

Lithium-Ion-Batteries – Metabo

Metabo Australia Pty Ltd

Chemwatch Hazard Alert Code: 4

Chemwatch: 5631-31
 Version No: 7.1
 Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

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 S.GHS.AUS.EN

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

Product Identifier

Product name	Lithium-Ion-Batteries – Metabo
Chemical Name	Not Applicable
Synonyms	LI-POWER BATTERY PACKS 18 V; LI-POWER BATTERY PACKS 12 V; LI-POWER BATTERY PACKS 36 V; LI-POWER BATTERY PACKS 14.4 V; LI-POWER PLUG-IN BATTERY PACKS; LIHD BATTERY PACKS18 V; LIHD BATTERY PACKS DS 18 V FOR FALL PROTECTIONS; LIHD BATTERY PACKS12 V; LIHD BATTERY PACKS36 V; 625026000/321001450 (Wh 36); 625596000/321000550 (Wh 36); 625027000/321001470 (Wh 72); 625028000/321001490 (Wh 94); 625406000/321001120 (Wh 24); 625453000/316046040 (Wh 54); 625529000/321000130 (Wh 187); 625590000/321000390 (Wh 58); 625595000/321000540 (Wh 29); 625438000/316045190 (Wh 24); 625585000/321000270 (Wh 48); 625367000/321001000 (Wh 72); 625368000/321001040 (Wh 99); 625369000/321000980 (Wh 144); 625549000/321001600 (Wh 180); 625349000/321001140 (Wh 48); 625344000/321000810 (Wh 223); 624989000/321001640 (Wh 72); 624990000/321001650 (Wh 99); 624991000/321001660 (Wh 180)
Proper shipping name	LITHIUM ION BATTERIES CONTAINED IN EQUIPMENT or LITHIUM ION BATTERIES PACKED WITH EQUIPMENT (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	Battery for electronic applications. 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. Use according to manufacturer's directions.
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Details of the manufacturer or supplier of the safety data sheet

Registered company name	Metabo Australia Pty Ltd
Address	10 Dalmore Drive Scoresby VIC 3179 Australia
Telephone	+61 3 9765 0199
Fax	+61 3 9765 0189
Website	www.metabo.com.au
Email	sales@metabo.com.au

Emergency telephone number

Association / Organisation	CHEMWATCH EMERGENCY RESPONSE (24/7)
Emergency telephone numbers	+61 1800 951 288
Other emergency telephone numbers	+61 3 9573 3188

Once connected and if the message is not in your preferred language then please dial 01

SECTION 2 Hazards identification

Classification of the substance or mixture

HAZARDOUS CHEMICAL. DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

Poisons Schedule	Not Applicable
Classification [1]	Acute Toxicity (Oral) Category 2, Skin Corrosion/Irritation Category 1B, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Germ Cell Mutagenicity Category 1A, Carcinogenicity Category 1B, Reproductive Toxicity Category 2, 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

Lithium-Ion-Batteries – Metabo

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

H300	Fatal if swallowed.
H314	Causes severe skin burns and eye damage.
H317	May cause an allergic skin reaction.
H340	May cause genetic defects.
H350	May cause cancer.
H361d	Suspected of damaging the unborn child.
H373	May cause damage to organs through prolonged or repeated exposure.
H411	Toxic to aquatic life with long lasting effects.
AUH019	May form explosive peroxides.

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.
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.
P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting. If more than 15 mins from Doctor, INDUCE VOMITING (if conscious).
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].
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.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P363	Wash contaminated clothing before reuse.
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.
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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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SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available		sealed metal can, containing
177997-13-6	20-50	<u>lithium nickel cobalt aluminium oxide</u>
7782-42-5	10-30	<u>graphite</u>
96-49-1	5-20	<u>ethylene carbonate</u>
108-32-7	5-20	<u>propylene carbonate</u>
105-58-8	5-20	<u>diethyl carbonate</u>
623-53-0	5-20	<u>ethyl methyl carbonate</u>
616-38-6	5-20	<u>dimethyl carbonate</u>

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CAS No	%[weight]	Name
114435-02-8	5-20	<u>fluoroethylene carbonate</u>
75-02-5	5-20	<u>vinyl fluoride</u>
Not Available	5-20	carbonate
7440-50-8	3-15	<u>copper</u>
7429-90-5	2-10	<u>aluminium</u>
21324-40-3	0.05-5	<u>lithium fluorophosphate</u>
24937-79-9	<1	<u>vinylidene fluoride homopolymer</u>
Not Available	trace	steel
7440-02-0	trace	<u>nickel</u>
Not Available	trace	inert components

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	<p>If battery is leaking and material contacts the eye.</p> <p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ 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. <p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Seek medical attention without delay.
Skin Contact	<p>If battery is leaking and material contacts the skin</p> <p>Remove all contaminated clothing, including footwear. Wash thoroughly all affected areas with water and soap.</p> <p>Seek medical attention if swelling/redness/blistering or irritation occurs.</p>
Inhalation	<p>If battery is leaking, contents may be irritating to respiratory passages.</p> <p>Remove patient to fresh air and seek medical attention.</p>
Ingestion	<p>If poisoning occurs, contact a doctor or Poisons Information Centre.</p>

Indication of any immediate medical attention and special treatment needed

Clinical effects of lithium intoxication appear to relate to duration of exposure as well as to level.

- ▶ Lithium produces a generalised slowing of the electroencephalogram; the anion gap may increase in severe cases.
- ▶ Emesis (or lavage if the patient is obtunded or convulsing) is indicated for ingestions exceeding 40 mg (Li)/Kg.
- ▶ Overdose may delay absorption; decontamination measures may be more effective several hours after cathartics.
- ▶ Charcoal is not useful. No clinical data are available to guide the administration of catharsis.
- ▶ Haemodialysis significantly increases lithium clearance; indications for haemodialysis include patients with serum levels above 4 meq/L.
- ▶ There are no antidotes.

[Ellenhorn and Barceloux: Medical Toxicology]

- ▶ Chronic exposures to cobalt and its compounds results in the so-called "hard metal pneumoconiosis" amongst industrial workers. The lesions consist of nodular conglomerate shadows in the lungs, together with peribronchial infiltration. The disease may be reversible. The acute form of the disease resembles a hypersensitivity reaction with malaise, cough and wheezing; the chronic form progresses to cor pulmonale.
- ▶ Chronic therapeutic administration may cause goiter and reduced thyroid activity.
- ▶ An allergic dermatitis, usually confined to elbow flexures, the ankles and sides of the neck, has been described.
- ▶ Cobalt cardiomyopathy may be diagnosed early by changes in the final part of the ventricular ECG (repolarisation). In the presence of such disturbances, the changes in carbohydrate metabolism (revealed by the glucose test) are of important diagnostic value.
- ▶ Treatment generally consists of a combination of Retabolil (1 injection per week over 4 weeks) and beta-blockers (average dose 60-80 mg Obsidan/24 hr). Potassium salts and diuretics have also proved useful.

BIOLOGICAL EXPOSURE INDEX (BEI)

Determinant	Sampling time	Index	Comments
Cobalt in urine	End of shift at end of workweek	15 ug/L	B
Cobalt in blood	End of shift at end of workweek	1 ug/L	B, SQ

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

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

Following acute or short term repeated exposure to hydrofluoric acid:

- ▶ Subcutaneous injections of Calcium Gluconate may be necessary around the burnt area. Continued application of Calcium Gluconate Gel or subcutaneous Calcium Gluconate should then continue for 3-4 days at a frequency of 4-6 times per day. If a "burning" sensation recurs, apply more frequently.
- ▶ Systemic effects of extensive hydrofluoric acid burns include renal damage, hypocalcaemia and consequent cardiac arrhythmias. Monitor haematological, respiratory, renal, cardiac and electrolyte status at least daily. Tests should include FBE, blood gases, chest X-ray, creatinine and electrolytes, urine output, Ca ions, Mg ions and phosphate ions. Continuous ECG monitoring may be required.
- ▶ Where serum calcium is low, or clinical, or ECG signs of hypocalcaemia develop, infusions of calcium gluconate, or if less serious, oral Sandocal, should be given. Hydrocortisone 500 mg in a four to six hourly infusion may help.
- ▶ Antibiotics should not be given as a routine, but only when indicated.
- ▶ Eye contact pain may be excruciating and 2-3 drops of 0.05% pentocaine hydrochloride may be instilled, followed by further irrigation

BIOLOGICAL EXPOSURE INDEX - BEI

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

Continued...

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 seen 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

- ▶ Alcohol stable foam.
- ▶ Dry chemical powder.
- ▶ BCF (where regulations permit).
- ▶ Carbon dioxide.
- ▶ Water spray or fog - Large fires only.
- ▶ There is no restriction on the type of extinguisher which may be used.
- ▶ Use extinguishing media suitable for surrounding area.

Special hazards arising from the substrate or mixture

Fire Incompatibility	▶ 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	<ul style="list-style-type: none"> ▶ 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. <p>Slight hazard when exposed to heat, flame and oxidisers.</p>
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ 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. <p>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.</p> <p>Metal powders, while generally regarded as non-combustible:</p> <ul style="list-style-type: none"> ▶ 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. <p>Note:</p> <ul style="list-style-type: none"> ▶ 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. ▶ 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. <p>Combustion products include: carbon monoxide (CO) carbon dioxide (CO2) aldehydes hydrogen fluoride metal oxides other pyrolysis products typical of burning organic material.</p> <p>When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles.</p> <p>Articles and manufactured articles may constitute a fire hazard where polymers form their outer layers or where combustible packaging remains in place.</p> <p>Certain substances, found throughout their construction, may degrade or become volatile when heated to high temperatures. This may create a secondary hazard.</p> <p>WARNING: Long standing in contact with air and light may result in the formation of potentially explosive peroxides.</p>
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	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ 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 course. ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Neutralise/decontaminate residue (see Section 13 for specific agent). ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▶ If contamination of drains or waterways occurs, advise emergency services. ▶ 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. <p>Minor hazard.</p> <ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ Limit all unnecessary personal contact. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ When handling DO NOT eat, drink or smoke. ▶ Always wash hands with soap and water after handling. ▶ Avoid physical damage to containers. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
Other information	<ul style="list-style-type: none"> ▶ 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. <p>Keep out of reach of children.</p>

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Packaging as recommended by manufacturer.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Avoid strong bases. ▶ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates. ▶ Avoid reaction with oxidising agents ▶ Keep dry

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 (fume)	0.2 mg/m3	Not Available	Not Available	Not Available

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	copper	Copper, dusts & mists (as Cu)	1 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
Australia Exposure Standards	aluminium	Aluminium (metal dust)	10 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	nickel	Nickel, powder	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	nickel	Nickel, metal	1 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
graphite	6 mg/m3	330 mg/m3	2,000 mg/m3
ethylene carbonate	30 mg/m3	330 mg/m3	2,000 mg/m3
propylene carbonate	34 mg/m3	370 mg/m3	2,200 mg/m3
diethyl carbonate	12 ppm	140 ppm	810 ppm
dimethyl carbonate	11 ppm	120 ppm	700 ppm
vinyl fluoride	570 ppm	6200* ppm	37000*** ppm
copper	3 mg/m3	33 mg/m3	200 mg/m3
lithium fluorophosphate	7.5 mg/m3	83 mg/m3	500 mg/m3
nickel	4.5 mg/m3	50 mg/m3	99 mg/m3


Ingredient	Original IDLH	Revised IDLH
lithium nickel cobalt aluminium oxide	10 mg/m3	Not Available
graphite	1,250 mg/m3	Not Available
ethylene carbonate	Not Available	Not Available
propylene carbonate	Not Available	Not Available
diethyl carbonate	Not Available	Not Available
ethyl methyl carbonate	Not Available	Not Available
dimethyl carbonate	Not Available	Not Available
fluoroethylene carbonate	Not Available	Not Available
vinyl fluoride	Not Available	Not Available
copper	100 mg/m3	Not Available
aluminium	Not Available	Not Available
lithium fluorophosphate	Not Available	Not Available
vinylidene fluoride homopolymer	Not Available	Not Available
nickel	10 mg/m3	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
lithium nickel cobalt aluminium oxide	D	> 0.01 to ≤ 0.1 mg/m ³
ethylene carbonate	E	≤ 0.01 mg/m ³
propylene carbonate	E	≤ 0.1 ppm
diethyl carbonate	E	≤ 0.1 ppm
fluoroethylene carbonate	E	≤ 0.1 ppm
vinyl fluoride	C	> 1 to ≤ 10 parts per million (ppm)
lithium fluorophosphate	E	≤ 0.01 mg/m ³

Notes: Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.

Exposure controls

Appropriate engineering controls	General exhaust is adequate under normal operating conditions.
Individual protection measures, such as personal protective equipment	

Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].
Skin protection	See Hand protection below
Hands/feet protection	<p>NOTE:</p> <ul style="list-style-type: none"> ▶ 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. ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber <p>None under normal operating conditions.</p> <p>OTHERWISE:</p>
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit. <p>No special equipment needed when handling small quantities otherwise use</p>

Respiratory protection

Type AX-P 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	AX-AUS P3	-	AX-PAPR-AUS / Class 1 P3
up to 50 x ES	-	AX-AUS / Class 1 P3	-
up to 100 x ES	-	AX-2 P3	AX-PAPR-2 P3 ^

^ - 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)

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Li-power 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 Available	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	Immiscible	pH as a solution (1%)	Not Available

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Vapour density (Air = 1) | Not Available

VOC g/L | Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Contact with battery contents will cause irritation. A shorted lithium battery can cause thermal and chemical burns upon contact with skin. Not normally a hazard due to physical form of product.
Ingestion	Contents of a cell if opened destructively or leaking may be harmful if swallowed. Not normally a hazard due to physical form of product. 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.
Skin Contact	Contact with battery contents will cause irritation. A shorted lithium battery can cause thermal and chemical burns upon contact with skin. Not normally a hazard due to physical form of product.
Eye	Contact with battery contents will cause irritation. Not normally a hazard due to physical form of product.
Chronic	The chemicals in this product are contained in a sealed can and exposure does not occur during normal handling and use. Overexposure can cause symptoms of non-fibrotic lung injury and membrane irritation. [Manufacturer] Lithium compounds can affect the nervous system and muscle. This can cause tremor, inco-ordination, spastic jerks and very brisk reflexes. Inhalation of cobalt powder can induce asthma, chest tightness and chronic inflammation of the bronchi. Chronic exposure to cobalt causes increase in blood haemoglobin, increased production of cells in the blood marrow and thyroid gland, discharge from around the heart and damage to the alpha cells of the pancreas. Not normally a hazard due to physical form of product. 55r33?55r43?R62?

	TOXICITY	IRRITATION
Lithium-Ion-Batteries – Metabo	Not Available	Not Available
lithium nickel cobalt aluminium oxide	dermal (rat) LD50: >2000 mg/kg ^[1]	Not Available
	Inhalation(Rat) LC50: 0.15 mg/l4h ^[1]	
	Oral (Rat) LD50: >2000 mg/kg ^[1]	
graphite	Inhalation(Rat) LC50: >2 mg/L4h ^[1]	Not Available
	Oral (Rat) LD50: >200 mg/kg ^[1]	
ethylene carbonate	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 20 mg - mild [CCInfo]*
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit): 660 mg - moderate Skin: no adverse effect observed (not irritating) ^[1]
propylene carbonate	Dermal (rabbit) LD50: >=2000 mg/kg ^[1]	Eye (rabbit): 60 mg - moderate
	Oral (Rat) LD50: >5000 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]
		Skin (human): 100 mg/3d-I moderate Skin (rabbit): 500 mg moderate
		Skin: no adverse effect observed (not irritating) ^[1]
diethyl carbonate	Inhalation(Rat) LC50: >17.75 mg/L4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >4876 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
ethyl methyl carbonate	Inhalation(Rat) LC50: >17.6 mg/l4h ^[1]	Not Available

	Oral (Rat) LD50: >5000 mg/kg ^[1]	
dimethyl carbonate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation(Rat) LC50: >5.36 mg/l4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >5000 mg/kg ^[1]	
fluoroethylene carbonate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[2]	Not Available
	Oral (Rat) LD50: ~500 mg/kg ^[1]	
vinyl fluoride	TOXICITY	IRRITATION
	Inhalation(Rat) LC50: 849511.401 ppm4h ^[2]	Not Available
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]
lithium fluorophosphate	TOXICITY	IRRITATION
	Oral (Rat) LD50: 50-300 mg/kg ^[1]	Not Available
vinylidene fluoride homopolymer	TOXICITY	IRRITATION
	Not Available	Not Available
nickel	TOXICITY	IRRITATION
	Oral (Rat) LD50: 5000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[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

LITHIUM NICKEL COBALT ALUMINIUM OXIDE

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 (a goitre).
Goitrogens include:

- Vitexin, a flavonoid, which inhibits thyroid peroxidase, contributing to goitre
- Thiocyanate and perchlorate, which decrease iodide uptake by competitive inhibition and consequently increase release of TSH from the pituitary gland
- Lithium, which inhibits thyroid hormone release
- Certain foods, such as soy and millet (containing vitexins) and vegetables in the genus Brassica (which includes broccoli, Brussels sprouts, cabbage, cauliflower and horseradish).
- Caffeine (found in coffee, tea, cola and chocolate), which acts on thyroid function as a suppressant.

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

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 diseases. 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 sensitiser 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 haptensation 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 produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

For ethylene carbonate: Ethylene carbonate is rapidly converted to ethylene glycol, and both substances have similar toxicity in animals. In animals, chronic exposure has resulted in kidney damage. Testing has not shown ethylene carbonate to cause genetic toxicity. At sufficient doses, ethylene carbonate caused birth defects.

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

ETHYLENE CARBONATE

	<p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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).</p> <p>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.</p> <p>Reproductive effects: Animal testing showed that ethylene glycol may affect fertility, survival of fetuses and the male reproductive organs.</p> <p>Effects on development: Animal studies indicate that birth defects may occur after exposure in pregnancy; there may also be reduction in foetal weight.</p> <p>Cancer: No studies are known regarding cancer effects in humans or animal, after skin exposure to ethylene glycol.</p> <p>Genetic toxicity: No human studies available, but animal testing results are consistently negative.</p>
<p>PROPYLENE CARBONATE</p>	<p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>for propylene carbonate: Numerous adequate and reliable acute toxicity tests are available on propylene carbonate. Oral and dermal tests meet OECD and EPA test guidelines. Propylene carbonate is practically nontoxic following acute exposures; the oral LD50 is >.5000 mg/kg and the dermal LD50 is >3000 mg/kg. No further testing is recommended.</p> <p>Subchronic studies (13- 14 weeks) of propylene carbonate by inhalation (aerosol) and oral (gavage) routes were conducted in rats according to current guidelines. The oral study indicated low systemic toxicity from propylene carbonate (NOAEL = 5000 mg/kg/day). In the inhalation study, no systemic toxicity was seen at concentrations up to 1000 mg/m³; however, there was periocular irritation and swelling in a few males at 500 and 1000 mg/m³. A dermal carcinogenicity study in mice did not indicate tumorigenic potential or systemic toxicity from 2 years of exposure to propylene carbonate. No further testing is recommended.</p> <p>There is a negative Ames in vitro mutagenicity assay of propylene carbonate. A single intraperitoneal injection of 1666 mg/kg propylene carbonate did not induce an increase in micronuclei when examined after 30,48 and 72 hours. The mutagenicity battery is satisfactorily filled; no further mutagenicity testing is recommended.</p> <p>Gavage administration of propylene carbonate to pregnant rats days 6-15 of gestation resulted in systemic toxicity at doses of 3000 and 5000 mg/kg/day, including mortality (not seen in 13 week study of non-pregnant rats). The NOAEL for maternal toxicity was 1000 mg/kg/day. This indicates that pregnant rats are more susceptible to propylene carbonate than are non-pregnant rats. There were no significant differences in live litter size, average fetal weight, percentage of males, or malformed fetuses.</p> <p>No studies of the effect of propylene carbonate on reproduction are available. However, no adverse effects on testis, ovaries, or accessory sex organs were noted in rats following oral or inhalation of propylene carbonate for 13 weeks. Therefore, reproductive effects from propylene carbonate are unlikely</p>
<p>DIETHYL CARBONATE</p>	<p>Equivocal tumorigen by RTECS criteria Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).</p>
<p>FLUOROETHYLENE CARBONATE</p>	<p>A study was performed to assess the skin sensitisation potential of Monofluoroethylene carbonate in the CBA/Ca strain mouse following topical application to the dorsal surface of the ear. The test material was considered to be a sensitiser under the conditions of the test. An inverse dose response relationship was noted in the Stimulation Index results. The reason for this is unknown but could be due to decreased bioactivity of the test material with increasing concentrations in dimethyl formamide, or due to immunosuppression at higher concentrations of test material.</p> <p>Genetic toxicity: in vitro Significant increases of revertant colonies were observed in Salmonella typhimurium TA98 in the presence of metabolic activation system and Salmonella typhimurium TA 100 in the absence and presence of metabolic activation system. It is concluded that Monofluoroethylene carbonate exhibited mutagenic activity in Salmonella typhimurium TA98, TA 100 under the conditions employed for this test.</p> <p>Genetic toxicity: in vivo Monofluoroethylene carbonate was cytotoxic to bone marrow cells, but did not show any indication of chromosomal damage and/or damage to the mitotic apparatus of the bone marrow target cells in female mice, treated intraperitoneally with it is concluded that Monofluoroethylene carbonate was cytotoxic to the bone marrow cells, but did not show any indication of chromosomal damage and/or damage to the mitotic apparatus of the bone marrow target cells in female mice, treated intraperitoneally with monofluoroethylene carbonate, up to 100 mg/kg bw., up to 100 mg/kg bw. *REACH Dossier</p>
<p>VINYL FLUORIDE</p>	<p>VF is mutagenic in Salmonella typhimurium with metabolic activation. In addition, VF induces gene mutations and chromosomal aberrations in Chinese hamster ovary cells (with metabolic activation), sex-linked recessive lethal mutations in Drosophila melanogaster, and micronuclei in bone marrow cells of female mice. The biotransformation pathway for VF is thought to be similar to that of vinyl chloride, that is, cytochrome P-450 mediated oxidation to the haloethylene oxide (fluoroethylene oxide), followed by rearrangement to the haloacetaldehyde (2-fluoroacetaldehyde), which is oxidised to fluoroacetic acid. Human liver microsomes metabolise VF at a rate similar to that of rat or mouse liver microsomes. VF metabolites form covalent DNA adducts. A dose-related increase in the formation of the promutagenic adduct N2,3-ethenoguanine was detected in liver DNA of rats and mice exposed to VF by inhalation. No data are available that would suggest that mechanisms thought to account for tumour induction by VF in experimental animals would not also operate in humans. VF toxicity is mediated via epoxide formation. Oxidative metabolism of inhaled VF in the presence of Aroclor 1254 (a hepatic cytochrome P-450 inducer) resulted in enhanced toxicity. In addition, administration of trichloropropylene oxide (an inhibitor of epoxide hydrolase) also increased VF toxicity. The major metabolites of VF are expected to be fluoroethylene oxide and fluoroacetaldehyde, based on indirect evidence of metabolism similar to that of vinyl chloride (VC) and vinyl bromide (VB); fluoroacetaldehyde can be further metabolized to fluoroacetic acid. In a manner analogous to</p>

	metabolism of VC (and VB), VF may initially be oxidized by microsomal monooxygenase(s) to fluoroethylene oxide (C ₂ H ₃ FO, mol wt 122.95), Inhalation of perfluoroalkenes can cause lung injury, kidney damage, brain changes and death. Repeated exposures may alter blood pressure and the production of blood cells. The potential for causing cancer is the subject of speculation. Fluoroalkanes, in contrast, are less toxic.		
COPPER	<p>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.</p> <p>for copper and its compounds (typically copper chloride):</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>Carcinogenicity: there was insufficient information to evaluate the carcinogenic activity of copper monochloride.</p> <p>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).</p>		
NICKEL	Oral (rat) TDLo: 500 mg/kg/5D-I Inhalation (rat) TCLo: 0.1 mg/m ³ /24H/17W-C		
LITHIUM NICKEL COBALT ALUMINIUM OXIDE & FLUOROETHYLENE CARBONATE & COPPER & NICKEL	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.		
LITHIUM NICKEL COBALT ALUMINIUM OXIDE & GRAPHITE & ETHYL METHYL CARBONATE & ALUMINIUM & LITHIUM FLUOROPHOSPHATE & VINYLIDENE FLUORIDE HOMOPOLYMER	No significant acute toxicological data identified in literature search.		
GRAPHITE & ETHYLENE CARBONATE & DIETHYL 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.		
ETHYLENE CARBONATE & PROPYLENE CARBONATE	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.		
PROPYLENE CARBONATE & NICKEL	WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.		
VINYL FLUORIDE & NICKEL	Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [National Toxicology Program: U.S. Dep. of Health & Human Services 2002]		
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

Lithium-Ion-Batteries – Metabo

Toxicity

Lithium-Ion-Batteries – Metabo	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
lithium nickel cobalt aluminium oxide	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>1mg/l	2
	NOEC(ECx)	672h	Fish	>0.1<=1mg/l	2
graphite	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)	48h	Crustacea	>=100mg/l	2
	LC50	96h	Fish	>100mg/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
propylene carbonate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	>1000mg/l	1
	EC50	72h	Algae or other aquatic plants	>900mg/l	1
	NOEC(ECx)	72h	Algae or other aquatic plants	900mg/l	1
diethyl carbonate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	96h	Algae or other aquatic plants	47.6-68.8mg/l	2
	EC50	48h	Crustacea	>74.16mg/l	2
	EC50	72h	Algae or other aquatic plants	>57.29mg/l	2
	NOEC(ECx)	Not Available	Crustacea	25mg/l	2
ethyl methyl carbonate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	>100mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	62mg/l	2
	EC50	72h	Algae or other aquatic plants	>62mg/l	2
	LC50	96h	Fish	>100mg/l	2
dimethyl carbonate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	>74.16mg/l	2
	EC50	96h	Algae or other aquatic plants	166.6-211mg/l	2
	NOEC(ECx)	504h	Crustacea	25mg/l	2
	EC50	72h	Algae or other aquatic plants	>57.29mg/l	2
fluoroethylene carbonate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	8.4mg/l	Not Available
	EC50	72h	Algae or other aquatic plants	6.3mg/l	2
	NOEC(ECx)	48h	Crustacea	2.8mg/l	Not Available
vinyl fluoride	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	96h	Algae or other aquatic plants	46.7mg/l	2
	EC50(ECx)	96h	Algae or other aquatic plants	46.7mg/l	2
copper	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	0.0006-0.0017mg/l	4

Lithium-Ion-Batteries – Metabo

	EC50	96h	Algae or other aquatic plants	0.03-0.058mg/l	4
	EC50	72h	Algae or other aquatic plants	0.011-0.017mg/L	4
	NOEC(ECx)	48h	Fish	0.00009mg/l	4
	LC50	96h	Fish	0.003mg/L	2
aluminium	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	0.736mg/L	2
	EC50	96h	Algae or other aquatic plants	0.005mg/L	2
	EC50	72h	Algae or other aquatic plants	0.017mg/L	2
	NOEC(ECx)	48h	Crustacea	>100mg/l	1
	LC50	96h	Fish	0.078-0.108mg/l	2
lithium fluorophosphate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	98mg/l	2
	EC50	96h	Algae or other aquatic plants	43mg/l	2
	EC50	72h	Algae or other aquatic plants	62mg/l	2
	LC50	96h	Fish	42mg/l	2
	NOEC(ECx)	528h	Fish	0.2mg/l	2
vinylidene fluoride homopolymer	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
nickel	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	>100mg/l	1
	EC50	96h	Algae or other aquatic plants	0.174-0.311mg/l	4
	EC50	72h	Algae or other aquatic plants	0.18mg/l	1
	EC50(ECx)	72h	Algae or other aquatic plants	0.18mg/l	1
	LC50	96h	Fish	0.06mg/l	4
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				

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
ethylene carbonate	HIGH	HIGH
propylene carbonate	HIGH	HIGH
diethyl carbonate	HIGH	HIGH
ethyl methyl carbonate	HIGH	HIGH
dimethyl carbonate	HIGH	HIGH
vinyl fluoride	LOW	LOW
vinylidene fluoride homopolymer	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
ethylene carbonate	LOW (LogKOW = -0.3388)
propylene carbonate	LOW (LogKOW = -0.41)
diethyl carbonate	LOW (LogKOW = 1.21)
ethyl methyl carbonate	LOW (LogKOW = 0.7247)
dimethyl carbonate	LOW (LogKOW = 0.2336)
vinyl fluoride	LOW (LogKOW = 1.1855)
vinylidene fluoride homopolymer	LOW (LogKOW = 1.24)

Mobility in soil

Ingredient	Mobility
ethylene carbonate	LOW (KOC = 9.168)
propylene carbonate	LOW (KOC = 14.85)
diethyl carbonate	LOW (KOC = 28.08)
ethyl methyl carbonate	LOW (KOC = 15.22)
dimethyl carbonate	LOW (KOC = 8.254)

Continued...

Ingredient	Mobility
vinyl fluoride	LOW (KOC = 23.74)
vinylidene fluoride homopolymer	LOW (KOC = 35.04)



SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Management Authority for disposal. ▶ Bury residue in an authorised landfill. ▶ Recycle containers if possible, or dispose of in an authorised landfill.
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SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	2Y

Land transport (ADG)

14.1. UN number or ID number	3481	
14.2. UN proper shipping name	LITHIUM ION BATTERIES CONTAINED IN EQUIPMENT or LITHIUM ION BATTERIES PACKED WITH EQUIPMENT (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	188 230 310 348 360 376 377 384 387 390
	Limited quantity	0

Air transport (ICAO-IATA / DGR)

14.1. UN number	3481	
14.2. UN proper shipping name	Lithium ion batteries packed with equipment (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 A181 A185 A213 A802
	Cargo Only Packing Instructions	966
	Cargo Only Maximum Qty / Pack	35 kg
	Passenger and Cargo Packing Instructions	966
	Passenger and Cargo Maximum Qty / Pack	5 kg
	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	3481
14.2. UN proper shipping name	LITHIUM ION BATTERIES PACKED WITH EQUIPMENT (including lithium ion polymer batteries)

Lithium-Ion-Batteries – Metabo

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	188 230 310 348 360 376 377 384 387 390
	Limited Quantities	0

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
lithium nickel cobalt aluminium oxide	Not Available
graphite	Not Available
ethylene carbonate	Not Available
propylene carbonate	Not Available
diethyl carbonate	Not Available
ethyl methyl carbonate	Not Available
dimethyl carbonate	Not Available
fluoroethylene carbonate	Not Available
vinyl fluoride	Not Available
copper	Not Available
aluminium	Not Available
lithium fluorophosphate	Not Available
vinylidene fluoride homopolymer	Not Available
nickel	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
lithium nickel cobalt aluminium oxide	Not Available
graphite	Not Available
ethylene carbonate	Not Available
propylene carbonate	Not Available
diethyl carbonate	Not Available
ethyl methyl carbonate	Not Available
dimethyl carbonate	Not Available
fluoroethylene carbonate	Not Available
vinyl fluoride	Not Available
copper	Not Available
aluminium	Not Available
lithium fluorophosphate	Not Available
vinylidene fluoride homopolymer	Not Available
nickel	Not Available

SECTION 15 Regulatory information

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

lithium nickel cobalt aluminium oxide is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List

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 1: Carcinogenic to humans

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)

ethylene carbonate is found on the following regulatory lists

Continued...

Australian Inventory of Industrial Chemicals (AIIC)

propylene carbonate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

diethyl carbonate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

ethyl methyl carbonate is found on the following regulatory lists

Not Applicable

dimethyl carbonate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

fluoroethylene carbonate is found on the following regulatory lists

Not Applicable

vinyl fluoride is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

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

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)

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)

vinylidene fluoride homopolymer 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)

nickel is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

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 2B: Possibly carcinogenic to humans

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 nickel cobalt aluminium oxide; ethyl methyl carbonate; fluoroethylene carbonate)
Canada - DSL	No (lithium nickel cobalt aluminium oxide; ethyl methyl carbonate; fluoroethylene carbonate; vinyl fluoride; lithium fluorophosphate)
Canada - NDSL	No (graphite; ethylene carbonate; propylene carbonate; diethyl carbonate; dimethyl carbonate; copper; aluminium; vinylidene fluoride homopolymer; nickel)
China - IECSC	No (lithium nickel cobalt aluminium oxide; fluoroethylene carbonate; vinyl fluoride)
Europe - EINEC / ELINCS / NLP	No (lithium nickel cobalt aluminium oxide; vinylidene fluoride homopolymer)
Japan - ENCS	No (lithium nickel cobalt aluminium oxide; graphite; copper; aluminium; lithium fluorophosphate; nickel)
Korea - KECI	No (vinyl fluoride)
New Zealand - NZIoC	No (lithium nickel cobalt aluminium oxide; ethyl methyl carbonate; fluoroethylene carbonate; vinyl fluoride; lithium fluorophosphate)
Philippines - PICCS	No (lithium nickel cobalt aluminium oxide; fluoroethylene carbonate)
USA - TSCA	Yes
Taiwan - TCSI	No (vinyl fluoride)
Mexico - INSQ	No (lithium nickel cobalt aluminium oxide; ethylene carbonate; ethyl methyl carbonate; fluoroethylene carbonate; lithium fluorophosphate; vinylidene fluoride homopolymer)

Continued...

National Inventory	Status
Vietnam - NCI	No (vinyl fluoride)
Russia - FBEPH	No (lithium nickel cobalt aluminium oxide; 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	01/03/2024
Initial Date	06/09/2023

SDS Version Summary

Version	Date of Update	Sections Updated
6.1	21/02/2024	Toxicological information - Chronic Health, Hazards identification - Classification, Firefighting measures - Fire Fighter (extinguishing media), Firefighting measures - Fire Fighter (fire/explosion hazard), First Aid measures - First Aid (eye), Exposure controls / personal protection - Personal Protection (eye), Accidental release measures - Spills (major), Transport Information
7.1	01/03/2024	Stability and reactivity - Instability Condition

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

- ▶ 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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