Predator Raptor LiFePO4 Battery

R & J Batteries Pty Ltd

Chemwatch Hazard Alert Code: **4**

Chemwatch: **7952-69**
Version No: **2.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Issue Date: **24/04/2025**
Print Date: **24/04/2025**
L.GHS.AUS.EN.E

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

Product Identifier

Product name	Predator Raptor LiFePO4 Battery
Chemical Name	Not Applicable
Synonyms	Model Number;; PRL-04, PRL-06, PRL-09, PRL-12, PRL-18
Proper shipping name	LITHIUM ION BATTERIES (including lithium ion polymer batteries)
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	NOTE: Hazard statement relates to battery contents. Potential for exposure should not exist unless the battery leaks, is exposed to high temperatures or is mechanically, physically or electrically abused. Risk of exposure exists only in case of mechanical, electrical or thermal abuse. Thus the batteries should not short circuit, recharge, puncture, incinerate, crush, immerse in water, force discharge, or expose to temperatures above the temperature range of the cell or battery. Use according to manufacturer's directions. SDS are intended for use in the workplace ONLY. For domestic-use products, refer to consumer labels.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**R & J Batteries Pty Ltd**
Address	852 La Trobe St Ballarat Australia
Telephone	+61 3 5335 9888
Fax	+61 3 5336 4976
Website	rjbatt.com.au
Email	rjbatt@rjbatt.com.au

Emergency telephone number

Association / Organisation	**CHEMWATCH EMERGENCY RESPONSE (24/7)**
Emergency telephone number(s)	+61 1800 951 288 (ID#: 7952-69)
Other emergency telephone number(s)	+61 3 9573 3188

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	S6
Classification [1]	Acute Toxicity (Oral) Category 3, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Germ Cell Mutagenicity Category 1A, Specific Target Organ Toxicity - Repeated Exposure Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 2
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)

Page 1 continued...

H301	Toxic if swallowed.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H318	Causes serious eye damage.
H335	May cause respiratory irritation.
H340	May cause genetic defects.
H373	May cause damage to organs through prolonged or repeated exposure.
H411	Toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
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.
P330	Rinse mouth.
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.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
15365-14-7	36.8	lithium iron phosphate
7782-42-5	17.5	graphite
623-53-0	14.6	ethyl methyl carbonate
7440-50-8	8.2	copper
7429-90-5	13.6	aluminium
96-49-1	7.3	ethylene carbonate
21324-40-3	2	lithium fluorophosphate
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available***	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. ▸ Generally not applicable.
Skin Contact	If skin or hair contact occurs: ▸ Immediately flush body and clothes with large amounts of water, using safety shower if available.

Continued...

	▸ 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. ▸ Generally not applicable.
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. ▸ Generally not applicable.
Ingestion	▸ **IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY.** ▸ For advice, contact a Poisons Information Centre or a doctor. ▸ Urgent hospital treatment is likely to be needed. ▸ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. ▸ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist. ▸ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS. **Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:** ▸ **INDUCE** vomiting with fingers down the back of the throat, **ONLY IF CONSCIOUS**. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. **NOTE:** Wear a protective glove when inducing vomiting by mechanical means. ▸ Generally not applicable.

Indication of any immediate medical attention and special treatment needed

for phosphate salts intoxication:

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

Treat symptomatically.

SECTION 5 Firefighting measures

Extinguishing media

Metal dust fires need to be smothered with sand, inert dry powders.
DO NOT USE WATER, CO2 or FOAM.

- Use DRY sand, graphite powder, dry sodium chloride based extinguishers, G-1 or Met L-X to smother fire.
- Confining or smothering material is preferable to applying water as chemical reaction may produce flammable and explosive hydrogen gas.
- Chemical reaction with CO2 may produce flammable and explosive methane.
- If impossible to extinguish, withdraw, protect surroundings and allow fire to burn itself out.
- Sand, dry powder extinguishers or other inerts should be used to smother dust fires.

At temperatures above 1500 C, carbon, graphite or graphene reacts with substances containing oxygen, including water and carbon dioxide. In case of intensely hot fires sand should be used to cover and isolate these materials.

- **DO NOT** use halogenated fire extinguishing agents.

Special hazards arising from the substrate or mixture

Fire Incompatibility	▸ Reacts with acids producing flammable / explosive hydrogen (H2) gas ▸ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result ▸ Keep dry ▸ **NOTE:** May develop pressure in containers; open carefully. Vent periodically.

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. Slight hazard when exposed to heat, flame and oxidisers.
Fire/Explosion Hazard	▸ **DO NOT** disturb burning dust. Explosion may result if dust is stirred into a cloud, by providing oxygen to a large surface of hot metal. ▸ **DO NOT** use water or foam as generation of explosive hydrogen may result. With the exception of the metals that burn in contact with air or water (for example, sodium), masses of combustible metals do not represent unusual fire risks because they have the ability to conduct heat away from hot spots so efficiently that the heat of combustion cannot be maintained - this means that it will require a lot of heat to ignite a mass of combustible metal. Generally, metal fire risks exist when sawdust, machine shavings and other metal 'fines' are present. Metal powders, while generally regarded as non-combustible: ▸ May burn when metal is finely divided and energy input is high. ▸ May react explosively with water. ▸ May be ignited by friction, heat, sparks or flame. ▸ May **REIGNITE** after fire is extinguished. ▸ Will burn with intense heat. Note: ▸ Metal dust fires are slow moving but intense and difficult to extinguish. ▸ Containers may explode on heating. ▸ Dusts or fumes may form explosive mixtures with air. ▸ Gases generated in fire may be poisonous, corrosive or irritating.

Continued...

	▸ Hot or burning metals may react violently upon contact with other materials, such as oxidising agents and extinguishing agents used on fires involving ordinary combustibles or flammable liquids. ▸ Temperatures produced by burning metals can be higher than temperatures generated by burning flammable liquids ▸ Some metals can continue to burn in carbon dioxide, nitrogen, water, or steam atmospheres in which ordinary combustibles or flammable liquids would be incapable of burning. Combustion products include: carbon monoxide (CO) carbon dioxide (CO2) phosphorus oxides (POx) metal oxides other pyrolysis products typical of burning organic material. Articles and manufactured articles may constitute a fire hazard where polymers form their outer layers or where combustible packaging remains in place. Certain substances, found throughout their construction, may degrade or become volatile when heated to high temperatures. This may create a secondary hazard.
HAZCHEM	2Y

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. ▸ Clean up all spills immediately. ▸ Secure load if safe to do so. ▸ Bundle/collect recoverable product. ▸ Collect remaining material in containers with covers for disposal.
Major Spills	Environmental hazard - contain spillage. · Do not use compressed air to remove metal dusts from floors, beams or equipment · Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation. · Use non-sparking handling equipment, tools and natural bristle brushes. · Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations · Cover and reseal partially empty containers. · Do not allow chips, fines or dusts to contact water, particularly in enclosed areas. If molten: ▸ Contain the flow using dry sand or salt flux as a dam. ▸ All tooling (e.g., shovels or hand tools) and containers which come in contact with molten metal must be preheated or specially coated, rust free and approved for such use. ▸ Allow the spill to cool before remelting scrap. ▸ Clean up all spills immediately. ▸ Wear protective clothing, safety glasses, dust mask, gloves. ▸ Secure load if safe to do so. Bundle/collect recoverable product. ▸ Use dry clean up procedures and avoid generating dust. ▸ Vacuum up (consider explosion-proof machines designed to be grounded during storage and use). ▸ Water may be used to prevent dusting. ▸ Collect remaining material in containers with covers for disposal. ▸ Flush spill area with water. Minor hazard. ▸ Clear area of personnel. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Control personal contact with the substance, by using protective equipment as required. ▸ Prevent spillage from entering drains or water ways. ▸ Contain spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Absorb remaining product with sand, earth or vermiculite and place in appropriate containers for disposal. ▸ Wash area and prevent runoff into drains or waterways. ▸ 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	For molten metals: · Molten metal and water can be an explosive combination. The risk is greatest when there is sufficient molten metal to entrap or seal off water. Water and other forms of contamination on or contained in scrap or remelt ingot are known to have caused explosions in melting operations. While the products may have minimal surface roughness and internal voids, there remains the possibility of moisture contamination or entrapment. If confined, even a few drops can lead to violent explosions. · All tooling, containers, molds and ladles, which come in contact with molten metal must be preheated or specially coated, rust free and approved for such use. · Any surfaces that may contact molten metal (e.g. concrete) should be specially coated · Drops of molten metal in water (e.g. from plasma arc cutting), while not normally an explosion hazard, can generate enough flammable hydrogen gas to present an explosion hazard. Vigorous circulation of the water and removal of the particles minimise the hazard. During melting operations, the following minimum guidelines should be observed: · Inspect all materials prior to furnace charging and completely remove surface contamination such as water, ice, snow, deposits of grease and oil or other surface contamination resulting from weather exposure, shipment, or storage. · Store materials in dry, heated areas with any cracks or cavities pointed downwards. · Preheat and dry large objects adequately before charging in to a furnace containing molten metal. This is typically done by the use of a drying oven or homogenising furnace. The dry cycle should bring the metal temperature of the coldest item of the batch to 200 degree C (400 deg F) and then hold at that temperature for 6 hours. ▸ Avoid all personal contact, including inhalation.

Continued...

	▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ **DO NOT** allow material to contact humans, exposed food or food utensils. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Store away from incompatible materials.

Conditions for safe storage, including any incompatibilities

Suitable container	Generally packaging as originally supplied with the article or manufactured item is sufficient to protect against physical hazards. If repackaging is required ensure the article is intact and does not show signs of wear. As far as is practicably possible, reuse the original packaging or something providing a similar level of protection to both the article and the handler.
Storage incompatibility	▸ Segregate from alcohol, water. ▸ Avoid reaction with oxidising agents ▸ Avoid strong acids, bases.

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	graphite	Graphite (all forms except fibres) (respirable dust) (natural & synthetic)	3 mg/m3	Not Available	Not Available	(e) Containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	copper	Copper, dusts & mists (as Cu)	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	copper	Copper (fume)	0.2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium	Aluminium, pyro powders (as Al)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium	Aluminium (metal dust)	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium	Aluminium (welding fumes) (as Al)	5 mg/m3	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
lithium iron phosphate	Not Available	Not Available
graphite	1,250 mg/m3	Not Available
ethyl methyl carbonate	Not Available	Not Available
copper	100 mg/m3	Not Available
aluminium	Not Available	Not Available
ethylene carbonate	Not Available	Not Available
lithium fluorophosphate	Not Available	Not Available

MATERIAL DATA

For graphite:
Graphite pneumoconiosis resembles coal workers' pneumoconiosis. Data indicate that the higher the crystalline silica content of graphite the more likely the disease will increase in severity. The presence of anthracite coal in the production of some synthetic grades of graphite appears to make arbitrary the use of the term, "synthetic", "artificial" or "natural".

The TLV-TWA for carbon black is recommended to minimise complaints of excessive dirtiness and applies only to commercially produced carbon blacks or to soots derived from combustion sources containing absorbed polycyclic aromatic hydrocarbons (PAHs). When PAHs are present in carbon black (measured as the cyclohexane-extractable fraction) NIOSH has established a REL-TWA of 0.1 mg/m3 and considers the material to be an occupational carcinogen.
The NIOSH REL-TWA was "selected on the basis of professional judgement rather than on data delineating safe from unsafe concentrations of PAHs".
This limit was justified on the basis of feasibility of measurement and not on a demonstration of its safety.
For aluminium oxide and pyrophoric grades of aluminium:
Twenty seven year experience with aluminium oxide dust (particle size 96% 1,2 um) without adverse effects either systemically or on the lung, and at a calculated concentration equivalent to 2 mg/m3 over an 8-hour shift has lead to the current recommendation of the TLV-TWA.
The limit should also apply to aluminium pyro powders whose toxicity is reportedly greater than aluminium dusts and should be protective against lung changes.

For aluminium oxide:
The experimental and clinical data indicate that aluminium oxide acts as an "inert" material when inhaled and seems to have little effect on the lungs nor does it produce significant organic disease or toxic effects when exposures are kept under reasonable control.
[Documentation of the Threshold Limit Values], ACGIH, Sixth Edition

Continued...

Exposure controls

Appropriate engineering controls

Articles or manufactured items, in their original condition, generally don't require engineering controls during handling or in normal use. Exceptions may arise following extensive use and subsequent wear, during recycling or disposal operations where substances, found in the article, may be released to the environment.

Metal dusts must be collected at the source of generation as they are potentially explosive.

- Avoid ignition sources.
- Good housekeeping practices must be maintained.
- Dust accumulation on the floor, ledges and beams can present a risk of ignition, flame propagation and secondary explosions.
- Do not use compressed air to remove settled materials from floors, beams or equipment
- Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation.
- Use non-sparking handling equipment, tools and natural bristle brushes. Cover and reseal partially empty containers. Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations.
- Do not allow chips, fines or dusts to contact water, particularly in enclosed areas.
- Metal spraying and blasting should, where possible, be conducted in separate rooms. This minimises the risk of supplying oxygen, in the form of metal oxides, to potentially reactive finely divided metals such as aluminium, zinc, magnesium or titanium.
- Work-shops designed for metal spraying should possess smooth walls and a minimum of obstructions, such as ledges, on which dust accumulation is possible.
- Wet scrubbers are preferable to dry dust collectors.
- Bag or filter-type collectors should be sited outside the workrooms and be fitted with explosion relief doors.
- Cyclones should be protected against entry of moisture as reactive metal dusts are capable of spontaneous combustion in humid or partially wetted states.
- Local exhaust systems must be designed to provide a minimum capture velocity at the fume source, away from the worker, of 0.5 metre/sec.
- Local ventilation and vacuum systems must be designed to handle explosive dusts. Dry vacuum and electrostatic precipitators must not be used, unless specifically approved for use with flammable/ explosive dusts.

Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
welding, brazing fumes (released at relatively low velocity into moderately still air)	0.5-1.0 m/s (100-200 f/min.)

Within each range the appropriate value depends on:

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

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

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].
- Safety glasses with side shields.
- Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].

No special equipment required due to the physical form of the product.

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.

No special equipment required due to the physical form of the product.

Body protection

See Other protection below

Other protection

· During repair or maintenance activities the potential exists for exposures to toxic metal particulate in excess of the occupational standards. Under these circumstances, protecting workers can require the use of specific work practices or procedures involving the combined use of

Continued...

	ventilation, wet and vacuum cleaning methods, respiratory protection, decontamination, special protective clothing, and when necessary, restricted work zones. · Protective over-garments or work clothing must be worn by persons who may become contaminated with particulate during activities such as machining, furnace rebuilding, air cleaning equipment filter changes, maintenance, furnace tending, etc. Contaminated work clothing and over-garments must be managed in a controlled manner to prevent secondary exposure to workers of third parties, to prevent the spread of particulate to other areas, and to prevent particulate from being taken home by workers. · Personnel who handle and work with molten metal should utilise primary protective clothing like polycarbonate face shields, fire resistant tapper's jackets, neck shades (snoods), leggings, spats and similar equipment to prevent burn injuries. In addition to primary protection, secondary or day-to-day work clothing that is fire resistant and sheds metal splash is recommended for use with molten metal. Synthetic materials should never be worn even as secondary clothing (undergarments).

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required. Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS	-	A-PAPR-AUS / Class 1
up to 50 x ES	-	A-AUS / Class 1	-
up to 100 x ES	-	A-2	A-PAPR-2 ^

^ - Full-face
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)

Respiratory protection not normally required due to the physical form of the product.
Where significant concentrations of the material are likely to enter the breathing zone, a Class P3 respirator may be required.
Class P3 particulate filters are used for protection against highly toxic or highly irritant particulates.
Filtration rate: Filters at least 99.95% of airborne particles
Suitable for:
· Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.
· Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.
· Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS
· Highly toxic particles e.g. Organophosphate Insecticides, Radionuclides, Asbestos
Note: P3 Rating can only be achieved when used with a Full Face Respirator or Powered Air-Purifying Respirator (PAPR). If used with any other respirator, it will only provide filtration protection up to a P2 rating.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Quadrate, odourless, solid battery.

Physical state	Manufactured	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Applicable
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Not Available	**pH as a solution (1%)**	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

Continued...

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	Based on available data, the classification criteria are not met.
h) STOT - Single Exposure	There is sufficient evidence to classify this material as toxic to specific organs through single exposure
i) STOT - Repeated Exposure	There is sufficient evidence to classify this material as toxic to specific organs through repeated exposure
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	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. 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. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Copper poisoning following exposure to copper dusts and fume may result in headache, cold sweat and weak pulse. Capillary, kidney, liver and brain damage are the longer term manifestations of such poisoning. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure. Inhalation of dusts, generated by the material, during the course of normal handling, may produce severely toxic effects; these may be fatal.
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. Severely toxic effects may result from the accidental ingestion of the material; animal experiments indicate that ingestion of less than 5 gram may be fatal or may produce serious damage to the health of the individual.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact. The material may accentuate any pre-existing dermatitis condition Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus. Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles. Irritation and skin reactions are possible with sensitive skin Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. There is sufficient evidence to provide a strong presumption that human exposure to the material may produce heritable genetic damage.

Continued...

There is sufficient evidence to provide a strong presumption that human exposure to the material may result in the development of heritable genetic damage, generally on the basis of
- appropriate animal studies,
- other relevant information
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.

Predator Raptor LiFePO4 Battery	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

lithium iron phosphate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: 2000 mg/kg[1]	Not Available
	Inhalation (Rat) LC50: >3.2 mg/l4h[1]	
	Oral (Rat) LD50: >2000 mg/kg[1]	

graphite	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: >2 mg/L4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >200 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

ethyl methyl carbonate	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: >17.6 mg/l4h[1]	Not Available
	Oral (Rat) LD50: >5000 mg/kg[1]	

copper	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: 0.733 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Mouse) LD50; 0.7 mg/kg[2]	

aluminium	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: >2.3 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

ethylene carbonate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[1]	Skin (Rodent - rabbit): 660mg - Mild
		Skin: no adverse effect observed (not irritating)[1]

lithium fluorophosphate	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 50-300 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
		Skin: adverse effect observed (corrosive)[1]

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

Predator Raptor LiFePO4 Battery

For aluminium compounds:
Aluminium present in food and drinking water is poorly absorbed through the gastrointestinal tract. The bioavailability of aluminium is dependent on the form in which it is ingested and the presence of dietary constituents with which the metal cation can complex Ligands in food can have a marked effect on absorption of aluminium, as they can either enhance uptake by forming absorbable (usually water soluble) complexes (e.g., with carboxylic acids such as citric and lactic), or reduce it by forming insoluble compounds (e.g., with phosphate or dissolved silicate).
Considering the available human and animal data it is likely that the oral absorption of aluminium can vary 10-fold based on chemical form alone. Although bioavailability appears to generally parallel water solubility, insufficient data are available to directly extrapolate from solubility in water to bioavailability.
For oral intake from food, the European Food Safety Authority (EFSA) has derived a tolerable weekly intake (TWI) of 1 milligram (mg) of aluminium per kilogram of bodyweight. In its health assessment, the EFSA states a medium bioavailability of 0.1 % for all aluminium compounds which are ingested with food. This corresponds to a systemically available tolerable daily dose of 0.143 microgrammes (µg) per kilogramme (kg) of body weight. This means that for an adult weighing 60 kg, a systemically available dose of 8.6 µg per day is considered safe.
Based on a neuro-developmental toxicity study of aluminium citrate administered via drinking water to rats, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) established a Provisional Tolerable Weekly Intake (PTWI) of 2 mg/kg bw (expressed as aluminium) for all aluminium compounds in food, including food additives. The Committee on Toxicity of chemicals in food, consumer products and the environment (COT) considers that the derivation of this PTWI was sound and that it should be used in assessing potential risks from dietary exposure to aluminium.
The Federal Institute for Risk Assessment (BfR) of Germany has assessed the estimated aluminium absorption from antiperspirants. For this purpose, the data, derived from experimental studies, on dermal absorption of aluminium from antiperspirants for healthy and damaged skin was used as a basis. At about 10.5 µg, the calculated systemic intake values for healthy skin are above the 8.6 µg per day that are considered safe for an adult weighing 60 kg. If aluminium -containing antiperspirants are used on a daily basis, the tolerable weekly intake determined by the EFSA is therefore exceeded. The values for damaged skin, for example injuries from shaving, are many times higher. This means that in case of daily use of an aluminium-containing antiperspirant alone, the TWI may be completely exhausted. In addition, further aluminium absorption sources such as food, cooking utensils and other cosmetic products must be taken into account
Systemic toxicity after repeated exposure

Continued...

	No studies were located regarding dermal effects in animals following intermediate or chronic-duration dermal exposure to various forms of aluminium. When orally administered to rats, aluminium compounds (including aluminium nitrate, aluminium sulfate and potassium aluminium sulfate) have produced various effects, including decreased gain in body weight and mild histopathological changes in the spleen, kidney and liver of rats (104 mg Al/kg bw/day) and dogs (88-93 mg Al/kg bw/day) during subchronic oral exposure. Effects on nerve cells, testes, bone and stomach have been reported at higher doses. Severity of effects increased with dose. The main toxic effects of aluminium that have been observed in experimental animals are neurotoxicity and nephrotoxicity. Neurotoxicity has also been described in patients dialysed with water containing high concentrations of aluminium, but epidemiological data on possible adverse effects in humans at lower exposures are inconsistent Reproductive and developmental toxicity: Studies of reproductive toxicity in male mice (intraperitoneal or subcutaneous administration of aluminium nitrate or chloride) and rabbits (administration of aluminium chloride by gavage) have demonstrated the ability of aluminium to cause testicular toxicity, decreased sperm quality in mice and rabbits and reduced fertility in mice. No reproductive toxicity was seen in females given aluminium nitrate by gavage or dissolved in drinking water. Multi-generation reproductive studies in which aluminium sulfate and aluminium ammonium sulfate were administered to rats in drinking water, showed no evidence of reproductive toxicity High doses of aluminium compounds given by gavage have induced signs of embryotoxicity in mice and rats in particular, reduced fetal body weight or pup weight at birth and delayed ossification. Developmental toxicity studies in which aluminium chloride was administered by gavage to pregnant rats showed evidence of foetotoxicity, but it was unclear whether the findings were secondary to maternal toxicity. A twelve-month neuro-development with aluminium citrate administered via the drinking water to Sprague-Dawley rats, was conducted according to Good Laboratory Practice (GLP). Aluminium citrate was selected for the study since it is the most soluble and bioavailable aluminium salt. Pregnant rats were exposed to aluminium citrate from gestational day 6 through lactation, and then the offspring were exposed post-weaning until postnatal day 364. An extensive functional observational battery of tests was performed at various times. Evidence of aluminium toxicity was demonstrated in the high (300 mg/kg bw/day of aluminium) and to a lesser extent, the mid-dose groups (100 mg/kg bw/day of aluminium). In the high-dose group, the main effect was renal damage, resulting in high mortality in the male offspring. No major neurological pathology or neurobehavioural effects were observed, other than in the neuromuscular subdomain (reduced grip strength and increased foot splay). Thus, the lowest observed adverse effect level (LOAEL) was 100 mg/kg bw/day and the no observed adverse effect level (NOAEL) was 30 mg/kg bw/day. Bioavailability of aluminium chloride, sulfate and nitrate and aluminium hydroxide was much lower than that of aluminium citrate This study was used by JECFA as key study to derive the PTWI. Genotoxicity Aluminium compounds were non-mutagenic in bacterial and mammalian cell systems, but some produced DNA damage and effects on chromosome integrity and segregation in vitro. Clastogenic effects were also observed in vivo when aluminium sulfate was administered at high doses by gavage or by the intraperitoneal route. Several indirect mechanisms have been proposed to explain the variety of genotoxic effects elicited by aluminium salts in experimental systems. Cross-linking of DNA with chromosomal proteins, interaction with microtubule assembly and mitotic spindle functioning, induction of oxidative damage, damage of lysosomal membranes with liberation of DNAase, have been suggested to explain the induction of structural chromosomal aberrations, sister chromatid exchanges, chromosome loss and formation of oxidized bases in experimental systems. The EFSA Panel noted that these indirect mechanisms of genotoxicity, occurring at relatively high levels of exposure, are unlikely to be of relevance for humans exposed to aluminium via the diet. Aluminium compounds do not cause gene mutations in either bacteria or mammalian cells. Exposure to aluminium compounds does result in both structural and numerical chromosome aberrations both in in-vitro and in-vivo mutagenicity tests. DNA damage is probably the result of indirect mechanisms. The DNA damage was observed only at high exposure levels. Carcinogenicity. The available epidemiological studies provide limited evidence that certain exposures in the aluminium production industry are carcinogenic to humans, giving rise to cancer of the lung and bladder. However, the aluminium exposure was confounded by exposure to other agents including polycyclic aromatic hydrocarbons, aromatic amines, nitro compounds and asbestos. There is no evidence of increased cancer risk in non-occupationally exposed persons. Neurodegenerative diseases. Following the observation that high levels of aluminium in dialysis fluid could cause a form of dementia in dialysis patients, a number of studies were carried out to determine if aluminium could cause dementia or cognitive impairment as a consequence of environmental exposure over long periods. Aluminium was identified, along with other elements, in the amyloid plaques that are one of the diagnostic lesions in the brain for Alzheimer disease, a common form of senile and pre-senile dementia. some of the epidemiology studies suggest the possibility of an association of Alzheimer disease with aluminium in water, but other studies do not confirm this association. All studies lack information on ingestion of aluminium from food and how concentrations of aluminium in food affect the association between aluminium in water and Alzheimer disease." There are suggestions that persons with some genetic variants may absorb more aluminium than others, but there is a need for more analytical research to determine whether aluminium from various sources has a significant causal association with Alzheimer disease and other neurodegenerative diseases.Aluminium is a neurotoxicant in experimental animals. However, most of the animal studies performed have several limitations and therefore cannot be used for quantitative risk assessment. Contact sensitivity: It has been suggested that the body burden of aluminium may be linked to different iseases. Macrophagic myofasciitis and chronic fatigue syndrome can be caused by aluminium-containing adjuvants in vaccines. Macrophagic myofasciitis (MMF) has been described as a disease in adults presenting with ascending myalgia and severe fatigue following exposure to aluminium hydroxide-containing vaccines The corresponding histological findings include aluminium-containing macrophages infiltrating muscle tissue at the injection site. The hypothesis is that the long-lasting granuloma triggers the development of the systemic syndrome. Aluminium acts not only as an adjuvant,stimulating the immune system either to fend off infections or to tolerate antigens, it also acts as a sensitisers causing contact allergy and allergic contact dermatitis. In general, metal allergies are very common and aluminium is considered to be a weak allergen. A metal must be ionised to be able to act as a contact allergen, then it has to undergo haptenisation to be immunogenic and to initiate an immune response.Once inside the skin, the metal ions must bind to proteins to become immunologically reactive.The most important routes of exposure and sensitisation to aluminium are through aluminium-containing vaccines. One Swedish study showed a statistically significant association between contact allergy to aluminium and persistent itching nodules in children treated with allergen-specific immunotherapy (ASIT) Nodules were overrepresented in patients with contact allergy to aluminium Other routes of sensitisation reported in the literature are the prolonged use of aluminium-containing antiperspirants, topical medication, and tattooing of the skin with aluminium-containing pigments. Most of the patients experienced eczematous reactions whereas tattooing caused granulomas. Even though aluminium is used extensively in industry, only a low number of cases of occupational skin sensitisation to aluminium have been reported Systemic allergic contact dermatitis in the form of flare-up reactions after re-exposure to aluminium has been documented: pruritic nodules at present and previous injection sites, eczema at the site of vaccination as well as at typically atopic localisations after vaccination with aluminium-containing vaccines and/or patch testing with aluminium, and also after use of aluminium-containing toothpaste The material may trigger oculogyric crisis. The term "oculogyric" refers to the bilateral elevation of the visual gaze. Initial symptoms include restlessness, agitation, malaise, or a fixed stare. Then comes the more characteristically described extreme and sustained upward deviation of the eyes. In addition, the eyes may converge, deviate upward and laterally, or deviate downward. The most frequently reported associated findings are backwards and lateral flexion of the neck, widely opened mouth, tongue protrusion, and ocular pain. However, the condition may also be associated with intensely painful jaw spasm which may result in the breaking of a tooth. A wave of exhaustion may follow an episode. The abrupt termination of the psychiatric symptoms at the conclusion of the crisis is most striking. Other features that are noted during attacks include mutism, palilalia, eye blinking, lacrimation, pupil dilation, drooling, respiratory dyskinesia, increased blood pressure and heart rate, facial flushing, headache, vertigo, anxiety, agitation, compulsive thinking, paranoia, depression, recurrent fixed ideas, depersonalization, violence, and obscene language. In addition to the acute presentation, oculogyric crisis can develop as a recurrent syndrome, triggered by stress and by exposure to the drugs. The diagnosis of oculogyric crisis is largely clinical and involves taking a focused history and physical examination to identify possible triggers for the crisis and rule out other causes of abnormal ocular movements.
COPPER	WARNING: Inhalation of high concentrations of copper fume may cause "metal fume fever", an acute industrial disease of short duration. Symptoms are tiredness, influenza like respiratory tract irritation with fever. for copper and its compounds (typically copper chloride):

Continued...

	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).
ETHYLENE CARBONATE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin. for ethylene carbonate **Mammalian toxicity:** Reliable acute toxicity tests are available on ethylene carbonate. Ethylene carbonate is practically nontoxic following acute oral exposure in a test that meets OECD and EPA test guidelines; the LD50 is >5000 mg/kg. The dermal LD50 is >2000 mg/kg, in a test that meets OECD and EPA test guidelines. Ethylene carbonate is rapidly metabolized to ethylene glycol. Following gavage administration to rats, ethylene carbonate is rapidly converted into ethylene glycol; the half-life for disappearance of ethylene carbonate from blood was 0.25 hours. As a result, the mammalian toxicity of ethylene carbonate is nearly identical to that of ethylene glycol for endpoints where both have been tested Ethylene carbonate was mixed in the diet of 26 male and 26 female Crl: CD(SD) rats for 18 months at concentrations of 25,000 ppm for males and females and 50,000 ppm for females; males were also fed 50,000 ppm for 42 weeks, and 40,000 ppm for 16 weeks. Survivors were observed to 24 months. Compound intake (mg/kg/day) was not reported, but is estimated to be approximately 250 and 500 mg/kg/day. No toxic effects were found in females, but increased mortality was seen in males at both dose levels. No high-dose males survived week 60 and only 10 low-dose males survived to week 78. Males had severe nephrotoxicity, characteristic of ethylene glycol toxicity. The following ***in vitro*** genotoxicity tests were conducted on ethylene carbonate, without indications of genotoxicity: an Ames mutagenicity assay, an unscheduled DNA synthesis assay using rat hepatocytes, and a cell transformation assay using BALB/3T3 cells. No ***in vivo*** genotoxicity studies on ethylene carbonate were found; however, ethylene glycol has been tested and was negative in a rat dominant lethal assay. Gavage administration of ethylene carbonate to pregnant rats days 6-15 of gestation resulted in systemic toxicity at doses of 3000 mg/kg/day, including post-dose salivation. The NOAEL for maternal toxicity was 1500 mg/kg/day. Similar to ethylene glycol, there were increased soft tissue (hydrocephalus, umbilical herniation, gastroschisis, cleft palate, misshapen and compressed stomach) and skeletal malformations at 3000 mg/kg/day, but not at 1500 mg/kg/day. For ethylene glycol: Ethylene glycol is quickly and extensively absorbed throughout the gastrointestinal tract. Limited information suggests that it is also absorbed through the airways; absorption through skin is apparently slow. Following absorption, it is distributed throughout the body. In humans, it is initially metabolized by alcohol dehydrogenase to form glycoaldehyde, which is rapidly converted to glycolic acid and glyoxal. These breakdown products are oxidized to glyoxylate, which may be further metabolized to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate carbon dioxide, which is one of the major elimination products of ethylene glycol. In addition to exhaled carbon dioxide, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination is rapid and occurs within a few hours. Respiratory effects: Respiratory system involvement occurs 12-24 hours after swallowing sufficient amounts of ethylene glycol. Symptoms include hyperventilation, shallow rapid breathing, and generalized swelling of the lungs with calcium oxalate deposits occasionally appearing in the lungs. Respiratory system involvement appears to be dose-dependent and occurs at the same time as cardiovascular changes. Later, there may be other changes compatible with adult respiratory distress syndrome (ARDS). Swelling of the lung can be a result of heart failure, ARDS, or aspiration of stomach contents. Symptoms related to acidosis such as fast or excessive breathing are frequently observed; however, major symptoms such as swelling of the lung and inflammation of the bronchi and lungs are relatively rare, and are usually seen only in extreme poisoning. Cardiovascular effects: Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of ethylene glycol poisoning by swallowing, which is 12-24 hours after acute exposure. The symptoms of poisoning involving the heart include increased heart rate, heart enlargement and ventricular gallop. There may also be high or low blood pressure, which may progress to cardiogenic shock. In lethal cases, inflammation of the heart muscle has been observed at autopsy. Cardiovascular involvement appears to be rare and usually seen after swallowing higher doses of ethylene glycol. In summary, acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown. Gastrointestinal effects: Common early acute effects of swallowing ethylene glycol include nausea, vomiting with or without blood, heartburn and abdominal cramping and pain. One patient showed intermittent diarrhea and pain, and after surgery, deposition of oxalate crystals was shown to have occurred. Musculoskeletal effects: Reported musculoskeletal effects in cases of acute ethylene glycol poisoning include diffuse muscle tenderness and pain, associated with high levels of creatinine in the blood, and jerks and contractions associated with low calcium. Liver effects: Autopsies carried out on people who died following acute ethylene glycol poisoning showed deposition of calcium oxalate in the liver as well as hydropic and fatty degeneration and cell death (necrosis) of the liver. Kidney effects: Adverse kidney effects are seen during the third stage of ethylene glycol poisoning, 2-3 days after acute exposure. Calcium oxalate crystals are deposited in the tubules and are seen in the urine. There may also be degeneration and death of tubule cells, and inflammation of the tubule interstitium. If untreated, the degree of kidney damage progresses and leads to blood and protein in the urine, decreased kidney function, reduction in urine output and ultimately, kidney failure. With adequate supportive therapy, kidney function can return to normal or near normal. Metabolic effects: Metabolic changes can occur within 12 hours of exposure to ethylene glycol. There may be metabolic acidosis, caused by accumulation of glycolic acid in the blood and therefore a reduction in blood pH. The anion gap is increased, due to increased unmeasured anions (mainly glycolate).

Continued...

	Effects on the nervous system: Adverse reactions involving the nervous system are among the first symptoms to appear in humans after ethylene glycol is swallowed. These early effects are also the only symptoms caused by unmetabolised ethylene glycol. Together with metabolic effects (see above), they occur from 0.5-12 hours after exposure and are considered to be part of the first stage in ethylene glycol poisoning. Inco-ordination, slurred speech, confusion and sleepiness are common in the early stages, as are irritation, restlessness and disorientation. Later, there may be effects on cranial nerves (which may be reversible over many months). Swelling of the brain (cerebrum) and crystal deposits of calcium oxalate in the walls of the small blood vessels of the brain were found at autopsy in people who died after acute ethylene glycol poisoning. Reproductive effects: Animal testing showed that ethylene glycol may affect fertility, survival of fetuses and the male reproductive organs. Effects on development: Animal studies indicate that birth defects may occur after exposure in pregnancy; there may also be reduction in foetal weight. Cancer: No studies are known regarding cancer effects in humans or animal, after skin exposure to ethylene glycol. Genetic toxicity: No human studies available, but animal testing results are consistently negative.
Predator Raptor LiFePO4 Battery & COPPER	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.
Predator Raptor LiFePO4 Battery & GRAPHITE & ETHYLENE CARBONATE & LITHIUM FLUOROPHOSPHATE	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.
Predator Raptor LiFePO4 Battery & LITHIUM IRON PHOSPHATE & GRAPHITE & ETHYL METHYL CARBONATE & ALUMINIUM & LITHIUM FLUOROPHOSPHATE	No significant acute toxicological data identified in literature search.
Predator Raptor LiFePO4 Battery & LITHIUM IRON PHOSPHATE	Goitrogenic:. Goitrogens are substances that suppress the function of the thyroid gland by interfering with iodine uptake, which can, as a result, cause an enlargement of the thyroid, i.e., a goitre Goitrogens include: ▸ Vitexin, a flavanoid, which inhibits thyroid peroxidase thus contributing to goiter. ▸ Ions such as thiocyanate and perchlorate which decrease iodide uptake by competitive inhibition; as a consequence of reduced thyroxine and triiodothyronine secretion by the gland, at low doses, this causes an increased release of thyrotropin (by reduced negative feedback), which then stimulates the gland. ▸ Lithium which inhibits thyroid hormone release. ▸ Certain foods, such as soy and millet (containing vitexins) and vegetables in the genus Brassica (e.g. broccoli, brussels sprouts, cabbage, horseradish). ▸ Caffeine (in coffee, tea, cola, chocolate) which acts on thyroid function as a suppressant.

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

Predator Raptor LiFePO4 Battery

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

lithium iron phosphate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>28mg/l	2
EC50	72h	Algae or other aquatic plants	>24mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	>=24mg/l	2
LC50	96h	Fish	>28mg/l	2

graphite

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>100mg/l	2
NOEC(ECx)	96h	Fish	>=100mg/l	2
EC50	72h	Algae or other aquatic plants	>100mg/l	2
LC50	96h	Fish	>100mg/l	2

ethyl methyl carbonate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>100mg/l	2

Continued...

	EC50	72h	Algae or other aquatic plants	>62mg/l	2
	LC50	96h	Fish	>100mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	62mg/l	2

copper					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	NOEC(ECx)	48h	Fish	<0.001mg/L	4
	EC50	72h	Algae or other aquatic plants	0.011-0.017mg/L	4
	EC50	96h	Algae or other aquatic plants	0.03-0.058mg/l	4
	LC50	96h	Fish	0.003mg/L	2
	EC50	48h	Crustacea	<0.001mg/L	4

aluminium					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	48h	Crustacea	0.736mg/L	2
	NOEC(ECx)	72h	Algae or other aquatic plants	>100mg/l	1
	EC50	72h	Algae or other aquatic plants	0.017mg/L	2
	EC50	96h	Algae or other aquatic plants	0.005mg/L	2
	LC50	96h	Fish	0.078-0.108mg/l	2

ethylene carbonate					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	48h	Crustacea	>100mg/l	2
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	100mg/l	2
	LC50	96h	Fish	>100mg/l	2

lithium fluorophosphate					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	48h	Crustacea	98mg/l	2
	EC50	72h	Algae or other aquatic plants	62mg/l	2
	EC50	96h	Algae or other aquatic plants	43mg/l	2
	NOEC(ECx)	528h	Fish	0.2mg/l	2
	LC50	96h	Fish	42mg/l	2

Legend:	***Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data***

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.
Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ethyl methyl carbonate	HIGH	HIGH
ethylene carbonate	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
ethyl methyl carbonate	LOW (LogKOW = 0.7247)
aluminium	LOW (LogKOW = 0.33)
ethylene carbonate	LOW (LogKOW = -0.3388)

Mobility in soil

Ingredient	Mobility
ethyl methyl carbonate	LOW (Log KOC = 15.22)
ethylene carbonate	LOW (Log KOC = 9.168)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Management Authority for disposal. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site.

Continued...

	▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	2Y

Land transport (ADG)

14.1. **UN number or ID number**	3480
14.2. **UN proper shipping name**	LITHIUM ION BATTERIES (including lithium ion polymer batteries)
14.3. **Transport hazard class(es)**	Class: 9 Subsidiary Hazard: Not Applicable
14.4. **Packing group**	Not Applicable
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: 188 230 310 348 376 377 384 387 Limited quantity: 0

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	3480
14.2. **UN proper shipping name**	Lithium ion batteries (including lithium ion polymer batteries)
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 9 ICAO / IATA Subsidiary Hazard: Not Applicable ERG Code: 12FZ
14.4. **Packing group**	Not Applicable
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: A88 A99 A154 A164 A183 A201 A213 A331 A334 A802 Cargo Only Packing Instructions: See 965 Cargo Only Maximum Qty / Pack: See 965 Passenger and Cargo Packing Instructions: Forbidden Passenger and Cargo Maximum Qty / Pack: Forbidden Passenger and Cargo Limited Quantity Packing Instructions: Forbidden Passenger and Cargo Limited Maximum Qty / Pack: Forbidden

Sea transport (IMDG-Code / GGVSee)

14.1. **UN number**	3480
14.2. **UN proper shipping name**	LITHIUM ION BATTERIES (including lithium ion polymer batteries)
14.3. **Transport hazard class(es)**	IMDG Class: 9 IMDG Subsidiary Hazard: Not Applicable
14.4. **Packing group**	Not Applicable
14.5 **Environmental hazard**	Marine Pollutant
14.6. **Special precautions for user**	EMS Number: F-A , S-I Special provisions: 188 230 310 348 376 377 384 387 Limited Quantities: 0

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

Continued...

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

Product name	Group
lithium iron phosphate	Not Available
graphite	Not Available
ethyl methyl carbonate	Not Available
copper	Not Available
aluminium	Not Available
ethylene carbonate	Not Available
lithium fluorophosphate	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
lithium iron phosphate	Not Available
graphite	Not Available
ethyl methyl carbonate	Not Available
copper	Not Available
aluminium	Not Available
ethylene carbonate	Not Available
lithium fluorophosphate	Not Available

SECTION 15 Regulatory information

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

lithium iron phosphate is found on the following regulatory lists

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

graphite 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)

ethyl methyl carbonate is found on the following regulatory lists

Not Applicable

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

aluminium is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

ethylene carbonate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

lithium fluorophosphate 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)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (lithium iron phosphate; ethyl methyl carbonate)
Canada - DSL	No (ethyl methyl carbonate; lithium fluorophosphate)
Canada - NDSL	No (lithium iron phosphate; graphite; copper; aluminium; ethylene carbonate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (graphite; copper; aluminium)
Korea - KECI	Yes
New Zealand - NZIoC	No (lithium iron phosphate; ethyl methyl carbonate; lithium fluorophosphate)
Philippines - PICCS	No (lithium iron phosphate)

Continued...

National Inventory	Status
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	No (lithium iron phosphate; ethyl methyl carbonate; ethylene carbonate; lithium fluorophosphate)
Vietnam - NCI	Yes
Russia - FBEPH	No (lithium iron phosphate; lithium fluorophosphate)
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	24/04/2025
Initial Date	24/04/2025

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	24/04/2025	Composition / information on ingredients - Ingredients, Identification of the substance / mixture and of the company / undertaking - Synonyms, Name

Other information

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

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

Definitions and abbreviations

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

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