolyte balance in the body is maintained by adjusting total dietary intake and by excretion via the kidneys and gastrointestinal tract. Chloride is almost completely absorbed in normal individuals, mostly from the proximal half of the small intestine. Normal fluid loss amounts to about 1.5-2 liters/day, together with about 4 g of chloride per day. Most (90 - 95%) is excreted in the urine, with minor amounts in faeces (4-8%) and sweat (2%).

Chloride increases the electrical conductivity of water and thus increases its corrosivity. In metal pipes, chloride reacts with metal ions to form soluble salts thus increasing levels of metals in drinking-water. In lead pipes, a protective oxide layer is built up, but chloride enhances galvanic corrosion. It can also increase the rate of pitting corrosion of metal pipes.

The nitrates are of environmental concern because of their high water solubility and consequent leaching, diffusion, and environmental mobility in soil and water. Nitrate can contaminate groundwater to unacceptable levels. Nitrite is formed from nitrate or ammonium ion by micro-organisms in soil, water, sewage and the alimentary tract. The concern with nitrate in the environment is related to its conversion to nitrite.

Methaemoglobinaemia is caused following exposure to high levels of nitrite and produces difficulties in oxygen transport in the blood. Thousands of cases involving poisoning of infants, particularly in rural areas, have been reported as a result of drinking nitrate rich well-water.

Other concerns deriving from exposure to environmental nitrates relate to the production of nitrosamines following the reaction of food nitrites and secondary amines. Other nitroso-compounds may result following reaction with nitrites and amides, ureas, carbamates and other nitrogenous compounds. Nitrosamines produce liver damage, haemorrhagic lung lesions, convulsions and coma in rats, and teratogenic effects in experimental animals.

The N-nitroso class of compounds include potent carcinogens and mutagens: induction of tumors by single doses of N-nitroso compounds testify to this.

In air ammonia is persistent whilst, in water, it biodegrades rapidly to nitrate, producing a high oxygen demand. Ammonia is strongly adsorbed to soil. Ammonia is non-persistent in water (half-life 2 days) and is moderately toxic to fish under normal temperature and pH conditions. Ammonia is harmful to aquatic life at low concentrations but does not concentrate in the food chain. Ammonium ions may be toxic to fish at 0.3 mg/l

Drinking Water Standards:

0.5 mg/l (UK max.)

1.5 mg/l (WHO Levels)

Soil Guidelines: none available.

Air Quality Standards: none available.

The principal problems of phosphate contamination of the environment relates to eutrophication processes in lakes and ponds. Phosphorus is an essential plant nutrient and is usually the limiting nutrient for blue-green algae. A lake undergoing eutrophication shows a rapid growth of algae in surface waters. Planktonic algae cause turbidity and flotation films. Shore algae cause ugly muddying, films and damage to reeds. Decay of these algae causes oxygen depletion in the deep water and shallow water near the shore. The process is self-perpetuating because anoxic conditions at the sediment/water interface causes the release of more adsorbed phosphates from the sediment. The growth of algae produces undesirable effects on the treatment of water for drinking purposes, on fisheries, and on the use of lakes for recreational purposes.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ammonium phosphate, monobasic	HIGH	HIGH
potassium chloride	HIGH	HIGH
ammonium sulfate	HIGH	HIGH
calcium sulfate	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
ammonium phosphate, monobasic	LOW (LogKOW = -0.7699)
potassium chloride	LOW (LogKOW = -0.4608)
ammonium sulfate	LOW (LogKOW = -2.2002)
calcium sulfate	LOW (LogKOW = -2.2002)

Mobility in soil

Ingredient	Mobility
ammonium phosphate, monobasic	HIGH (KOC = 1)
potassium chloride	LOW (KOC = 14.3)
ammonium sulfate	LOW (KOC = 6.124)
calcium sulfate	LOW (KOC = 6.124)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. In most instances the supplier of the material should be consulted.</p> <ul style="list-style-type: none"> ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority. ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Management Authority for disposal. ▶ Bury residue in an authorised landfill. ▶ Recycle containers if possible, or dispose of in an authorised landfill.
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Ensure that the hazardous substance is disposed in accordance with the Hazardous Substances (Disposal) Notice 2017

Disposal Requirements

Packages that have been in direct contact with the hazardous substance must be only disposed if the hazardous substance was appropriately removed and cleaned out from the package. The package must be disposed according to the manufacturer's directions taking into account the material it is made of. Packages which hazardous content have been appropriately treated and removed may be recycled.

The hazardous substance must only be disposed if it has been treated by a method that changed the characteristics or composition of the substance and it is no longer hazardous.

Only dispose to the environment if a tolerable exposure limit has been set for the substance.

Only deposit the hazardous substance into or onto a landfill or sewage facility or incinerator, where the hazardous substance can be handled and treated appropriately.

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (UN): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

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
ammonium phosphate, monobasic	Not Available
potassium chloride	Not Available
ammonium nitrate	Not Available
diammonium phosphate	Not Available
ammonium sulfate	Not Available
calcium phosphate, dibasic	Not Available
potassium phosphate, monobasic	Not Available
calcium sulfate	Not Available
ammonium chloride	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
ammonium phosphate, monobasic	Not Available
potassium chloride	Not Available
ammonium nitrate	Not Available
diammonium phosphate	Not Available
ammonium sulfate	Not Available
calcium phosphate, dibasic	Not Available
potassium phosphate, monobasic	Not Available
calcium sulfate	Not Available

Product name	Ship Type
ammonium chloride	Not Available

SECTION 15 Regulatory information

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

This substance is to be managed using the conditions specified in an applicable Group Standard

HSR Number	Group Standard
HSR002571	Fertilisers Subsidiary Hazard Group Standard 2020

Please refer to Section 8 of the SDS for any applicable tolerable exposure limit or Section 12 for environmental exposure limit.

ammonium phosphate, monobasic is found on the following regulatory lists

New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)

potassium chloride is found on the following regulatory lists

New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)

ammonium nitrate is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2A: Probably carcinogenic to humans
New Zealand Approved Hazardous Substances with controls

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)

diammonium phosphate is found on the following regulatory lists

New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)

ammonium sulfate is found on the following regulatory lists

FEI Equine Prohibited Substances List - Banned Substances
FEI Equine Prohibited Substances List (EPLS)
New Zealand Approved Hazardous Substances with controls

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)

calcium phosphate, dibasic is found on the following regulatory lists

New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)

potassium phosphate, monobasic is found on the following regulatory lists

New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)

calcium sulfate is found on the following regulatory lists

New Zealand Inventory of Chemicals (NZIoC)

New Zealand Workplace Exposure Standards (WES)

ammonium chloride is found on the following regulatory lists

FEI Equine Prohibited Substances List - Banned Substances
FEI Equine Prohibited Substances List (EPLS)
New Zealand Approved Hazardous Substances with controls
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
New Zealand Inventory of Chemicals (NZIoC)
New Zealand Workplace Exposure Standards (WES)

Hazardous Substance Location

Subject to the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Quantities
Not Applicable	Not Applicable

Certified Handler

Subject to Part 4 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Class of substance	Quantities
Not Applicable	Not Applicable

Refer Group Standards for further information

Maximum quantities of certain hazardous substances permitted on passenger service vehicles

Subject to Regulation 13.14 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Gas (aggregate water capacity in mL)	Liquid (L)	Solid (kg)	Maximum quantity per package for each classification
Not Applicable	Not Applicable	Not Applicable	Not Applicable	Not Applicable

Tracking Requirements

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (ammonium phosphate, monobasic; potassium chloride; ammonium sulfate; calcium phosphate, dibasic; potassium phosphate, monobasic; calcium sulfate; ammonium chloride)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	01/11/2019
Initial Date	12/05/2015

SDS Version Summary

Version	Date of Update	Sections Updated
3.1.1.1	06/07/2018	Appearance, Use
5.1.1.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification
5.1.2.1	29/04/2021	Regulation Change
5.1.2.2	30/05/2021	Template Change
5.1.2.3	04/06/2021	Template Change
5.1.2.4	05/06/2021	Template Change
5.1.2.5	09/06/2021	Template Change
5.1.2.6	11/06/2021	Template Change
5.1.3.6	14/06/2021	Regulation Change
5.1.3.7	15/06/2021	Template Change
5.1.3.8	05/07/2021	Template Change
5.1.4.8	14/07/2021	Regulation Change
5.1.4.9	01/08/2021	Template Change
5.1.5.9	02/08/2021	Regulation Change
5.1.6.9	05/08/2021	Regulation Change
5.1.7.9	09/08/2021	Regulation Change
5.1.7.10	29/08/2021	Template Change

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

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