



AP300S

Stihl Pty Ltd.

Chemwatch: 5688-79

Version No: 2.1

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

Safety Data Sheet according to the Health and Safety at Work (Hazardous Substances) Regulations 2017

Issue Date: 18/07/2024

Print Date: 25/07/2024

L.GHS.AUS/NZ.EN.E

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

Product Identifier

Product name	AP300S
Chemical Name	Not Applicable
Synonyms	4850-400-6580
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. 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 involves discharge then regenerative charging cycle from external DC power source. CHARGING HAZARD. Completion of charging process includes evolution of highly flammable and explosive hydrogen gas which is readily detonated by electric spark. No smoking or naked lights. Do not attach/detach metal clips or operate open switches during charging process because of arcing/sparking hazard. Overcharging to excess results in vigorous hydrogen evolution - boiling - which may cause generation of corrosive acid mist.
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Details of the manufacturer or supplier of the safety data sheet

Registered company name	Stihl Pty Ltd.
Address	5 Kingston Park Court, Knoxfield, Victoria, 3180, Australia 9 Bishop Browne Place, East Tamaki, Auckland, 1730 New Zealand
Telephone	AU: +61 3 9215 6666 NZ: +64 9262 4000
Fax	Not Available
Website	Not Available
Email	enquiries@stihl.com.au

Emergency telephone number

Association / Organisation	Poisons Information Centre
Emergency telephone numbers	131 126 (AU)
Other emergency telephone numbers	0800 764 766 (NZ)

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Poisons Schedule	Not Applicable
Classification ^[1]	Acute Toxicity (Oral) Category 3, Acute Toxicity (Dermal) Category 4, Skin Corrosion/Irritation Category 1B, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Carcinogenicity Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
Signal word	Danger

Hazard statement(s)

H301	Toxic if swallowed.
H312	Harmful in contact with skin.
H314	Causes severe skin burns and eye damage.
H317	May cause an allergic skin reaction.
H336	May cause drowsiness or dizziness.
H351	Suspected of causing cancer.
H372	Causes damage to organs through prolonged or repeated exposure.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
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.
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.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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Considered a Hazardous Substance according to the criteria of the New Zealand Hazardous Substances New Organisms legislation. Classified as Dangerous Goods for transport purposes.

Classification ^[1]	Acute Toxicity (Oral) Category 3, Acute Toxicity (Dermal) Category 4, Skin Corrosion/Irritation Category 1B, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Carcinogenicity Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI
Determined by Chemwatch using GHS/HSNO criteria	6.1C (oral), 6.1D (dermal), 8.2B, 8.3A, 6.5B (contact), 6.7B, 6.9A, 6.9B (narcotic effects), 9.1C

Label elements

Hazard pictogram(s)	
Signal word	Danger

Hazard statement(s)

H301	Toxic if swallowed.
H312	Harmful in contact with skin.
H314	Causes severe skin burns and eye damage.
H317	May cause an allergic skin reaction.
H336	May cause drowsiness or dizziness.
H351	Suspected of causing cancer.
H372	Causes damage to organs through prolonged or repeated exposure.
H412	Harmful to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

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

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting. If more than 15 mins from Doctor, INDUCE VOMITING (if conscious).
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SECTION 3 Composition / information on ingredients**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available		hermetically sealed metal case with
207803-51-8	<25	<u>lithium nickel cobalt aluminium oxide</u>
7782-42-5	<20	<u>graphite</u>
Not Available		electrolyte components
Not Available		containing
21324-40-3	<20	<u>lithium fluorophosphate</u>
141-78-6	<20	<u>ethyl acetate</u>
96-49-1	<20	<u>ethylene carbonate</u>
616-38-6	<20	<u>dimethyl carbonate</u>

Legend: 1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available

SECTION 4 First aid measures**Description of first aid measures**

Eye Contact	<ul style="list-style-type: none"> ▶ Generally not applicable. If this product comes in contact with eyes: <ul style="list-style-type: none"> ▶ Wash out immediately with water. ▶ If irritation continues, seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<ul style="list-style-type: none"> ▶ Generally not applicable. If skin or hair contact occurs: <ul style="list-style-type: none"> ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ Generally not applicable. ▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▶ Other measures are usually unnecessary.
Ingestion	<ul style="list-style-type: none"> ▶ Generally not applicable. ▶ Not considered a normal route of entry. ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully.

Continued...

- ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.
- ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.
- ▶ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Firefighting measures**Extinguishing media**

- ▶ Dry chemical powder.
- ▶ BCF (where regulations permit).
- ▶ Carbon dioxide.

Special hazards arising from the substrate or mixture

Fire Incompatibility	<p>None known.</p> <ul style="list-style-type: none"> ▶ Keep dry ▶ NOTE: May develop pressure in containers; open carefully. Vent periodically.
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Advice for firefighters

Fire Fighting	Slight hazard when exposed to heat, flame and oxidisers.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Non combustible. ▶ Not considered to be a significant fire risk. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ May emit acrid smoke. May emit corrosive and poisonous fumes. <p>Articles and manufactured articles may constitute a fire hazard where polymers form their outer layers or where combustible packaging remains in place.</p> <p>Certain substances, found throughout their construction, may degrade or become volatile when heated to high temperatures. This may create a secondary hazard.</p>

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	<p>Clean up all spills immediately.</p> <p>Avoid contact with skin and eyes.</p> <p>Place in suitable containers for disposal.</p>
Major Spills	<ul style="list-style-type: none"> ▶ 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.

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

SECTION 7 Handling and storage**Precautions for safe handling**

Safe handling	<p>Do not connect the positive terminal to the negative terminal with electrical wire or chain. Avoid polarity reverse connection when installing the battery to an instrument. Do not wet the battery with water, seawater or acid; or expose to strong oxidizer. Do not damage or remove the external tube. Keep the battery away from heat and fire. Do not disassemble or reconstruct the battery; or solder the battery directly. Do not give a mechanical shock or deform. Do not use unauthorized charger or other charging method. This battery is manufactured in a charged state. It is NOT designed for recharging. Recharging can cause battery leakage or in some cases, high pressure rupture. Inadvertent charging can occur if a battery is installed backwards.</p> <p>Use good occupational work practice. Observe manufacturer's storage and handling recommendations contained within this SDS.</p> <p>Avoid physical damage to containers.</p>
Other information	<ul style="list-style-type: none"> ▶ Keep dry. ▶ Store under cover. ▶ Protect containers against physical damage. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. <p>Keep out of reach of children.</p> <p>Store out of direct sunlight</p> <ul style="list-style-type: none"> ▶ Store away from incompatible materials.

Conditions for safe storage, including any incompatibilities

Suitable container	<p>Generally packaging as originally supplied with the article or manufactured item is sufficient to protect against physical hazards.</p> <p>If repackaging is required ensure the article is intact and does not show signs of wear. As far as is practicably possible, reuse the original packaging or something providing a similar level of protection to both the article and the handler.</p>
Storage incompatibility	<p>Avoid contamination of water, foodstuffs, feed or seed.</p> <ul style="list-style-type: none"> ▶ Keep dry

► **NOTE:** May develop pressure in containers; open carefully. Vent periodically.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
New Zealand Workplace Exposure Standards (WES)	lithium nickel cobalt aluminium oxide	Respirable dust (not otherwise classified)	3 mg/m ³	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	lithium nickel cobalt aluminium oxide	Inhalable dust (not otherwise classified)	10 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	graphite	Graphite (all forms except fibres) (respirable dust) (natural & synthetic)	3 mg/m ³	Not Available	Not Available	(e) Containing no asbestos and < 1% crystalline silica.
New Zealand Workplace Exposure Standards (WES)	graphite	Graphite, all forms except graphite fibres respirable dust	3 mg/m ³	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	lithium fluorophosphate	Inhalable dust (not otherwise classified)	10 mg/m ³	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	lithium fluorophosphate	Respirable dust (not otherwise classified)	3 mg/m ³	Not Available	Not Available	Not Available
Australia Exposure Standards	ethyl acetate	Ethyl acetate	200 ppm / 720 mg/m ³	1440 mg/m ³ / 400 ppm	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	ethyl acetate	Ethyl acetate	200 ppm / 720 mg/m ³	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
graphite	6 mg/m ³	330 mg/m ³	2,000 mg/m ³
lithium fluorophosphate	7.5 mg/m ³	83 mg/m ³	500 mg/m ³
ethyl acetate	1,200 ppm	1,700 ppm	10000** ppm
ethylene carbonate	30 mg/m ³	330 mg/m ³	2,000 mg/m ³
dimethyl carbonate	11 ppm	120 ppm	700 ppm


Ingredient	Original IDLH	Revised IDLH
lithium nickel cobalt aluminium oxide	10 mg/m ³	Not Available
graphite	1,250 mg/m ³	Not Available
lithium fluorophosphate	Not Available	Not Available
ethyl acetate	Not Available	Not Available
ethylene carbonate	Not Available	Not Available
dimethyl carbonate	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
ethylene carbonate	E	≤ 0.01 mg/m ³
Notes:	<i>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.</i>	

MATERIAL DATA

Exposure controls

Appropriate engineering controls	General exhaust is adequate under normal operating conditions. Articles or manufactured items, in their original condition, generally don't require engineering controls during handling or in normal use. Exceptions may arise following extensive use and subsequent wear, during recycling or disposal operations where substances, found in the article, may be released to the environment.
Individual protection measures, such as personal protective equipment	
Eye and face protection	None under normal operating conditions. OTHERWISE: ► Safety glasses.
Skin protection	See Hand protection below
Hands/feet protection	None under normal operating conditions. OTHERWISE: ► Rubber Gloves
Body protection	See Other protection below
Other protection	No special equipment needed when handling small quantities

Recommended material(s)**GLOVE SELECTION INDEX**

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:

AP300S

Material	CPI
PE/EVAL/PE	A
PVA	A
SARANEX-23 2-PLY	A
BUTYL	B
TEFLON	B
VITON/CHLOROBUTYL	B
BUTYL/NEOPRENE	C
CPE	C
HYPALON	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PVC	C
SARANEX-23	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Ansell Glove Selection

Glove — In order of recommendation
AlphaTec 02-100
AlphaTec® 38-612
AlphaTec® 53-001
AlphaTec® 58-005
MICROFLEX® MidKnight® XTRA 93-862
MICROFLEX® LifeStar EC™ 93-868
AlphaTec® Solvex® 37-175
BioClean™ Emerald BENS
BioClean™ Extra BLAS
BioClean™ Fusion (Sterile) S-BFAP

The suggested gloves for use should be confirmed with the glove supplier.

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

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

^ - Full-face

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

Respiratory protection not normally required due to the physical form of the product.

SECTION 9 Physical and chemical properties**Information on basic physical and chemical properties**

Appearance	Solid.		
Physical state	Manufactured	Relative density (Water = 1)	Not Available
Odour	No Odour	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Applicable	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Applicable
Initial boiling point and boiling range (°C)	Not Applicable	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Applicable	Explosive properties	Not Available

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

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Vapors or fumes may cause respiratory tract irritation. Not normally a hazard due to physical form of product.
Ingestion	Considered an unlikely route of entry in commercial/industrial environments Ingestion may result in nausea, abdominal irritation, pain and vomiting
Skin Contact	The electrolyte causes severe skin burns and irritation. Not normally a hazard due to physical form of product.
Eye	The electrolyte causes eye irritation and damage. Not normally a hazard due to physical form of product.
Chronic	The chemicals in this product are contained in a sealed case and exposure does not occur during normal handling and use. Not normally a hazard due to physical form of product.

	TOXICITY	IRRITATION
	AP300S	Not Available
lithium nickel cobalt aluminium oxide	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
	Inhalation (Rat) LC50: 0.15 mg/l4h ^[1]	Skin: adverse effect observed (corrosive) ^[1]
	Oral (Rat) LD50: >2000 mg/kg ^[1]	
graphite	Inhalation (Rat) LC50: >2 mg/L4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >200 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
lithium fluorophosphate	Oral (Rat) LD50: 50-300 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
		Skin: adverse effect observed (corrosive) ^[1]
ethyl acetate	Dermal (rabbit) LD50: >18000 mg/kg ^[2]	Eye (human): 400 ppm
	Inhalation (Mouse) LC50: >18 mg/l4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Mouse) LD50: 4100 mg/kg ^[2]	Skin: no adverse effect observed (not irritating) ^[1]
ethylene carbonate	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 20 mg - mild [CCInfo]*
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit): 660 mg - moderate
		Skin: no adverse effect observed (not irritating) ^[1]
dimethyl carbonate	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]	

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

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.

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 Laboratory Practice (GLP). Aluminium citrate was selected for the study since it is the most soluble and bioavailable aluminium salt. Pregnant rats were exposed to aluminium citrate from gestational day 6 through lactation, and then the offspring were exposed post-weaning until postnatal day 364. An extensive functional observational battery of tests was performed at various times.

Evidence of aluminium toxicity was demonstrated in the high (300 mg/kg bw/day of aluminium) and to a lesser extent, the mid-dose groups (100 mg/kg bw/day of aluminium). In the high-dose group, the main effect was renal damage, resulting in high mortality in the male offspring. No major neurological pathology or neurobehavioural effects were observed, other than in the neuromuscular subdomain (reduced grip strength and increased foot splay). Thus, the lowest observed adverse effect level (LOAEL) was 100 mg/kg bw/day and the no observed adverse effect level (NOAEL) was 30 mg/kg bw/day. Bioavailability of aluminium chloride, sulfate and nitrate and aluminium hydroxide was much lower than that of aluminium citrate This study was used by JECFA as key study to derive the PTWI.

Genotoxicity

Aluminium compounds were non-mutagenic in bacterial and mammalian cell systems, but some produced DNA damage and effects on chromosome integrity and segregation in vitro. Clastogenic effects were also observed in vivo when aluminium sulfate was administered at high doses by gavage or by the intraperitoneal route. Several indirect mechanisms have been proposed to explain the variety of genotoxic effects elicited by aluminium salts in experimental systems. Cross-linking of DNA with chromosomal proteins, interaction with microtubule assembly and mitotic spindle functioning, induction of oxidative damage, damage of lysosomal membranes with liberation of DNAase, have been suggested to explain the induction of structural chromosomal aberrations, sister chromatid exchanges, chromosome loss and formation of oxidized bases in experimental systems. The EFSA Panel noted that these indirect mechanisms of genotoxicity, occurring at relatively high levels of exposure, are unlikely to be of relevance for humans exposed to aluminium via the diet. Aluminium compounds do not cause gene mutations in either bacteria or mammalian cells. Exposure to aluminium compounds does result in both structural and numerical chromosome aberrations both in in-vitro and in-vivo mutagenicity tests. DNA damage is probably the result of indirect mechanisms. The DNA damage was observed only at high exposure levels.

Carcinogenicity.

The available epidemiological studies provide limited evidence that certain exposures in the aluminium production industry are carcinogenic to humans, giving rise to cancer of the lung and bladder. However, the aluminium exposure was confounded by exposure to other agents including polycyclic aromatic hydrocarbons, aromatic amines, nitro compounds and asbestos. There is no evidence of increased cancer risk in non-occupationally exposed persons.

Neurodegenerative diseases.

Following the observation that high levels of aluminium in dialysis fluid could cause a form of dementia in dialysis patients, a number of studies were carried out to determine if aluminium could cause dementia or cognitive impairment as a consequence of environmental exposure over long periods. Aluminium was identified, along with other elements, in the amyloid plaques that are one of the diagnostic lesions in the brain for Alzheimer disease, a common form of senile and pre-senile dementia. Some of the epidemiology studies suggest the possibility of an association of Alzheimer disease with aluminium in water, but other studies do not confirm this association. All studies lack information on ingestion of aluminium from food and how concentrations of aluminium in food affect the association between aluminium in water and Alzheimer disease." There are suggestions that persons with some genetic variants may absorb more aluminium than others, but there is a need for more analytical research to determine whether aluminium from various sources has a significant causal association with Alzheimer disease and other neurodegenerative diseases. Aluminium is a neurotoxicant in experimental animals. However, most of the animal studies performed have several limitations and therefore cannot be used for quantitative risk assessment.

Contact sensitivity:

It has been suggested that the body burden of aluminium may be linked to different diseases. Macrophagic myofasciitis and chronic fatigue syndrome can be caused by aluminium-containing adjuvants in vaccines. Macrophagic myofasciitis (MMF) has been described as a disease in adults presenting with ascending myalgia and severe fatigue following exposure to aluminium hydroxide-containing vaccines. The corresponding histological findings include aluminium-containing macrophages infiltrating muscle tissue at the injection site. The hypothesis is that the long-lasting granuloma triggers the development of the systemic syndrome.

Aluminium acts not only as an adjuvant, stimulating the immune system either to fend off infections or to tolerate antigens, it also acts as a sensitiser causing contact allergy and allergic contact dermatitis. In general, metal allergies are very common and aluminium is considered to be a weak allergen. A metal must be ionised to be able to act as a contact allergen, then it has to undergo haptensation to be immunogenic and to initiate an immune response. Once inside the skin, the metal ions must bind to proteins to become immunologically reactive. The most important routes of exposure and sensitisation to aluminium are through aluminium-containing vaccines. One Swedish study showed a statistically significant association between contact allergy to aluminium and persistent itching nodules in children treated with allergen-specific immunotherapy (ASIT). Nodules were overrepresented in patients with contact allergy to aluminium.

Other routes of sensitisation reported in the literature are the prolonged use of aluminium-containing antiperspirants, topical medication, and tattooing of the skin with aluminium-containing pigments. Most of the patients experienced eczematous reactions whereas tattooing caused granulomas. Even though aluminium is used extensively in industry, only a low number of cases of occupational skin sensitisation to aluminium have been reported. Systemic allergic contact dermatitis in the form of flare-up reactions after re-exposure to aluminium has been documented: pruritic nodules at present and previous injection sites, eczema at the site of vaccination as well as at typically atopic localisations after vaccination with aluminium-containing vaccines and/or patch testing with aluminium, and also after use of aluminium-containing toothpaste.

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 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.

	<p>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.</p> <p>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.</p> <p>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.</p> <p>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).</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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 embryotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight.</p> <p>Cancer: No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol.</p> <p>Genotoxic Effects: Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available <i>in vivo</i> and <i>in vitro</i> laboratory studies provide consistently negative genotoxicity results for ethylene glycol.</p>
LITHIUM NICKEL COBALT ALUMINIUM OXIDE & GRAPHITE & LITHIUM FLUOROPHOSPHATE	No significant acute toxicological data identified in literature search.
GRAPHITE & LITHIUM FLUOROPHOSPHATE & ETHYL ACETATE & ETHYLENE CARBONATE	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.

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

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

SECTION 12 Ecological information

Toxicity					
	Endpoint	Test Duration (hr)	Species	Value	Source
	AP300S	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	72h	Algae or other aquatic plants	>100mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	LC50	96h	Fish	>100mg/l	2
	NOEC(ECx)	96h	Fish	>=100mg/l	2
lithium fluorophosphate	Endpoint	Test Duration (hr)	Species	Value	Source

Continued...

	LC50	96h	Fish	42mg/l	2
	EC50	72h	Algae or other aquatic plants	62mg/l	2
	EC50	48h	Crustacea	98mg/l	2
	NOEC(ECx)	528h	Fish	0.2mg/l	2
	EC50	96h	Algae or other aquatic plants	43mg/l	2
ethyl acetate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	1800-3200mg/L	4
	EC50	48h	Crustacea	164mg/l	1
	LC50	96h	Fish	>75.6mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	>100mg/l	1
ethylene carbonate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	LC50	96h	Fish	>100mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	100mg/l	2
dimethyl carbonate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>57.29mg/l	2
	EC50	48h	Crustacea	>74.16mg/l	2
	NOEC(ECx)	504h	Crustacea	25mg/l	2
	LC50	96h	Fish	>=100mg/l	2
	EC50	96h	Algae or other aquatic plants	166.6-211mg/l	2
Legend:	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data				

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ethyl acetate	LOW (Half-life = 14 days)	LOW (Half-life = 14.71 days)
ethylene carbonate	HIGH	HIGH
dimethyl carbonate	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
ethyl acetate	HIGH (BCF = 3300)
ethylene carbonate	LOW (LogKOW = -0.3388)
dimethyl carbonate	LOW (LogKOW = 0.2336)

Mobility in soil

Ingredient	Mobility
ethyl acetate	LOW (Log KOC = 6.131)
ethylene carbonate	LOW (Log KOC = 9.168)
dimethyl carbonate	LOW (Log KOC = 8.254)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Management Authority for disposal.
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Ensure that the hazardous substance is disposed in accordance with the Hazardous Substances (Disposal) Notice 2017

Disposal Requirements

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

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

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

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

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	NO
HAZCHEM	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	Not Applicable	
14.6. Special precautions for user	Special provisions	188 230 310 348 376 377 384 387
	Limited quantity	0

Land transport (UN)

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	Not Applicable	
14.6. Special precautions for user	Special provisions	188; 230; 310; 348; 376; 377; 384; 387
	Limited quantity	0

Air transport (ICAO-IATA / DGR)

14.1. UN number	3480	
14.2. UN proper shipping name	Lithium ion batteries (including lithium ion polymer batteries)	
14.3. Transport hazard class(es)	ICAO/IATA Class	9
	ICAO / IATA Subsidiary Hazard	Not Applicable
	ERG Code	12FZ
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
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	Not Applicable	
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
lithium fluorophosphate	Not Available
ethyl acetate	Not Available
ethylene carbonate	Not Available
dimethyl carbonate	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
lithium fluorophosphate	Not Available
ethyl acetate	Not Available
ethylene carbonate	Not Available
dimethyl carbonate	Not Available

SECTION 15 Regulatory information**Safety, health and environmental regulations / legislation specific for the substance or mixture**

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

HSR Number	Group Standard
HSR002627	N.O.S. Acutely Toxic Corrosive Carcinogenic Group Standard 2020
HSR100425	Pharmaceutical Active Ingredients Group Standard 2020
HSR100757	Veterinary Medicines Limited Pack Size Finished Dose Group Standard 2020
HSR100758	Veterinary Medicines Non dispersive Closed System Application Group Standard 2020

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

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)
 New Zealand Workplace Exposure Standards (WES)

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)
 New Zealand Inventory of Chemicals (NZIoC)
 New Zealand Workplace Exposure Standards (WES)

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)
 New Zealand Workplace Exposure Standards (WES)

ethyl acetate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
 Australian Inventory of Industrial Chemicals (AIIC)
 New Zealand Approved Hazardous Substances with controls
 New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
 New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
 New Zealand Inventory of Chemicals (NZIoC)
 New Zealand Workplace Exposure Standards (WES)

ethylene carbonate is found on the following regulatory lists

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

dimethyl carbonate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)
 New Zealand Approved Hazardous Substances with controls
 New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
 New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
 New Zealand Inventory of Chemicals (NZIoC)

Additional Regulatory Information

Not Applicable

Hazardous Substance Location

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

Hazard Class	Quantity (Compliance Certificate)	Quantity (Compliance Certificate - Farms >4 ha)
6.1C	1000 kg or 1000 L	3500 kg or 3500 L
8.2B	250 kg or 250 L	3500 kg or 3500 L

Certified Handler

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

Class of substance	Quantities
Not Applicable	Not Applicable

Refer Group Standards for further information

Maximum quantities of certain hazardous substances permitted on passenger service vehicles

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

Hazard Class	Gas (aggregate water capacity in mL)	Liquid (L)	Solid (kg)	Maximum quantity per package for each classification
6.1C	120	1	3	
6.5A or 6.5B	120	1	3	
8.2B	120	1	3	

Tracking Requirements

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (lithium nickel cobalt aluminium oxide)
Canada - DSL	No (lithium nickel cobalt aluminium oxide; lithium fluorophosphate)
Canada - NDSL	No (graphite; ethyl acetate; ethylene carbonate; dimethyl carbonate)
China - IECSC	No (lithium nickel cobalt aluminium oxide)
Europe - EINEC / ELINCS / NLP	No (lithium nickel cobalt aluminium oxide)
Japan - ENCS	No (lithium nickel cobalt aluminium oxide; graphite; lithium fluorophosphate)
Korea - KECI	Yes
New Zealand - NZIoC	No (lithium nickel cobalt aluminium oxide; lithium fluorophosphate)
Philippines - PICCS	No (lithium nickel cobalt aluminium oxide)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (lithium nickel cobalt aluminium oxide; lithium fluorophosphate; ethylene carbonate)
Vietnam - NCI	Yes
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	18/07/2024
Initial Date	18/07/2024

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,

Continued...

- ▶ 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

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