Lithium-Ion Batteries – Metabo

Metabo Australia Pty Ltd

Chemwatch Hazard Alert Code: **4**

Chemwatch: **5658-22**
Version No: **2.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

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

Product Identifier

Product name	Lithium-Ion Batteries – Metabo (Lithium-Ion Batteries – Metabo UN 3480)
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 (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.

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

Poisons Schedule	Not Applicable
Classification [1]	Acute Toxicity (Oral) Category 1, 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

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

Hazard statement(s)

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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.

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
Not Available		sealed metal can, containing
177997-13-6	20-50	lithium nickel cobalt aluminium oxide
7782-42-5	10-30	graphite
96-49-1	5-20	ethylene carbonate
108-32-7	5-20	propylene carbonate
105-58-8	5-20	diethyl carbonate
623-53-0	5-20	ethyl methyl carbonate
616-38-6	5-20	dimethyl carbonate
114435-02-8	5-20	fluoroethylene carbonate
75-02-5	5-20	vinyl fluoride
Not Available	5-20	carbonate
7440-50-8	3-15	copper
7429-90-5	2-10	aluminium
21324-40-3	0.05-5	lithium fluorophosphate
24937-79-9	<1	vinylidene fluoride homopolymer

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CAS No	%[weight]	Name
Not Available	trace	steel
7440-02-0	trace	nickel
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	If battery is leaking and material contacts the eye. 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. If this product comes in contact with the eyes: ▸ 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	If battery is leaking and material contacts the skin Remove all contaminated clothing, including footwear. Wash thoroughly all affected areas with water and soap. Seek medical attention if swelling/redness/blistering or irritation occurs.
Inhalation	If battery is leaking, contents may be irritating to respiratory passages. Remove patient to fresh air and seek medical attention.
Ingestion	▸ For advice, contact a Poisons Information Centre or a doctor.

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):

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.

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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

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. ‣ 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) aldehydes hydrogen fluoride metal oxides other pyrolysis products typical of burning organic material. 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. 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. **WARNING**: Long standing in contact with air and light may result in the formation of potentially explosive peroxides.
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	‣ 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	‣ 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.

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	▸ 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. 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	▸ 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	▸ 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. Keep out of reach of children.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Packaging as recommended by manufacturer.
Storage incompatibility	▸ 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
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

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
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/m³
ethylene carbonate	E	≤ 0.01 mg/m³
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/m³
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

Exposure controls

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

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Hands/feet protection	**NOTE:** ▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. ▸ Wear chemical protective gloves, e.g. PVC. ▸ Wear safety footwear or safety gumboots, e.g. Rubber None under normal operating conditions. **OTHERWISE:**
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ P.V.C apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit. No special equipment needed when handling small quantities otherwise use

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
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

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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] Neuromuscular effects result from chronic over-exposure to lithium compounds. These may include tremor, ataxia, clonus and hyperactive reflexes. Some animal studies have shown that exposure during pregnancy may produce birth defects. Other studies with rats, rabbits and monkeys have not shown teratogenic effects. Human data are ambiguous; it is well established that lithium can cross the human placenta. Of 225 registered pregnancies in which the mothers had received lithium (as a tranquiliser) there were 25 instances of congenital malformation. Although pharmacological doses of lithium cannot be unequivocally designated as a human teratogen, lithium therapy is contraindicated in women of childbearing potential. Prolonged exposure may produce anorexia, weight loss and emaciation. The kidneys, behavioural/ central nervous system and peripheral nervous system may also show adverse effects. Various types of dermatitis (psoriasis, alopecia, cutaneous ulcers, acne, follicular papules, xerosis cutis, exfoliative) may also result from chronic skin exposure. Lithium ion can be an effective treatment for manic depression. It is thought to bind the enzyme IMPase (inositol monophosphatase) and thereby mediates its influence in producing a response to calcium-induced production of neurotransmitters and hormones thought to be responsible for the clinical picture. Lithium ions interfere with ion transport processes (involving the “sodium pump”) that relay and amplify messages carried to the cells of the brain. Mania is associated with irregular increases in protein kinase C (PKC) activity within the brain. Lithium carbonate and sodium valproate, another drug traditionally used to treat the disorder, act in the brain by inhibiting PKC's activity and help to produce other compounds that also inhibit the PKC. Taking lithium salts has risks and side effects. Extended use of lithium to treat various mental disorders has been known to lead to acquired nephrogenic diabetes insipidus. Nephrogenic diabetes insipidus (NDI), also known as renal diabetes insipidus, is a form of diabetes insipidus primarily due to pathology of the kidney. This is in contrast to central or neurogenic diabetes insipidus, which is caused by insufficient levels of antidiuretic hormone (ADH, also called vasopressin). Nephrogenic diabetes insipidus is caused by an improper response of the kidney to ADH, leading to a decrease in the ability of the kidney to concentrate the urine by removing free water. Lithium intoxication can affect the central nervous system and renal system and can be lethal In subchronic studies, rats were exposed to 3 milliequivalents Li/kg/day (equivalent to 1450 mg for a 70 kg person) but did not accumulate Li whilst on a high sodium diet. However when sodium was restricted, fatal kidney toxicity developed. Dogs survived daily dose of 50 mg LiCl/kg for 150 days to the termination of the experiment on a normal sodium intake, whereas the same dose was lethal in 12 to 18 days on a low sodium diet: 20 mg LiCl/kg/day resulted in death in 18 to 30 days. Several reports have demonstrated that lithium may impair basal ganglia activity. Lithium intoxication has been associated, severe and persistent oculogyric crises. Oculogyric crisis (OGC) is the name of a dystonic reaction to certain drugs or medical conditions characterized by a prolonged involuntary upward deviation of the eyes. The term "oculogyric" refers to the bilateral elevation of the visual gaze but several other responses are associated with the crisis. In general, available cohort studies in humans have not reported a significant increase in total mortality as a result of cobalt exposure. Several studies have noted increased mortality rates resulting from lung cancer following occupational exposure to cobalt, either as a mixture of cobalt compounds or as hard metal, a metal alloy with a tungsten carbide and cobalt matrix. Fatal cases of hard metal disease and cardiomyopathy believed to have resulted from occupational cobalt exposure have also been reported. However, in the majority of these and other reported occupational studies, co-exposure to other substances was common, and was unable to be corrected for in the analysis. The effects of chronic occupational exposure to cobalt and cobalt compounds on the respiratory system in humans are well-documented. These effects include respiratory irritation, diminished pulmonary function, wheezing, asthma, pneumonia, and fibrosis and occurred at exposure levels ranging from 0.007 to 0.893 mg cobalt/m3 (exposure from 2 to 17 years). These effects have been observed in workers employed in cobalt refineries, as well as hard metal workers, diamond polishers, and ceramic dish painters (painting with cobalt blue dye). Occupational asthma attributed to the inhalation of cobalt powder has been confirmed following bronchial challenge tests. Chest tightness and chronic bronchitis have been recorded in hard-metal workers exposed to cobalt. Cobalt is known to function as a hapten, resulting in the generation of antibodies against cobalt-protein complexes. Although the minimum exposure level associated with cobalt sensitisation has not been determined, sensitisation has been demonstrated in hard metal workers with work-related asthma who have experienced prolonged occupational exposure (>3 years) to levels ranging from 0.007 to 0.893 mg cobalt/m3. The sensitisation phenomenon includes the production of IgE and IgA antibodies to cobalt. Exposure to inhaled cobalt chloride aerosols can precipitate an asthmatic attack in sensitised individuals believed to be the result of an allergic reaction within the lungs. Allergic dermatitis of an erythematous papular type may also occur following occupational exposure. Dermatitis is a common result of dermal exposure to cobalt in humans that has been verified in a large number of studies. Using patch tests and intradermal injections, it has been demonstrated that the dermatitis is probably caused by an allergic reaction to cobalt. Contact allergy was reported in 22 of 223 (9.9%) nurses who were tested with a patch test of 1.0% cobalt chloride as well as 16 of 79 (20.3%) of examined dentists. Persons with body piercings showed an increased prevalence of allergy to cobalt, with the incidence of contact allergy being proportional to number of piercings The prevalence of sensitivity to cobalt following exposure to cobalt as a component of metal implants is low, with only 3.8% of patients developing a new sensitivity to cobalt following insertion of the implant Exposure levels associated with the development of dermatitis have not been identified. It appears that the allergic properties of cobalt result mainly from exposure to the metal itself, rather than a salt, as it has been demonstrated that daily repeated exposure to aqueous cobalt salts did not result in hand eczema in patients known to have cobalt allergy. Occupational exposure to cobalt in humans has been reported to cause several effects on the nervous system, including memory loss , nerve deafness, and a decreased visual acuity. It should be noted though, that both of the studies reporting on these findings, had small numbers of subjects, and exposure characterization was not reported. Chronic exposure to cobalt produces polycythaemia (increase in blood haemoglobin), increased production of cells of the bone marrow and thyroid gland, pericardial effusion and damage to the alpha cells of the pancreas. Chronic exposure to cobalt compounds may result in pericardial effusion, polycardial effusion, cardiac failure, vomiting, convulsions and thyroid enlargement.

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Chronic administration of cobaltous chloride has produced goiter, reduced thyroid activity and lowered synthesis rates and levels of cytochrome P-450, an enzymatic system responsible for chemical detoxification, in the liver. A toxic nephritis (kidney disease) may also develop.
Epidemic cardiomyopathy (heart disease) among heavy beer drinkers in the 1960's in Canada, the USA and Belgium has been attributed to the addition of up to 1.5 ppm of cobalt as a foam restorative and stabiliser. Other factors are probably implicated as therapeutic doses of cobalt, up to 50 mg/day (in the treatment of refractory anaemias) do not produce this effect. Inadequate protein or vitamin intake amongst heavy drinkers, or the effects of alcohol in rendering the heart more susceptible to disease may be important.
Single and repeated subcutaneous or intramuscular injection of cobalt powder and salts to rats may cause sarcoma at the injection site but evidence for carcinogenicity by any other route of exposure does not exist. A number of single cases of malignant tumours, mostly sarcomas, have been reported at the site of orthopedic implants containing cobalt.
Animals, exposed to cobalt compounds also exhibit an increase in respiration, as well as tremor and convulsion. Exposure of rats and mice to aerosols of cobalt (as cobalt sulfate) at concentrations from 0.11 to 1.14 mg cobalt/m3 for 2 years resulted in a spectrum of inflammatory, fibrotic, and proliferative lesions in the respiratory tract of male and female rats and mice. Squamous metaplasia of the larynx occurred in rats and mice at exposure concentrations of .0.11 mg cobalt/m3, with severity of the lesion increasing with increased cobalt concentration. Hyperplastic lesions of the nasal epithelium occurred in rats at concentrations of .0.11 mg cobalt/3, and in mice at concentrations of .0.38 mg cobalt/m3. Both sexes of rats had greatly increased incidences (>90% incidence) of alveolar lesions at all exposure levels, including inflammatory changes, fibrosis, and metaplasia. Similar changes were seen in mice at all exposure levels, though the changes in mice were less severe.
Cobalt metal dust inhalations by miniature swine resulted in early marked decrease in lung compliance and increases in septal collagen. After a one-week "sensitising period", followed by a 10-day lapse period, further exposures resulted in wheezing produced by hypersensitivity reactions.
Not normally a hazard due to physical form of product.
55r33?55r43?R62?

Lithium-Ion Batteries-Metabo

TOXICITY	IRRITATION
Not Available	Not Available

lithium nickel cobalt aluminium oxide

TOXICITY	IRRITATION
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

TOXICITY	IRRITATION
Inhalation(Rat) LC50: >2 mg/L4h[1]	Not Available
Oral (Rat) LD50: >200 mg/kg[1]	

ethylene carbonate

TOXICITY	IRRITATION
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

TOXICITY	IRRITATION
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

TOXICITY	IRRITATION
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

TOXICITY	IRRITATION
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

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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, 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.

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

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	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
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 a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. 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 through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol. dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO2, which is one of the major elimination products of ethylene glycol. In addition to

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	exhaled CO2, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested. **Respiratory Effects.** Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases). **Cardiovascular Effects.** Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12- 24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol. Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases.Therefore, it appears that 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.** Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition. **Musculoskeletal Effects.** Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tenderness and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia. **Hepatic Effects.** Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. **Renal Effects.** Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, anuria , and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near normal renal function can return with adequate supportive therapy. **Metabolic Effects.** One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate). **Neurological Effects:** Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion. Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months. **Reproductive Effects:** Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration. **Developmental Effects:** The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embyrotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight. **Cancer:** No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol. **Genotoxic Effects**: Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available *in vivo* and *in vitro* laboratory studies provide consistently negative genotoxicity results for ethylene glycol.
PROPYLENE CARBONATE	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. 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. 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/m"; 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. 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. 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. 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
DIETHYL CARBONATE	Equivocal tumorigen by RTECS criteria Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).

Continued...

FLUOROETHYLENE CARBONATE	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. 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. 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
VINYL FLUORIDE	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, administraton 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 metabolism of VC (and VB), VF may initially be oxidized by microsomal monooxygenase(s) to fluoroethylene oxide (C2H3FO, mol wt 122.95), The fluoroalkenes vary widely in acute inhalation toxicity. Those, such as perfluoroisobutylene, PFIB, the most highly toxic member, attacks the pulmonary epithelium of rats eventuating in edema and death after a delay of about one day. Other fluoroalkenes, such as hexafluoropropylene (HFP) or chlorotrifluoroethylene (CTFE), also cause pulmonary injury but at lower concentrations produce concentration dependent changes in the renal concentrating mechanism of the rat. Changes in the CNS of rats and rabbits have also been reported for CTFE. CTFE, in repeated exposures, has produced blood pressure changes in dogs, CNS effects and changes in the erythropoietic system. Mechanisms of action research for fluoroalkenes is an important area of need. . The nucleophilic sensitivity of the fluoroalkenes and the potential carcinogenic effects stemming are the subject of speculation. Fluoralkanes, in contrast, are amongst the least toxic of all substances.
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): **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).
NICKEL	Oral (rat) TDLo: 500 mg/kg/5D-I Inhalation (rat) TCLo: 0.1 mg/m3/24H/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.

Continued...

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.
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*

SECTION 12 Ecological information

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
LC50	96h	Fish	>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
LC50	96h	Fish	1000mg/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
LC50	96h	Fish	45.1-419.4mg/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

Continued...

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
LC50	96h	Fish	>=100mg/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
LC50	96h	Fish	6-60mg/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
LC50	96h	Fish	331.6mg/l	2

copper

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	0.0006-0.0017mg/l	4
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

Continued...

Ingredient	Persistence: Water/Soil	Persistence: Air
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)
vinyl fluoride	LOW (KOC = 23.74)
vinylidene fluoride homopolymer	LOW (KOC = 35.04)

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. ▸ Bury residue in an authorised landfill. ▸ 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)

Continued...

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

14.7.1. 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

Continued...

Product name	Ship Type
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

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)

Continued...

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)
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	01/03/2024

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	01/03/2024	Hazards identification - Classification, 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

Continued...

- 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

end of SDS