

Optifert Finisher 15:0:17

Titan Ag

Chemwatch: 9642-93

Version No: 2.1

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

Chemwatch Hazard Alert Code: 3

Initial Date: 12/08/2026

Revision Date: 12/08/2026

Print Date: 14/08/2026

L.GHS.AUS.EN.E

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

Product Identifier

Product name	Optifert Finisher 15:0:17
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains copper sulfate, pentahydrate and zinc sulfate)
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. Use according to manufacturer's directions.
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Details of the manufacturer or importer of the safety data sheet

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

Emergency telephone number

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

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification ^[1]	Oxidizing Solids Category 3, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Germ Cell Mutagenicity Category 2, Reproductive Toxicity Category 1B, Hazardous to the Aquatic Environment Acute Hazard Category 3, Hazardous to the Aquatic Environment Long-Term Hazard Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	    
Signal word	Danger

Hazard statement(s)

H272	May intensify fire; oxidiser.
H302	Harmful if swallowed.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H318	Causes serious eye damage.
H341	Suspected of causing genetic defects.
H360FD	May damage fertility. May damage the unborn child.
H402	Harmful to aquatic life.
H410	Very toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P202	Do not handle until all safety precautions have been read and understood.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P220	Keep away from clothing and other combustible materials.
P261	Avoid breathing dust/fumes.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P370+P378	In case of fire: Use alcohol resistant foam or fine spray/water fog to extinguish.
P302+P352	IF ON SKIN: Wash with plenty of water.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P330	Rinse mouth.

Precautionary statement(s) Storage

P405	Store locked up.
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Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
6484-52-2	10-70	ammonium nitrate
7757-79-1	1-30	potassium nitrate
7778-18-9	1-30	calcium sulfate
1309-48-4.	<10	magnesium oxide
7446-20-0	<10	zinc sulfate
7758-99-8	<10	copper sulfate, pentahydrate
7782-63-0	<10	ferrous sulfate heptahydrate
12179-04-3	<10	sodium borate, pentahydrate
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: - Immediately hold eyelids apart and flush the eye continuously with running water. - Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.
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	▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs: ▶ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▶ Quickly remove all contaminated clothing, including footwear. ▶ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▶ Transport to hospital, or doctor.
Inhalation	▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay.
Ingestion	▶ For advice, contact a Poisons Information Centre or a doctor at once. ▶ Urgent hospital treatment is likely to be needed. ▶ 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. ▶ Transport to hospital or doctor without delay.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

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

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

Special hazards arising from the substrate or mixture

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

Fire Fighting	▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	▶ Combustible solid which burns but propagates flame with difficulty; it is estimated that most organic dusts are combustible (circa 70%) - according to the circumstances under which the combustion process occurs, such materials may cause fires and / or dust explosions. ▶ Organic powders when finely divided over a range of concentrations regardless of particulate size or shape and suspended in air or some other oxidizing medium may form explosive dust-air mixtures and result in a fire or dust explosion (including secondary explosions). ▶ Avoid generating dust, particularly clouds of dust in a confined or unventilated space as dusts may form an explosive mixture with air, and any source of ignition, i.e. flame or spark, will cause fire or explosion. Dust clouds generated by the fine grinding of the solid are a particular hazard; accumulations of fine dust (420 micron or less) may burn rapidly and fiercely if ignited - particles exceeding this limit will generally not form flammable dust clouds; once initiated, however, larger particles up to 1400 microns diameter will contribute to the propagation of an explosion. ▶ In the same way as gases and vapours, dusts in the form of a cloud are only ignitable over a range of concentrations; in principle, the concepts of lower explosive limit (LEL) and upper explosive limit (UEL) are applicable to dust clouds but only the LEL is of practical use;

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	- this is because of the inherent difficulty of achieving homogeneous dust clouds at high temperatures (for dusts the LEL is often called the "Minimum Explosible Concentration", MEC). ▶ When processed with flammable liquids/vapors/mists, ignitable (hybrid) mixtures may be formed with combustible dusts. Ignitable mixtures will increase the rate of explosion pressure rise and the Minimum Ignition Energy (the minimum amount of energy required to ignite dust clouds - MIE) will be lower than the pure dust in air mixture. The Lower Explosive Limit (LEL) of the vapour/dust mixture will be lower than the individual LELs for the vapors/mists or dusts. ▶ A dust explosion may release of large quantities of gaseous products; this in turn creates a subsequent pressure rise of explosive force capable of damaging plant and buildings and injuring people. ▶ Usually the initial or primary explosion takes place in a confined space such as plant or machinery, and can be of sufficient force to damage or rupture the plant. If the shock wave from the primary explosion enters the surrounding area, it will disturb any settled dust layers, forming a second dust cloud, and often initiate a much larger secondary explosion. All large scale explosions have resulted from chain reactions of this type. ▶ Dry dust can be charged electrostatically by turbulence, pneumatic transport, pouring, in exhaust ducts and during transport. ▶ Build-up of electrostatic charge may be prevented by bonding and grounding. ▶ Powder handling equipment such as dust collectors, dryers and mills may require additional protection measures such as explosion venting. ▶ All movable parts coming in contact with this material should have a speed of less than 1-meter/sec. ▶ A sudden release of statically charged materials from storage or process equipment, particularly at elevated temperatures and/or pressure, may result in ignition especially in the absence of an apparent ignition source. ▶ One important effect of the particulate nature of powders is that the surface area and surface structure (and often moisture content) can vary widely from sample to sample, depending of how the powder was manufactured and handled; this means that it is virtually impossible to use flammability data published in the literature for dusts (in contrast to that published for gases and vapours). ▶ Autoignition temperatures are often quoted for dust clouds (minimum ignition temperature (MIT)) and dust layers (layer ignition temperature (LIT)); LIT generally falls as the thickness of the layer increases. Combustion products include: ▶ carbon monoxide (CO) ▶ carbon dioxide (CO₂) nitrogen oxides (NO_x) sulfur oxides (SO_x) metal oxides ▶ other pyrolysis products typical of burning organic material.
HAZCHEM	2Z

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

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

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▶ Avoid skin contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ DO NOT allow material to come in direct contact with human skin or eyes. ▶ DO NOT allow material to come in contact with exposed food or food contact surfaces. ▶ Suitable PPE must be worn at all times. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained. ▶ Organic powders when finely divided over a range of concentrations regardless of particulate size or shape and suspended in air or some other oxidizing medium may form explosive dust-air mixtures and result in a fire or dust explosion (including secondary explosions) ▶ Minimise airborne dust and eliminate all ignition sources. Keep away from heat, hot surfaces, sparks, and flame. ▶ Establish good housekeeping practices.
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	- Remove dust accumulations on a regular basis by vacuuming or gentle sweeping to avoid creating dust clouds. - Use continuous suction at points of dust generation to capture and minimise the accumulation of dusts. Particular attention should be given to overhead and hidden horizontal surfaces to minimise the probability of a "secondary" explosion. According to NFPA Standard 654, dust layers 1/32 in. (0.8 mm) thick can be sufficient to warrant immediate cleaning of the area. - Do not use air hoses for cleaning. - Minimise dry sweeping to avoid generation of dust clouds. Vacuum dust-accumulating surfaces and remove to a chemical disposal area. Vacuums with explosion-proof motors should be used. - Control sources of static electricity. Dusts or their packages may accumulate static charges, and static discharge can be a source of ignition. - Solids handling systems must be designed in accordance with applicable standards (e.g. NFPA including 654 and 77) and other national guidance. - Do not empty directly into flammable solvents or in the presence of flammable vapors. - The operator, the packaging container and all equipment must be grounded with electrical bonding and grounding systems. Plastic bags and plastics cannot be grounded, and antistatic bags do not completely protect against development of static charges. Empty containers may contain residual dust which has the potential to accumulate following settling. Such dusts may explode in the presence of an appropriate ignition source. Do NOT cut, drill, grind or weld such containers. - In addition ensure such activity is not performed near full, partially empty or empty containers without appropriate workplace safety authorisation or permit.
Other information	- Store in original containers. - Keep containers securely sealed. - Store in a cool, dry area protected from environmental extremes. - Store away from incompatible materials and foodstuff containers. - Protect containers against physical damage and check regularly for leaks. - Observe manufacturer's storage and handling recommendations contained within this SDS. For major quantities: - Consider storage in bunded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams). - Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities.

Conditions for safe storage, including any incompatibilities

Suitable container	- Glass container is suitable for laboratory quantities - Polyethylene or polypropylene container. - Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Calcium sulfate: - reacts violently with reducing agents, acrolein, alcohols, chlorine trifluoride, diazomethane, ethers, fluorine, hydrazine, hydrazinium perchlorate, hydrogen peroxide, finely divided aluminium or magnesium, peroxyfuroic acid, red phosphorus, sodium acetylide - sensitises most organic azides which are unstable shock- and heat- sensitive explosives - may form explosive materials with 1,3-di(5-tetrazolyl)triazene - is incompatible with glycidol, isopropyl chlorocarbonate, nitrosyl perchlorate, sodium borohydride - is hygroscopic; reacts with water to form gypsum and Plaster of Paris Ammonium nitrate: - 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 NOTE:- Explosive detonations can occur when material is mixed with organic material, oils or charcoals and when heated or subjected to shock. The substance may be or contains a "metalloid" The following elements are considered to be metalloids; boron, silicon, germanium, arsenic, antimony, tellurium and (possibly) polonium. The electronegativities and ionisation energies of the metalloids are between those of the metals and nonmetals, so the metalloids exhibit characteristics of both classes. The reactivity of the metalloids depends on the element with which they are reacting. For example, boron acts as a nonmetal when reacting with sodium yet as a metal when reacting with fluorine. Unlike most metals, most metalloids are amphoteric- that is they can act as both an acid and a base. For instance, arsenic forms not only salts such as arsenic halides, by the reaction with certain strong acid, but it also forms arsenites by reactions with strong bases. Most metalloids have a multiplicity of oxidation states or valences. For instance, tellurium has the oxidation states +2, -2, +4, and +6. Metalloids react like non-metals when they react with metals and act like metals when they react with non-metals. Metal nitrites: - are incompatible with chlorates, hypophosphites, iodides, mercury salts, permanganates, sulfites, primary amines and amides, secondary amines and amides, ammonium salts, activated carbon, cyanogen compounds, thiocyanates, thiosulfates, cyanides, sodium amide, boron, acetanilide, antipyrine, tannic acid and cellulose - react explosively with hydrazine and liquid ammonia. - react explosively following fusion with metal cyanides - react (often) with salts of nitrogenous bases to produce an unstable corresponding nitrite salt. - Inorganic peroxy compounds are potent oxidisers that pose fire or explosive hazards when in contact with ordinary combustible materials. - Inorganic peroxides react with organic compounds to generate organic peroxide and hydroperoxide products that react violently with reducing agents. - Inorganic oxidising agents can react with reducing agents to generate heat and products that may be gaseous (causing pressurization of closed containers). The products may themselves be capable of further reactions (such as combustion in the air). - Organic compounds in general have some reducing power and can in principle react with compounds in this class. Actual reactivity varies greatly with the identity of the organic compound. - Inorganic oxidising agents can react violently with active metals, cyanides, esters, and thiocyanates. - Peroxides, in contact with inorganic cobalt and copper compounds, iron and iron compounds, acetone, metal oxide salts and acids and bases can react with rapid, uncontrolled decomposition, leading to fires and explosions. - Inorganic reducing agents react with oxidizing agents to generate heat and products that may be flammable, combustible, or otherwise reactive. Their reactions with oxidizing agents may be violent. - Incidents involving interaction of active oxidants and reducing agents, either by design or accident, are usually very energetic and examples of so-called redox reactions. - WARNING: Avoid or control reaction with peroxides. All transition metal peroxides should be considered as potentially explosive. For example transition metal complexes of alkyl hydroperoxides may decompose explosively. - The pi-complexes formed between chromium(0), vanadium(0) and other transition metals (haloarene-metal complexes) and mono- or poly-fluorobenzene show extreme sensitivity to heat and are explosive. - Avoid reaction with borohydrides or cyanoborohydrides

- ▶ Nitroaromatic and in particular polynitroaromatic compounds may present a severe explosion risk if subjected to shock or heated rapidly and uncontrollably as in fire situations.
 - ▶ In addition, when such compounds are heated more moderately with caustic alkalies, even when water or organic solvents are present, there is also a risk of violent decomposition or explosion - several industrial accidents, which probably were due to such interactions, have occurred; this potential hazard often remains unacknowledged.
 - ▶ Aromatic nitro compounds range from slight to strong oxidizing agents. If mixed with reducing agents, including hydrides, sulfides and nitrides, they may begin a vigorous reaction that culminates in a detonation. The explosive tendencies of aromatic nitro compounds are increased by the presence of multiple nitro groups.
 - ▶ A range of exothermic decomposition energies for nitro compounds is given as 220-410 kJ/mol. The relationship between energy of decomposition and processing hazards has been the subject of discussion; it is suggested that values of energy released per unit of mass, rather than on a molar basis (J/g) be used in the assessment. For example, in "open vessel processes" (with man-hole size openings, in an industrial setting), substances with exothermic decomposition energies below 500 J/g are unlikely to present a danger, whilst those in "closed vessel processes" (opening is a safety valve or bursting disk) present some danger where the decomposition energy exceeds 150 J/g.
- BREThERICK: Handbook of Reactive Chemical Hazards, 4th Edition
- for metal nitrates:
- ▶ Segregate from heavy metals, phosphides, sodium acetate, lead nitrate, tartrates, trichloroethylene,
 - ▶ Avoid shock and heat.
 - ▶ Mixtures of metal nitrates with alkyl esters may explode due to the formation of unstable alkyl nitrates.
 - ▶ Mixtures of a nitrate with phosphorous, tin(II) chloride and other reducing agents may react explosively.
 - ▶ Mixtures containing nitrates and organic materials are potentially dangerous, especially if acidic materials or heavy metals are present.
 - ▶ Fibrous organic material, jute, wood and similar cellulosic material can become highly combustible by nitrate impregnation
 - ▶ Metal nitrates are incompatible with cyanides, thiocyanates, isothiocyanates and hypophosphites. Avoid reaction with the following which can cause an explosion: barium thiocyanate, boron phosphide, cyanides, sodium hypophosphite, sulfur and charcoal, powdered aluminium or aluminium oxide, sodium thiosulfate.
 - ▶ Mixtures of metal nitrates and phosphinates may explode on heating
 - ▶ A mixture of aluminium powder, water and metal nitrate may explode due to a self accelerating reaction.
 - ▶ Mixtures containing nitrates, nitrites and organic materials are potentially dangerous, especially in the presence of acidic materials and heavy metals
 - ▶ Avoid any contamination of this material as it is very reactive and any contamination is potentially hazardous
- Copper sulfate:
- ▶ reacts violently with strong bases, hydroxylamine.(with ignition), magnesium (producing hydrogen gas)
 - ▶ in contact with potassium chlorate is potentially explosive
 - ▶ solutions are acidic and can react with metals to evolve flammable hydrogen gas. - corrosive to some metals including steel.
 - ▶ is incompatible with sulfuric acid, caustics, ammonia, aliphatic amines, alkanolamines, amides, alkylene oxides, epichlorohydrin, organic anhydrides, isocyanates, vinyl acetate
 - ▶ dusts or mists may react with acetylene to form shock-sensitive copper acetylides
 - ▶ Avoid storage with reducing agents.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	calcium sulfate	Calcium sulphate	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits	calcium sulfate	Calcium sulfate (respirable)	1.5 mg/m3	Not Available	Not Available	Containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	magnesium oxide	Magnesium oxide (fume)	10 mg/m3	Not Available	Not Available	Not Available
Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits	magnesium oxide	Magnesium oxide (fume)	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	ferrous sulfate heptahydrate	Iron salts, soluble (as Fe)	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	sodium borate, pentahydrate	Borates, tetra, sodium salts (pentahydrate)	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	sodium borate, pentahydrate	Borates, tetra, sodium salts (anhydrous)	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	sodium borate, pentahydrate	Borates, tetra, sodium salts (decahydrate)	5 mg/m3	Not Available	Not Available	Not Available
Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits	sodium borate, pentahydrate	Borates, tetra, sodium salts, incl anhydrous, decahydrate, pentahydrate	0.75 mg/m3	Not Available	Not Available	Not Available

MATERIAL DATA

Airborne particulate or vapour must be kept to levels as low as is practicably achievable given access to modern engineering controls and monitoring hardware. Biologically active compounds may produce idiosyncratic effects which are entirely unpredictable on the basis of literature searches and prior clinical experience (both recent and past).

for calcium sulfate:

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

These exposure guidelines have been derived from a screening level of risk assessment and should not be construed as unequivocally safe limits. ORGS represent an 8-hour time-weighted average unless specified otherwise.

CR = Cancer Risk/10000; UF = Uncertainty factor:

TLV believed to be adequate to protect reproductive health:

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LOD: Limit of detection

Toxic endpoints have also been identified as:

D = Developmental; R = Reproductive; TC = Transplacental carcinogen

Jankovic J., Drake F.: A Screening Method for Occupational Reproductive

American Industrial Hygiene Association Journal 57: 641-649 (1996)

The recommended TLV is thought to reduce the likelihood of respiratory irritation and skin irritation from exposure to aerosols and mists of soluble iron salts.

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Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. - Local exhaust ventilation is required where solids are handled as powders or crystals; even when particulates are relatively large, a certain proportion will be powdered by mutual friction. - Exhaust ventilation should be designed to prevent accumulation and recirculation of particulates in the workplace. - If in spite of local exhaust an adverse concentration of the substance in air could occur, respiratory protection should be considered. Such protection might consist of: (a): particle dust respirators, if necessary, combined with an absorption cartridge; (b): filter respirators with absorption cartridge or canister of the right type; (c): fresh-air hoods or masks - Build-up of electrostatic charge on the dust particle, may be prevented by bonding and grounding. - Powder handling equipment such as dust collectors, dryers and mills may require additional protection measures such as explosion venting. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to efficiently remove the contaminant. <table border="1"> <thead> <tr> <th>Type of Contaminant:</th><th>Air Speed:</th></tr> </thead> <tbody> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td><td>1-2.5 m/s (200-500 ft/min)</td></tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td><td>2.5-10 m/s (500-2000 ft/min)</td></tr> </tbody> </table> Within each range the appropriate value depends on: <table border="1"> <thead> <tr> <th>Lower end of the range</th><th>Upper end of the range</th></tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td><td>1: Disturbing room air currents</td></tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only</td><td>2: Contaminants of high toxicity</td></tr> <tr> <td>3: Intermittent, low production.</td><td>3: High production, heavy use</td></tr> <tr> <td>4: Large hood or large air mass in motion</td><td>4: Small hood-local control only</td></tr> </tbody> </table> Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 4-10 m/s (800-2000 ft/min) for extraction of crusher dusts generated 2 metres distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.	Type of Contaminant:	Air Speed:	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 ft/min)	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 ft/min)	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use	4: Large hood or large air mass in motion	4: Small hood-local control only
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Individual protection measures, such as personal protective equipment																	
Eye and face protection	- Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure. - Chemical goggles. Whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted. [AS/NZS 1337.1, EN166 or national equivalent] - Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection. - Alternatively a gas mask may replace splash goggles and face shields. - Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].																
Skin protection	See Hand protection below																
Hands/feet protection	- Elbow length PVC gloves NOTE: - The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. - Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: - frequency and duration of contact,																

Continued...

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- chemical resistance of glove material,
- glove thickness and
- dexterity

Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).

- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.

· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.

- Contaminated gloves should be replaced.

As defined in ASTM F-739-96 in any application, gloves are rated as:

- Excellent when breakthrough time > 480 min
- Good when breakthrough time > 20 min
- Fair when breakthrough time < 20 min
- Poor when glove material degrades

For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.

It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.

Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.

Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:

- Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.

- Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential

Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

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

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

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

Body protection See Other protection below

Other protection

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

Respiratory protection

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

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

* - Negative pressure demand ** - Continuous flow

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

- Respirators may be necessary when engineering and administrative controls do not adequately prevent exposures.

· The decision to use respiratory protection should be based on professional judgment that takes into account toxicity information, exposure measurement data, and frequency and likelihood of the worker's exposure - ensure users are not subject to high thermal loads which may result in heat stress or distress due to personal protective equipment (powered, positive flow, full face apparatus may be an option).

- Published occupational exposure limits, where they exist, will assist in determining the adequacy of the selected respiratory protection. These may be government mandated or vendor recommended.

· Certified respirators will be useful for protecting workers from inhalation of particulates when properly selected and fit tested as part of a complete respiratory protection program.

· Where protection from nuisance levels of dusts are desired, use type N95 (US) or type P1 (EN143) dust masks. Use respirators and components tested and approved under appropriate government standards such as NIOSH (US) or CEN (EU)

- Use approved positive flow mask if significant quantities of dust becomes airborne.

· Try to avoid creating dust conditions.

Class P2 particulate filters are used for protection against mechanically and thermally generated particulates or both.

P2 is a respiratory filter rating under various international standards, Filters at least 94% of airborne particles

Suitable for:

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Pellets.		
Physical state	Divided Solid	Relative density (Water = 1)	0.85-0.95

Continued...

Odour	Slight	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	6	Decomposition temperature (°C)	>130
Melting point / freezing point (°C)	Not Available	Viscosity (mm²/s)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Available	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Applicable
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Heat of Combustion (kJ/g)	Not Available	Ignition Distance (cm)	Not Available
Flame Height (cm)	Not Available	Flame Duration (s)	Not Available
Enclosed Space Ignition Time Equivalent (s/m³)	Not Available	Enclosed Space Ignition Deflagration Density (g/m³)	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

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

Inhaled	Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Inhalation of dusts, generated by the material during the course of normal handling, may be damaging to the health of the individual. Persons with impaired respiratory function, airway diseases and conditions such as emphysema or chronic bronchitis, may incur further disability if excessive concentrations of particulate are inhaled. If prior damage to the circulatory or nervous systems has occurred or if kidney damage has been sustained, proper screenings should be conducted on individuals who may be exposed to further risk if handling and use of the material result in excessive exposures. Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. The material can produce severe chemical burns within the oral cavity and gastrointestinal tract following ingestion. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing.

Magnesium salts are generally absorbed so slowly that oral administration causes few toxic effects with purging being the most significant. If evacuation fails due to bowel obstruction or atony, mucosal irritation and absorption may result.

Side effects of magnesium salts include upset stomach, dry mouth, dry nose and dry throat, drowsiness, nausea, heartburn and thickening of the mucous in the throat and nose.

Systemically the magnesium ion produces electrolyte imbalance, central nervous system depression, neurological and cardiac involvement, reflex abolition and death from respiratory paralysis. These effects are rare in the absence of intestinal or renal disorders.

Early signs and symptoms of magnesium intoxication include nausea, vomiting, malaise and confusion. Deep tendon reflexes may be diminished. central nervous system depression may progress to coma and paralysis of the release of acetylcholine at myoneuronal junctions. Central nervous system depression may be compounded by depressed function of the respiratory musculature. Hypotension may also ensue as a result of

peripheral vasodilation and/ or decreased cardiac output secondary to conduction defects. Bradycardia is common, leading to eventual arrest in diastole.

The substance and/or its metabolites may bind to haemoglobin inhibiting normal uptake of oxygen. This condition, known as "methaemoglobinemia", is a form of oxygen starvation (anoxia).

Symptoms include cyanosis (a bluish discolouration skin and mucous membranes) and breathing difficulties. Symptoms may not be evident until several hours after exposure.

At about 15% concentration of blood methaemoglobin there is observable cyanosis of the lips, nose and earlobes. Symptoms may be absent although euphoria, flushed face and headache are commonly experienced. At 25-40%, cyanosis is marked but little disability occurs other than that produced on physical exertion. At 40-60%, symptoms include weakness, dizziness, lightheadedness, increasingly severe headache, ataxia, rapid shallow respiration, drowsiness, nausea, vomiting, confusion, lethargy and stupor. Above 60% symptoms include dyspnea, respiratory depression, tachycardia or bradycardia, and convulsions. Levels exceeding 70% may be fatal.

Symptoms of borate poisoning include nausea, vomiting, diarrhoea, epigastric pain. These may be accompanied headache, weakness and a distinctive red skin rash. In severe cases there may be shock, increased heart rate and the skin may appear blue. Vomiting (which may be violent) is often persistent and vomitus and faeces may contain blood. Weakness, lethargy, headache, restlessness, tremors and intermittent convulsions may also occur. Poisoning produces central nervous system stimulation followed by depression, gastrointestinal disturbance (haemorrhagic gastro-enteritis), erythematous skin eruptions (giving rise to a boiled lobster appearance) and may also involve kidneys (producing oliguria, albuminuria, anuria) and, rarely, liver (hepatomegaly, jaundice). Toxic symptoms may be delayed for several hours. Ingested borates are readily absorbed and do not appear to be metabolised via the liver. Excretion occurs mainly through the kidneys in the urine with about half excreted in the first 12 hours and the remainder over 5-12 days. Borates are excreted primarily in the urine regardless of the route of administration.

The borates (tetra-, di-, meta, or ortho- salts, in contrast to perborates) once solubilised in the acid of gastric juices, cannot be distinguished from each other on chemical or toxicological grounds. In humans acute gastroenteric (or percutaneous absorption of as little as 1 gm of sodium borate can result in severe gastrointestinal irritation, kidney damage. In adults the mean lethal dose of sodium borate or boric acid probably exceeds 30 gms (Gosselin) and death occurs due to vascular collapse in the early stages or to central nervous system depression in later stages.

Children are thought to be more susceptible to the effects of borate intoxication.

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, 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

Sulfate salts are poorly absorbed from the gastro-intestinal tract but because of osmotic activity are able to draw water from the lumen to produce diarrhoea (purging). Sulfate ion usually has little toxicological potential.

The lethal oral dose of nitrite for adults has been variously reported to be between 0.7 and 6 g NO₂⁻ (approximately 10 to 100 mg NO₂⁻/kg). Lower doses may apply for children (especially neonates), the elderly and people with certain enzyme deficiencies. The first symptoms of oral nitrite poisoning develop within 15 to 45 minutes

In humans, inorganic nitrites produce smooth muscle relaxation, methaemoglobinaemia and cyanosis. The primary effect of nitrite intoxication in animals is methaemoglobinaemia whilst secondary effects include vasodilation, relaxation of smooth muscle and lowering of blood pressure. Other nitrite-induced toxic effects include abdominal pain, diarrhoea, atrophied intestinal villi and apoptotic cell death in the intestinal crypts. Nitrite may also cause sudden fall in blood pressure due to its vasodilating properties. Nitrite has vasodilating properties, probably through transformation into nitric oxide (NO) or a NO-containing molecule acting as a signal factor for smooth muscle relaxation. Fatal poisonings in infants, resulting from ingestion of nitrates in water or spinach, have been reported.

When sodium nitrite was administered in drinking water for 6 weeks (0.06-1%), mice showed a slight degeneration and spotty necrosis of hepatocytes and haemosiderin deposition in the liver, spleen and lymph nodes, indicating haemolysis. At 2%, mice died within 3 weeks. In rats, subject to the same treatment regime, abnormal blood and spleen colours, due to MHG, were seen in 0.5% and 1.0% treatment groups. Hepatic microsomal lipoperoxidation (as measured by malondialdehyde formation) was increased in male rats given 0.2% sodium nitrite in drinking water. Liver lysosomal enzymes (acid phosphatase and cathepsin) and superoxide dismutase activities were also increased. This data suggests that the nitrite stimulates generation of superoxide radicals in the liver causing damage to cellular and subcellular membranes. Decreased plasma vitamin E and greater reduced glutathione-per erythrocyte were also reported in male rats receiving sodium nitrite in drinking water.

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. 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.

The material can produce severe chemical burns following direct contact with the skin.

Skin Contact

Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact.

Four students received severe hand burns whilst making moulds of their hands with dental plaster substituted for Plaster of Paris. The dental plaster known as "Stone" was a special form of calcium sulfate hemihydrate containing alpha-hemihydrate crystals that provide high compression strength to the moulds. Beta-hemihydrate (normal Plaster of Paris) does not cause skin burns in similar circumstances.

Open cuts, abraded or irritated skin should not be exposed to this material

Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.

Limited 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.

Eye

The material can produce severe chemical burns to the eye following direct contact. Vapours or mists may be extremely irritating.

When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.

Chronic

Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis.

Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure.

Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.

Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.

Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers. Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.

Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.

Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.

Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.

There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: - clear evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects.

There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of:

- clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.

Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.

On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

Chronic poisoning by borates may be characterised gastrointestinal disturbances and skin rash. Chronic absorption of small amounts of borax causes mild gastroenteritis and dermatitis.

Chronic feeding studies involving borate administration to rats and dogs leads to accumulation in the testes, germ cell depletion and testicular atrophy. Hair loss in a young woman was traced to chronic ingestion of boric acid-containing mouthwashes whilst hair loss, dermatitis, gastric ulcer and hypoplastic anaemia in an adult male was attributed to the consumption of an uncharacterised "boric tartrate" for 20 years (symptoms disappeared following withdrawal). Repeated ingestion or inhalation of sub-acute doses of boric acid produces gastrointestinal irritation and disturbance, loss of appetite, disturbed digestion, nausea and vomiting, erythematous rash which may become hard and purpuric, dryness of the skin and mucous membranes, reddening of the tongue, cracking of the lips, conjunctivitis, palpebral oedema and kidney injury. Workers exposed to dust levels containing in excess of 31 mg/m³ boric acid, showed atrophic and subatrophic changes of the respiratory mucous membranes. Prolonged ingestion by animals produces a variety of reproductive effects including changes to the ovaries, fallopian tubes, the testes, epididymis and sperm ducts.

Inorganic borates convert to boric acid at physiological pH in the aqueous layer overlying the mucosal surfaces prior to absorption. Boric acid is known to be readily taken up from the gastrointestinal tract in rats and humans, as demonstrated by experimental evidence in both human and animal studies, where more than 90% of the administered dose of borate was excreted as boric acid.

Boric acid is not metabolized in either animals or humans, owing to the high energy level required (523 kJ/mol) to break the B-O bond. Because of the high pKa, regardless of the form of inorganic borate ingested (e.g., boric acid, disodium tetraborate decahydrate or boron associated with animal or plant tissues), uptake is almost exclusively (>98%) as undissociated boric acid.

A case of chronic abuse of magnesium citrate (a mild purgative), by a 62 year-old woman, has been reported. Symptoms of abuse included lethargy and severe refractory hypotension. Pathology revealed extreme hypermagnesaemia [6.25 mmol per litre]. She also was found to have a perforated duodenal ulcer. She died after peritoneal dialysis (which reduced serum-magnesium and reduced hypotension).

A patient with normal kidney function developed symptomatic hypermagnesaemia with respiratory arrest and bradycardia after receiving 90 grams of magnesium sulfate over 18 hours.

When magnesium sulfate was given to pregnant rats, a sharp reduction of both the number and the weight of the offspring was observed.

Prolonged inhalation of high concentrations of magnesite (magnesium carbonate) dust caused pulmonary deposition and retention. Roasted magnesite (magnesium oxide) produced a greater degree of fibrosis than did crude magnesite. No cases of human systemic poisoning due to exposure to magnesite have been recorded. Pneumoconiosis was found in about 2% of workers exposed to high concentrations of dust from crude or roasted magnesite that also contained 1-3% silicon dioxide. Exposure periods ranged from 6-20 years. This condition occurred mainly in workers exposed to roasted (calcined) magnesite. The pneumoconiosis appeared to be "benign" and was often associated with chronic bronchitis and lung emphysema.

In other reports the severity of the pneumoconiosis was associated with the crystalline silica content of the dust or in a case of magnesium carbonate used in insulating materials, the severity of the disease depended on the asbestos content.

Complaints of coughing are rare amongst magnesite workers, and more frequent among dianase and grog (crushed refractory materials) workers.

Airborne dust concentrations were lowest in dianase facilities but crystalline silica was high. Chronic bronchitis then, appears to increase where concentrations of crystalline silica are highest.

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.

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.

Chronic excessive iron exposure has been associated with haemosiderosis and consequent possible damage to the liver and pancreas.

Haemosiderin is a golden-brown insoluble protein produced by phagocytic digestion of haematin (an iron-based pigment). Haemosiderin is found in most tissues, especially in the liver, in the form of granules. Other sites of haemosiderin deposition include the pancreas and skin. A related condition, haemochromatosis, which involves a disorder of metabolism of these deposits, may produce cirrhosis of the liver, diabetes, and bronze pigmentation of the skin - heart failure may eventually occur.

Such exposure may also produce conjunctivitis, choroiditis, retinitis (both inflammatory conditions involving the eye) and siderosis of tissues if iron remains in these tissues. Siderosis is a form of pneumoconiosis produced by iron dusts. Siderosis also includes discoloration of organs, excess circulating iron and degeneration of the retina, lens and uvea as a result of the deposition of intraocular iron. Siderosis might also involve the lungs - involvement rarely develops before ten years of regular exposure. Often there is an accompanying inflammatory reaction of the bronchi. Permanent scarring of the lungs does not normally occur.

High levels of iron may raise the risk of cancer. This concern stems from the theory that iron causes oxidative damage to tissues and organs by generating highly reactive chemicals, called free radicals, which subsequently react with DNA. Cells may be disrupted and may be

become cancerous. People whose genetic disposition prevents them from keeping tight control over iron (e.g. those with the inherited disorder, haemochromatosis) may be at increased risk.

Iron overload in men may lead to diabetes, arthritis, liver cancer, heart irregularities and problems with other organs as iron builds up. [K. Schmidt, New Scientist, No. 1919 pp.11-12, 2nd April, 1994]

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.

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

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

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.

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.

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Under certain conditions, nitrites can react with secondary amines, either alone or in biological systems, to form carcinogenic nitrosamines. 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.

Optifert Finisher 15:0:17	TOXICITY	IRRITATION
	Not Available	Not Available
ammonium nitrate	TOXICITY	IRRITATION
	dermal (rat) LD50: >5000 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
	Inhalation (Rat) LC50: >88.8 mg/l4h ^[2]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: 2217 mg/kg ^[2]	
potassium nitrate	TOXICITY	IRRITATION
	dermal (rat) LD50: >5000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (Rat) LC50: >0.527 mg/l4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Rabbit) LD50; 1901 mg/kg ^[2]	
calcium sulfate	TOXICITY	IRRITATION
	Inhalation (Rat) LC50: >3.26 mg/l4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >1581 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
magnesium oxide	TOXICITY	IRRITATION
	Not Available	Not Available
zinc sulfate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (Rodent - rabbit): 420ug - Moderate
	Oral (Mouse) LD50; 200 mg/kg ^[2]	Eye: adverse effect observed (irreversible damage) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
copper sulfate, pentahydrate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[2]	Skin (Human - woman): 2%
	Oral (Mouse) LD50; 43 mg/kg ^[2]	
ferrous sulfate heptahydrate	TOXICITY	IRRITATION
	Oral (Mouse) LD50; 1520 mg/kg ^[2]	Not Available
sodium borate, pentahydrate	TOXICITY	IRRITATION
	Oral (Rat) LD50: 2660 mg/kg ^[2]	Eye (Rodent - rabbit): 100mg - 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

Optifert Finisher 15:0:17	Laboratory (in vitro) and animal studies show, exposure to the material may result in a possible risk of irreversible effects, with the possibility of producing mutation.
ZINC SULFATE	Oral (human) TDLo: 45 mg/kg/7d-C Eye (rabbit): 0.42 mg moderate Oral (man) TDLo: 180 mg/kg/6w-I Equivocal tumorigenic agent by RTECS criteria. for zinc sulfate heptahydrate Sleep, ataxia, respiratory stimulation, somnolence, coma, diarrhoea, changes in endocrine pancreas recorded. Exposure may produce irreversible effects*. NOTE: Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.
COPPER SULFATE, PENTAHYDRATE	for copper and its compounds (typically copper chloride):

Optifert Finisher 15:0:17

	Acute toxicity: There are no reliable acute oral toxicity results available. In an acute dermal toxicity study (OECD TG 402), one group of 5 male rats and 5 groups of 5 female rats received doses of 1000, 1500 and 2000 mg/kg bw via dermal application for 24 hours. The LD50 values of copper monochloride were 2,000 mg/kg bw or greater for male (no deaths observed) and 1,224 mg/kg bw for female. Four females died at both 1500 and 2000 mg/kg bw, and one at 1,000 mg/kg bw. Symptom of the hardness of skin, an exudation of hardness site, the formation of scar and reddish changes were observed on application sites in all treated animals. Skin inflammation and injury were also noted. In addition, a reddish or black urine was observed in females at 2,000, 1,500 and 1,000 mg/kg bw. Female rats appeared to be more sensitive than male based on mortality and clinical signs. No reliable skin/eye irritation studies were available. The acute dermal study with copper monochloride suggests that it has a potential to cause skin irritation. Repeat dose toxicity: In repeated dose toxicity study performed according to OECD TG 422, copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39 - 51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL value was 5 and 1.3 mg/kg bw/day for male and female rats, respectively. No deaths were observed in male rats. One treatment-related death was observed in female rats in the high dose group. Erythropoietic toxicity (anaemia) was seen in both sexes at the 80 mg/kg bw/day. The frequency of squamous cell hyperplasia of the forestomach was increased in a dose-dependent manner in male and female rats at all treatment groups, and was statistically significant in males at doses of =20 mg/kg bw/day and in females at doses of =5 mg/kg bw/day doses. The observed effects are considered to be local, non-systemic effect on the forestomach which result from oral (gavage) administration of copper monochloride. Genotoxicity: An in vitro genotoxicity study with copper monochloride showed negative results in a bacterial reverse mutation test with Salmonella typhimurium strains (TA 98, TA 100, TA 1535, and TA 1537) with and without S9 mix at concentrations of up to 1,000 ug/plate. An in vitro test for chromosome aberration in Chinese hamster lung (CHL) cells showed that copper monochloride induced structural and numerical aberrations at the concentration of 50, 70 and 100 ug/mL without S9 mix. In the presence of the metabolic activation system, significant increases of structural aberrations were observed at 50 and 70 ug/mL and significant increases of numerical aberrations were observed at 70 ug/mL. In an in vivo mammalian erythrocyte micronucleus assay, all animals dosed (15 - 60 mg/kg bw) with copper monochloride exhibited similar PCE/(PCE+NCE) ratios and MNPCE frequencies compared to those of the negative control animals. Therefore copper monochloride is not an in vivo mutagen. Carcinogenicity: there was insufficient information to evaluate the carcinogenic activity of copper monochloride. Reproductive and developmental toxicity: In the combined repeated dose toxicity study with the reproduction/developmental toxicity screening test (OECD TG 422), copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39-51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL of copper monochloride for fertility toxicity was 80 mg/kg bw/day for the parental animals. No treatment-related effects were observed on the reproductive organs and the fertility parameters assessed. For developmental toxicity the NOAEL was 20 mg/kg bw/day. Three of 120 pups appeared to have icterus at birth; 4 of 120 pups appeared runted at the highest dose tested (80 mg/kg bw/day).
SODIUM BORATE, PENTAHYDRATE	for sodium borate, decahydrate. Reproductive effector in rats Mutagenic towards bacteria The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
Optifert Finisher 15:0:17 & MAGNESIUM OXIDE & COPPER SULFATE, PENTAHYDRATE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
Optifert Finisher 15:0:17 & COPPER SULFATE, PENTAHYDRATE	For copper sulfate ▶ Acute toxicity: Copper sulfate is corrosive and acute toxicity is largely due to this property. There have been reports of human suicide resulting from the ingestion of gram quantities of this material. The lowest dose of copper sulfate that has been toxic when ingested by humans is 11 mg/kg. Ingestion of copper sulfate is often not toxic because vomiting is automatically triggered by its irritating effect on the gastrointestinal tract. Symptoms are severe, however, if copper sulfate is retained in the stomach, as in the unconscious victim. Some of the signs of poisoning which occurred after 1 to 12 g of copper sulfate was swallowed include a metallic taste in the mouth, burning pain in the chest and abdomen, intense nausea, repeated vomiting, diarrhea, headache, sweating, shock, discontinued urination leading to yellowing of the skin. Injury to the brain, liver, kidneys, and stomach and intestinal linings may also occur in copper sulfate poisoning. Copper sulfate can be corrosive to the skin and eyes. It is readily absorbed through the skin and can produce a burning pain, as well as the other symptoms of poisoning resulting from ingestion. Skin contact may result in itching or eczema. It is a skin sensitizer and can cause allergic reactions in some individuals. Eye contact with this material can cause conjunctivitis, inflammation of the eyelid lining, cornea tissue deterioration, and clouding of the cornea. Examination of copper sulfate poisoned animals showed signs of acute toxicity in the spleen, liver, and kidneys. Injury may also occur to the brain, liver, kidneys, and gastrointestinal tract in response to overexposure to this material. ▶ Chronic toxicity: Vineyard sprayers experienced liver disease after 3 to 15 years of exposure to copper sulfate solution in Bordeaux mixture. Long term effects are more likely in individuals with Wilson's disease, a condition which causes excessive absorption and storage of copper. Chronic exposure to low levels of copper can lead to anemia. The growth of rats was retarded when given dietary doses of 25 mg/kg/day of copper sulfate. Dietary doses of 200 mg/kg/day caused starvation and death. Sheep given oral doses of 20 mg/kg/day showed blood cell and kidney damage. They also showed an absence of appetite, anemia, and degenerative changes. ▶ Reproductive effects: Copper sulfate has been shown to cause reproductive effects in test animals. Testicular atrophy increased in birds as they were fed larger amounts of copper sulfate. Sperm production was also interrupted to varying degrees. Reproduction and fertility was affected in pregnant rats given this material on day 3 of pregnancy. ▶ Teratogenic effects: There is very limited evidence about the teratogenic effects of copper sulfate. Heart disease occurred in the surviving offspring of pregnant hamsters given intravenous copper salts on day 8 of gestation. These data suggest that copper sulfate is unlikely to be teratogenic in humans at expected exposure levels. ▶ Mutagenic effects: Copper sulfate may cause mutagenic effects at high doses. At 400 and 1000 ppm, copper sulfate caused mutations in two types of microorganisms. Such effects are not expected in humans under normal conditions. ▶ Carcinogenic effects: Copper sulfate at 10 mg/kg/day caused endocrine tumors in chickens given the material parenterally, that is, outside of the gastrointestinal tract through an intravenous or intramuscular injection. However, the relevance of these results to mammals, including humans, is not known. ▶ Organ toxicity: Long-term animal studies indicate that the testes and endocrine glands have been affected. ▶ Fate in humans and animals: Absorption of copper sulfate into the blood occurs primarily under the acidic conditions of the stomach. The mucous membrane lining of the intestines acts as a barrier to absorption of ingested copper. After ingestion, more than 99% of copper is excreted in the faeces. However, residual copper is an essential trace element that is strongly bioaccumulated. It is stored primarily in the liver, brain, heart, kidney, and muscles diverse effects of NAADP modulators are not well-documented because they are still under investigation, but potential concerns include cardiac arrhythmias, pulmonary hypertension, and impact on T-cell differentiation and intestinal inflammation. Modulating the potent calcium-mobilizing molecule NAADP can have serious consequences, particularly in the cardiovascular system, and inhibiting it could suppress beneficial immune responses while promoting others. ased on the mechanism of NAADP, inhibiting it could lead to side effects related to calcium (signaling disruptions, such as impacts on muscle contraction, cellular processes like autophagy, and potentially cardiac function. he mechanism of action of NAADP appears to involve different intracellular Ca2+ stores, e.g. acidic stores), nuclear envelope (, endoplasmic reticulum, 6), or secretory vesicles). Similarly, different candidate Ca2+ channels have been proposed, e.g. members of the two-pore family), ryanodine receptors (RyRs), or transient receptor potential channels, subtype mucolipin 1 (TRP-ML1). A unifying hypothesis to integrate these different pathways of NAADP action was recently proposed (the central idea is that NAADP does not directly modulate channels but requires specific binding protein(s) to modulate different Ca2+ channels (

	Steroid Receptor Coactivator (SRC) modulators (like inhibitors or knockouts) affect hormone-driven processes, potentially causing reproductive issues (dwarfism, infertility), immune dysregulation (autoimmunity, T-cell changes), metabolic shifts, and influencing cancer growth (both promoting and inhibiting depending on the context). Side effects often relate to their fundamental role in regulating steroid hormones (estrogen, androgen, progesterone) in tissues like the brain, reproductive organs, and immune cells, leading to broad systemic impacts, though specific inhibitors (like SRC-3 inhibitors in Tregs) show promise with fewer general toxicities than broader therapies Key Adverse Effects & Considerations: Reproductive & Endocrine: Dysfunction: SRC knockout in mice causes reproductive failure, delayed puberty, and dwarfism. Behavioral: SRC-1 inhibition can alter sex behaviors and brain development in rodents. Immune System: Treg Modulation: Inhibiting SRCs (especially SRC-3) in Regulatory T cells (Tregs) can boost anti-cancer immunity but might also impact normal immune balance. Potential Autoimmunity: While SRC-3 KO Tregs seem benign, broader immune targeting can lead to severe autoimmunity, though SRC inhibitors aim to avoid this. Cancer: Dual Role: SRCs drive many cancers (breast, ovarian, prostate). Modulators can inhibit tumor growth, but some (like pan-SRC inhibitors) might impair anti-tumor immunity General Systemic Effects: Metabolic: SRC dysregulation is linked to Polycystic Ovary Syndrome (PCOS) and fibroids. Brain Function: SRCs are crucial for neuroendocrine regulation, affecting seasonal cycles and hormone responses in the brain Therapeutic Context Matters: Specific vs. Pan-Inhibitors: Targeting specific SRCs (like SRC-3 in Tregs) or using targeted inhibitors (like SI-2) aims for anti-cancer effects with fewer systemic side effects than broader immune checkpoint inhibitors, avoiding issues like cytokine release syndrome. Drug Development: Natural compounds (gossypol, bufalin) and synthetic molecules (SI-2) are being developed, showing potential for non-hormonal treatments in women's health, but clinical application needs careful study. In essence, SRC modulators are powerful tools affecting fundamental hormone signaling, meaning their "adverse effects" are often the flip side of their therapeutic action, necessitating precise targeting for safe use.
Optifert Finisher 15:0:17 & CALCIUM SULFATE	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. 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 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. 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. 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. 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. 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. 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. 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." 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. Inflammation (specifically, chronic alveolitis with macrophage and neutrophil aggregation) was observed in the lung. 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. 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 ug Hg/g dry weight. Total mercury loss from the original FGD gypsum content was about 0.045 g Hg/ton dry gypsum processed 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. Cytotoxicity: In Syrian hamster embryo cells, gypsum (up to 10 ug/cm2) did not induce apoptosis. Negative results were also found in mouse peritoneal macrophages (tested at 150 ug/mL gypsum dust) and in Chinese hamster lung V79-4 cells (tested up to 100 ug/mL). 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. 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. In guinea pigs, inhalation of gypsum (doses not provided) for 24 months produced no lung tumours. 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. 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 of 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. 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. 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 (embryo implantation); developmental effects were also not seen.
Optifert Finisher 15:0:17 & CALCIUM SULFATE & MAGNESIUM OXIDE & SODIUM BORATE, PENTAHYDRATE	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.

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					
Optifert Finisher 15:0:17	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
ammonium nitrate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	490mg/l	2
	NOEC(ECx)	480h	Fish	0.003mg/l	4
	LC50	96h	Fish	48.184-59.63mg/L	4
potassium nitrate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	490mg/l	2
	LC50	96h	Fish	>100mg/l	2
	NOEC(ECx)	144h	Fish	0.1mg/l	4
calcium sulfate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>79mg/l	2
	EC50	96h	Algae or other aquatic plants	3200mg/L	4
	LC50	96h	Fish	>79mg/l	2
	NOEC(ECx)	0.25h	Fish	75mg/l	4
magnesium oxide	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
zinc sulfate	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1344h	Fish	59-112	7
	EC50	72h	Algae or other aquatic plants	0.01-0.122mg/l	4
	EC50	48h	Crustacea	0.06mg/L	4
	EC50	96h	Algae or other aquatic plants	0.01mg/L	4
	LC50	96h	Fish	<0.001mg/L	4
	EC20(ECx)	72h	Algae or other aquatic plants	0.001-0.075mg/l	4
copper sulfate, pentahydrate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	0.8mg/L	5
	EC50	48h	Crustacea	0.003mg/L	5

	LC50	96h	Fish	0.073mg/L	4
	EC50(ECx)	96h	Crustacea	0.001mg/L	5
ferrous sulfate heptahydrate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	12.35-16.72mg/L	4
	LC50	96h	Fish	6.27-50.35mg/L	4
	EC50(ECx)	48h	Crustacea	12.35-16.72mg/L	4
sodium borate, pentahydrate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	1332-2135mg/l	4
	EC50(ECx)	48h	Crustacea	1332-2135mg/l	4
	EC50(ECx)	96h	Algae or other aquatic plants	2.6-21.8mg/l	4
	EC50	96h	Algae or other aquatic plants	2.6-21.8mg/l	4
	LC50	96h	Fish	1900mg/l	4
Legend:	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. US EPA, Ecotox database - Aquatic Toxicity Data 4. ECETOC Aquatic Hazard Assessment Data 5. NITE (Japan) - Bioconcentration Data 6. METI (Japan) - Bioconcentration Data 7. Vendor Data				

Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

For copper sulfate:

Cupric Sulfate is a Severe Marine Pollutant (49 CFR 172.322) and requires the marine pollutant mark for vessel transportation. Because Copper Sulfate is listed as a Severe Marine Pollutant as found in Appendix B to 172.101 and when shipped by vessel, each inner package which exceeds 500 g (17.6 oz) will need a marine pollutant marking, UN certified package, marked with the Proper Shipping Name, UN Number will be required when shipped by vessel, when each inner package exceeds 500 g (17.6 oz).

Environmental fate:

If released to soil, copper sulfate may leach to groundwater, be partly oxidised or bind to humic materials, clay or hydrous oxides of iron and manganese. In water, it will bind to carbonates as well as humic materials, clay and hydrous oxides of iron and manganese. Copper is accumulated by plants and animals, but it does not appear to biomagnify from plants to animals. In air, copper aerosols have a residence time of 2 to 10 days in an unpolluted atmosphere and 0.1 to greater than 4 days in polluted, urban areas.

Breakdown in soil and groundwater: Since copper is an element it will persist indefinitely. Copper is bound, or adsorbed, to organic materials, and to clay and mineral surfaces. The degree of adsorption to soils depends on the acidity or alkalinity of the soil. Because copper sulfate is highly water soluble, it is considered one of the more mobile metals in soils. However, because of its binding capacity, its leaching potential is low in all but sandy soils. When applied with irrigation water, copper sulfate does not accumulate in the surrounding soils. Some (60%) is deposited in the sediments at the bottom of the irrigation ditch, where it becomes adsorbed to clay, mineral, and organic particles. Copper compounds also settle out of solution.

Breakdown in water: As an element, copper can persist indefinitely. However, it will bind to water particulates and sediment.

Breakdown in vegetation: One of the limiting factors in the use of copper compounds is their serious potential for phytotoxicity. Copper sulfate can kill plants by disrupting photosynthesis. Blue-green algae in some copper sulfate treated Minnesota lakes became increasingly resistant to the algacide after 26 years of use

Ecotoxicity:

Effects on birds:

Oral (wild bird) LD50: 300 mg/kg; duck LD50: 600 mg/kg

Copper sulfate is practically nontoxic to birds. It poses less of a threat to birds than to other animals. The lowest lethal dose (LDLo) is 1000 mg/kg in pigeons and 600 mg/kg in ducks. The oral LD50 for Bordeaux mixture in young mallards is 2000 mg/kg.

Effects on aquatic organisms:

Fish LC50 (96 hr): rainbow trout, harlequin fish, goldfish, eel: 0.1-2.5 mg/l*

Daphnia magna LC50 (48 h): 24 ug/l*

*[The Dictionary of Substances and their Effects (Ed Richardson M.L.)]

Copper sulfate is highly toxic to fish. Even at recommended rates of application, this material may be poisonous to trout and other fish, especially in soft or acid waters. Its toxicity to fish generally decreases as water hardness increases. Fish eggs are more resistant than young fish fry to the toxic effects of copper sulfate. Copper sulfate is toxic to aquatic invertebrates, such as crab, shrimp, and oysters. The 96-hour LC50 of copper sulfate to pond snails is 0.39 mg/L at 20 C. Higher concentrations of the material caused some behavioral changes, such as secretion of mucous, and discharge of eggs and embryos.

Effects on other organisms: Bees are endangered by Bordeaux mixture. Copper sulfate may be poisonous to sheep and chickens at normal application rates. Most animal life in soil, including large earthworms, have been eliminated by the extensive use of copper containing fungicides in orchards

for magnesium compounds in general:

Fish LC50: 100-400 mg/l

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 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,

For boron and borates:

Environmental fate:

Boron is generally found in nature bound to oxygen and is never found as the free element. Atmospheric boron may be in the form of particulate matter or aerosols as borides, boron oxides, borates, boranes, organoboron compounds, trihalide boron compounds, or borazines. Borates are relatively soluble in water, and will probably be removed from the atmosphere by precipitation and dry deposition. The half-life of airborne particles is usually on the order of days, depending on the size of the particle and atmospheric conditions.

Boron readily hydrolyses in water to form the electrically neutral, weak monobasic acid boric acid (H3BO3) and the monovalent ion, B(OH)4-. In concentrated solutions, boron may polymerise, leading to the formation of complex and diverse molecular arrangements. Because most environmentally relevant boron minerals are highly soluble in water, it is unlikely that mineral equilibria will control the fate of boron in water. Boron was found to not be significantly removed during the conventional treatment of waste water. Boron may, however, be co-precipitated with aluminum, silicon, or iron to form hydroxyborate compounds on the surfaces of minerals.

Waterborne boron may be adsorbed by soils and sediments. Adsorption-desorption reactions are expected to be the only significant mechanism that will influence the fate of boron in water. The extent of boron adsorption depends on the pH of the water and the chemical composition of the soil. The greatest adsorption is generally observed at pH 7.5-9.0. The single most important property of soil that will influence the mobility of boron is the abundance of amorphous aluminum oxide. The extent of boron adsorption has also been attributed to the levels of iron oxide, and to a lesser extent, the organic matter present in the soil, although other studies found that the amount of organic matter present was not important. The adsorption of boron may not be reversible in some soils. The lack of reversibility may be the result of solid-phase formation on mineral surfaces and/or the slow release of boron by diffusion from the interior of clay minerals.

It is unlikely that boron is bioconcentrated significantly by organisms from water. A bioconcentration factor (BCF) relates the concentration of a chemical in the tissues of aquatic and terrestrial animals or plants to the concentration of the chemical in water or soil. The BCFs of boron in marine and freshwater plants, fish, and invertebrates were estimated to be <100. Experimentally measured BCFs for fish have ranged from 52 to 198. These BCFs suggest that boron is not significantly bioconcentrated.

As an element, boron itself cannot be degraded in the environment; however, it may undergo various reactions that change the form of boron (e.g., precipitation, polymerization, and acid-base reactions) depending on conditions such as its concentration in water and pH. In nature, boron in generally found in its oxygenated form. In aqueous solution, boron is normally present as boric acid and borate ions, with the dominant form of inorganic boron in natural aqueous systems as undissociated boric acid. Boric acid acts as an electron acceptor in aqueous solution, accepting an hydroxide ion from water to form (B(OH)4)-ion. In dilute solution, the favored form of boron is B(OH)4. In more concentrated solutions (>0.1 M boric acid) and at neutral to alkaline pH (6–11), polymeric species are formed (e.g., B3O3(OH)4-, B5O6(OH)4-, B3O3(OH)52-, and B4O5(OH)42-)

Most boron compounds are transformed to borates in soil due to the presence of moisture. Borates themselves are not further degraded in soil. However, borates can exist in a variety of forms in soil. Borates are removed from soils by water leaching and by assimilation by plants.

The most appreciable boron exposure to the general population is likely to be ingestion of food and to a lesser extent in water. As boron is a natural component of the environment, individuals will have some exposure from foods and drinking water

Boron-containing salts (borates) are ubiquitous in the environment. Surface soil, unpolluted waterways and seawater all typically contain significant amounts of boron as borate. Boron is an essential micronutrient for healthy growth of plants, however, it can be harmful to boron sensitive plants in higher quantities. In some areas such as the American Southwest, boron occurs naturally in surface waters in concentrations that have been shown to be toxic to commercially important plants.

Based on the collected information regarding aquatic toxicity, boron is not regarded as dangerous to aquatic organisms. The concentration in treated municipal waste water is a factor 100 lower than the NOEC-value for *Daphnia magna*.

No quality criteria exist for the concentration of boron in soil and compost. Boron is added to farmland when sewage sludge is applied as a soil improving agent, but there is not sufficient data to evaluate its effect on soil organisms. Being an essential micro-nutrient, no adverse effects of boron are expected at low concentrations.

Ecotoxicity:

In aquatic environments low concentrations of borates generally promote the growth of algae, whereas higher concentrations inhibited algal growth. In a growth inhibition test with *Scenedesmus subspicatus*, an EC50 value of 34 mg B/l was determined. Boric acid toxicity in *Daphnia* 48 h-LC50 (static test) was found to be 95 mg B/l. In a separate study it was concluded that chronic effects of boron to *Daphnia* may occur at a concentration of > 10 mg/l.

The toxicity of boron in fish is often higher in soft water than in hard water. The acute toxicity of boron towards *Danio rerio* (96 h-LC50) has been determined to 14.2 mg B/l . In a fish early life stage test with rainbow trout NOEC levels of boron have been determined in the range between 0.009 and 0.103 mg B/l, whereas the EC50 ranged from 27 to 100 mg B/l dependent on the water hardness.

For Ammonia:

Atmospheric Fate: Ammonia reacts rapidly with available acids (mainly sulfuric, nitric, and sometimes hydrochloric acid) to form the corresponding salts. Ammonia is persistent in the air.

Aquatic Fate: Biodegrades rapidly to nitrate, producing a high oxygen demand. Non-persistent in water (half-life 2 days).

Ecotoxicity: Moderately toxic to fish under normal temperature and pH conditions and harmful to aquatic life at low concentrations. Does not concentrate in food chain.

The nitrites 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.

Methaemoglobinemia 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 nitrites 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.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
potassium nitrate	LOW	LOW
calcium sulfate	HIGH	HIGH
zinc sulfate	HIGH	HIGH
copper sulfate, pentahydrate	HIGH	HIGH
ferrous sulfate heptahydrate	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
potassium nitrate	LOW (BCF = 3.162)
calcium sulfate	LOW (BCF = 3.162)
zinc sulfate	LOW (BCF = 112)
copper sulfate, pentahydrate	LOW (BCF = 3.162)
ferrous sulfate heptahydrate	LOW (BCF = 52)

Mobility in soil

Ingredient	Mobility
potassium nitrate	LOW (Log KOC = 14.3)

Ingredient	Mobility
calcium sulfate	LOW (Log KOC = 6.124)
zinc sulfate	LOW (Log KOC = 6.124)
copper sulfate, pentahydrate	LOW (Log KOC = 6.124)
ferrous sulfate heptahydrate	LOW (Log KOC = 6.124)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	- Containers may still present a chemical hazard/ danger when empty. - Return to supplier for reuse/ recycling if possible.
	Otherwise: - If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. - Where possible retain label warnings and SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: - Reduction - Reuse - Recycling - Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. In most instances the supplier of the material should be consulted. For ammonium nitrate: Add slowly to large amount of water, mix in slight excess of soda ash and neutralise with 6M hydrochloric acid before washing to sewer with large excess of water. Bury residue in an authorised landfill. Incineration requires scrubbers for NO_x, oxidation or reduction processes require scrubbers for NO DO NOT allow wash water from cleaning or process equipment to enter drains. - It may be necessary to collect all wash water for treatment before disposal. - In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. - Where in doubt contact the responsible authority.

SECTION 14 Transport information

Labels Required

Marine Pollutant	
HAZCHEM	2Z

Land transport (ADG)

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

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

(a) packagings;

(b) IBCs; or

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

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

Air transport (ICAO-IATA / DGR)

14.1. UN number	3077				
14.2 UN proper shipping name	Environmentally hazardous substance, solid, n.o.s. (contains copper sulfate, pentahydrate and zinc sulfate)				
14.3 Transport hazard class(es)	<table> <tr> <td>ICAO/IATA Class</td><td>9</td></tr> <tr> <td>ICAO / IATA Subsidiary Hazard</td><td>Not Applicable</td></tr> </table>	ICAO/IATA Class	9	ICAO / IATA Subsidiary Hazard	Not Applicable
ICAO/IATA Class	9				
ICAO / IATA Subsidiary Hazard	Not Applicable				

	ERG Code	9L
14.4. Packing group	III	
14.5. Environmental hazard	Environmentally hazardous	
14.6 Special precautions for user	Special provisions	A97 A158 A179 A197 A215
	Cargo Only Packing Instructions	956
	Cargo Only Maximum Qty / Pack	400 kg
	Passenger and Cargo Packing Instructions	956
	Passenger and Cargo Maximum Qty / Pack	400 kg
	Passenger and Cargo Limited Quantity Packing Instructions	Y956
	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G

Sea transport (IMDG-Code / GGVSee)

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

14.7. Maritime transport in bulk according to IMO instruments

14.7.1. Transport in bulk according to Annex II of MARPOL and the IBC code
Not Applicable

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

Product name	Group
ammonium nitrate	Not Applicable
potassium nitrate	Not Applicable
calcium sulfate	Not Applicable
magnesium oxide	Not Applicable
zinc sulfate	Not Applicable
copper sulfate, pentahydrate	Not Applicable
ferrous sulfate heptahydrate	Not Applicable
sodium borate, pentahydrate	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
ammonium nitrate	Not Applicable
potassium nitrate	Not Applicable
calcium sulfate	Not Applicable
magnesium oxide	Not Applicable
zinc sulfate	Not Applicable
copper sulfate, pentahydrate	Not Applicable
ferrous sulfate heptahydrate	Not Applicable
sodium borate, pentahydrate	Not Applicable

SECTION 15 Regulatory information

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

- ammonium nitrate is found on the following regulatory lists
- Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2A: Probably carcinogenic to humans
- potassium nitrate is found on the following regulatory lists
- Australian Inventory of Industrial Chemicals (AIIC)
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

calcium sulfate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

magnesium oxide is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

zinc sulfate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

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

Australian Inventory of Industrial Chemicals (AIIC)

copper sulfate, pentahydrate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

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

Australian Inventory of Industrial Chemicals (AIIC)

ferrous sulfate heptahydrate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

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

Australian Inventory of Industrial Chemicals (AIIC)

sodium borate, pentahydrate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Industrial Chemicals Environmental Management (Register) Instrument 2022 (IChEMS Register) - Schedule 2 - Relevant industrial chemicals that are unlikely to cause harm to the environment

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (potassium nitrate; calcium sulfate; magnesium oxide; zinc sulfate; copper sulfate, pentahydrate; ferrous sulfate heptahydrate; sodium borate, pentahydrate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
UAE - Control List (Banned/Restricted Substances)	No (magnesium oxide; zinc sulfate; ferrous sulfate heptahydrate)
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	12/08/2026
Initial Date	12/08/2026

Other information

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

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

Continued...

considered.

Definitions and abbreviations

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

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

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TEL (+61 3) 9572 4700.