Lithium-ion Battery Pack

Einhell Australia Pty Ltd

Chemwatch Hazard Alert Code: **3**

Chemwatch: **5545-19**
Version No: **2.2**
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 Battery Pack
Chemical Name	Not Applicable
Synonyms	OLBP-0185, XLBP-0185, PXBP-250, XCSBP-0185, PXBP-25B
Proper shipping name	LITHIUM ION BATTERIES CONTAINED IN EQUIPMENT or LITHIUM ION BATTERIES PACKED WITH EQUIPMENT
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	18V XU1 and Ozito Power exchange Tools NOTE: Hazard statement relates to battery contents. Potential for exposure should not exist unless the battery leaks, is exposed to high temperatures or is mechanically, physically or electrically abused. Risk of exposure exists only in case of mechanical, electrical or thermal abuse. Thus the batteries should not short circuit, recharge, puncture, incinerate, crush, immerse in water, force discharge, or expose to temperatures above the temperature range of the cell or battery. Use according to manufacturer's directions.

Details of the supplier of the safety data sheet

Registered company name	**Einhell Australia Pty Ltd**
Address	25 Fox Drive Dandenong South VIC 3175 Australia
Telephone	1800 069 486
Fax	Not Available
Website	www.ozito.com.au
Email	Not Available

Emergency telephone number

Association / Organisation	**Poison Information Centre (Australia)**
Emergency telephone numbers	13 11 26 (24 hours)
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	S6
Classification [1]	Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Sensitisation (Respiratory) Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Carcinogenicity Category 1A, 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)

H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H318	Causes serious eye damage.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H335	May cause respiratory irritation.
H350	May cause cancer.

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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.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P284	[In case of inadequate ventilation] wear respiratory protection.
P270	Do not eat, drink or smoke when using this product.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

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.

Not Applicable

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
12031-65-1	10-30	lithium nickel oxide
12190-79-3	1-10	lithium cobaltate
12057-17-9	1-10	lithium manganate
7782-42-5	10-30	graphite
21324-40-3	1-10	lithium fluorophosphate
96-49-1	1-10	ethylene carbonate
623-53-0	1-10	ethyl methyl carbonate
108-32-7	1-10	propylene carbonate
7440-02-0	1-10	nickel
7429-90-5	10-30	aluminium
7440-50-8	1-10	copper
7440-44-0	1-10	carbon, non-activated
24937-79-9	1-10	vinylidene fluoride homopolymer
9002-88-4	1-10	polyethylene
25038-59-9	1-10	polyethylene terephthalate
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available*	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.

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	▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. ▸ Generally not applicable.
Skin Contact	If skin or hair contact occurs: ▸ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▸ Quickly remove all contaminated clothing, including footwear. ▸ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▸ Transport to hospital, or doctor. ▸ Generally not applicable.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay. ▸ Generally not applicable.
Ingestion	▸ **IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY.** ▸ For advice, contact a Poisons Information Centre or a doctor. ▸ Urgent hospital treatment is likely to be needed. ▸ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. ▸ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist. ▸ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS. **Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:** ▸ **INDUCE** vomiting with fingers down the back of the throat, **ONLY IF CONSCIOUS**. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. **NOTE:** Wear a protective glove when inducing vomiting by mechanical means. ▸ Generally not applicable. ▸ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Firefighting measures

Extinguishing media

- **DO NOT** use halogenated fire extinguishing agents.
- Alcohol stable foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.
- Water spray or fog - Large fires only.

Special hazards arising from the substrate or mixture

Fire Incompatibility	▸ Reacts with acids producing flammable / explosive hydrogen (H2) gas ▸ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

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	Combustible. Will burn if ignited. Combustion products include: carbon monoxide (CO) carbon dioxide (CO2) hydrogen fluoride nitrogen oxides (NOx) phosphorus oxides (POx) metal oxides other pyrolysis products typical of burning organic material. Articles and manufactured articles may constitute a fire hazard where polymers form their outer layers or where combustible packaging remains in place. Certain substances, found throughout their construction, may degrade or become volatile when heated to high temperatures. This may create a secondary hazard.
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SECTION 6 Accidental release measures

Continued...

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

Conditions for safe storage, including any incompatibilities

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

+	x	+	x	+	+	+

X — *Must not be stored together*
0 — *May be stored together with specific preventions*
+ — *May be stored together*

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

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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	lithium manganate	Manganese, dust & compounds (as Mn)	1 mg/m3	Not Available	Not Available	Not Available
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	nickel	Nickel, metal	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	nickel	Nickel, powder	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	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	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

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
graphite	6 mg/m3	330 mg/m3	2,000 mg/m3
lithium fluorophosphate	7.5 mg/m3	83 mg/m3	500 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
nickel	4.5 mg/m3	50 mg/m3	99 mg/m3
copper	3 mg/m3	33 mg/m3	200 mg/m3
carbon, non-activated	6 mg/m3	330 mg/m3	2,000 mg/m3
polyethylene	16 mg/m3	170 mg/m3	1,000 mg/m3

Ingredient	Original IDLH	Revised IDLH
lithium nickel oxide	10 mg/m3	Not Available
lithium cobaltate	Not Available	Not Available
lithium manganate	500 mg/m3	Not Available
graphite	1,250 mg/m3	Not Available
lithium fluorophosphate	Not Available	Not Available
ethylene carbonate	Not Available	Not Available
ethyl methyl carbonate	Not Available	Not Available
propylene carbonate	Not Available	Not Available
nickel	10 mg/m3	Not Available
aluminium	Not Available	Not Available
copper	100 mg/m3	Not Available
carbon, non-activated	Not Available	Not Available
vinylidene fluoride homopolymer	Not Available	Not Available
polyethylene	Not Available	Not Available
polyethylene terephthalate	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
lithium nickel oxide	E	≤ 0.01 mg/m³
lithium cobaltate	E	≤ 0.01 mg/m³
lithium fluorophosphate	E	≤ 0.01 mg/m³
ethylene carbonate	E	≤ 0.01 mg/m³
propylene carbonate	E	≤ 0.1 ppm
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

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Exposure controls

Appropriate engineering controls	Articles or manufactured items, in their original condition, generally don't require engineering controls during handling or in normal use. Exceptions may arise following extensive use and subsequent wear, during recycling or disposal operations where substances, found in the article, may be released to the environment.
Personal protection	
Eye and face protection	▸ Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure. ▸ Chemical goggles.whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted. ▸ Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection. ▸ Alternatively a gas mask may replace splash goggles and face shields. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] No special equipment required due to the physical form of the product. ▸ Safety glasses with side shields. ▸ Chemical goggles. ▸ 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], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	▸ Elbow length PVC gloves **NOTE:** ▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. No special equipment required due to the physical form of the product.
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ P.V.C apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.

Respiratory protection

Type A-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	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

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

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Manufactured sealed battery.

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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**	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 (Not Available%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may produce toxic effects. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation. Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.
Ingestion	Toxic effects may result from the accidental ingestion of the material; animal experiments indicate that ingestion of less than 40 gram may be fatal or may produce serious damage to the health of the individual. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact. The material may accentuate any pre-existing dermatitis condition Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	On the basis of epidemiological data, it has been concluded that prolonged inhalation of the material, in an occupational setting, may produce cancer in humans. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population.

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	Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in the development of heritable genetic damage, generally on the basis of - appropriate animal studies, - other relevant information Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.

XU1 Lithium-ion Battery Pack	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
lithium nickel oxide	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
lithium cobaltate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Not Available
	Inhalation(Rat) LC50; 5.05 mg/l4h[1]	
	Oral (Rat) LD50; >5000 mg/kg[1]	
lithium manganate	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
graphite	**TOXICITY**	**IRRITATION**
	Inhalation(Rat) LC50; >2 mg/L4h[1]	Not Available
	Oral (Rat) LD50; >2000 mg/kg[1]	
lithium fluorophosphate	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; 50-300 mg/kg[1]	Not Available
ethylene carbonate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit): 20 mg - mild
	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]
ethyl methyl carbonate	**TOXICITY**	**IRRITATION**
	Inhalation(Rat) LC50; >17.6 mg/l4h[1]	Not Available
	Oral (Rat) LD50; >5000 mg/kg[1]	
propylene carbonate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >=2000 mg/kg[1]	Eye (rabbit): 60 mg - moderate
	Oral (Rat) LD50; >5000 mg/kg[1]	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]
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]

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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]
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]	
carbon, non-activated	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
vinylidene fluoride homopolymer	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
polyethylene	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Not Available
	Oral (Rat) LD50; >2000 mg/kg[1]	
polyethylene terephthalate	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
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 COBALTATE

Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances.
Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens).
Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis.
Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.
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.

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

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	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 CO_2, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO_2, 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

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	carbonate are unlikely
NICKEL	Oral (rat) TDLo: 500 mg/kg/5D-I Inhalation (rat) TCLo: 0.1 mg/m3/24H/17W-C Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [*National Toxicology Program: U.S. Dep. of Health & Human Services 2002*]
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).
CARBON, NON-ACTIVATED	Substance has been investigated as a reproductive effector.
POLYETHYLENE	polyethylene pyrolyzate for poly-alpha-olefins (PAOs): PAOs are highly branched isoparaffinic chemicals produced by oligomerisation of 1-octene, 1-decene, and/or 1-dodecene. The crude polyalphaolefin mixture is then distilled into appropriate product fractions to meet specific viscosity specifications and hydrogenated. Read across data exist for health effects endpoints from the following similar *hydrogenated* long chain branched alkanes derived from a C8, C10, and/or C12 alpha olefins: ▸ Decene homopolymer ▸ Decene/dodecene copolymer ▸ Octene/decene/dodecene copolymer ▸ Dodecene trimer The data for these structural analogs demonstrated no evidence of health effects. In addition, there is evidence in the literature that alkanes with 30 or more carbon atoms are unlikely to be absorbed when administered orally. The physicochemical data suggest that it is unlikely that significant absorption will occur. If a substance of the size and structure of a typical PAO is absorbed, then the principal mechanisms of absorption after oral administration are likely to be passive diffusion and absorption by way of the lymphatic system. The former requires both good lipid solubility and good water solubility as the substance has to partition from an aqueous environment through a lipophilic membrane into another aqueous environment during absorption. Absorption by way of the lymphatics occurs by mechanisms analogous to those that absorb fatty acids and is limited by the size of the molecule. Lipophilicity generally enhances the ability of chemicals to cross biological membranes. Biotransformation by mixed function oxidases often increases the water solubility of a substance; however, existing data suggest that these substances will not undergo oxidation to more hydrophilic metabolites. Finally, a chemical must have an active functional group that can interact chemically or physically with the target cell or receptor upon reaching it; there are no moieties in PAOs that represent a functional group that may have biological activity. The water solubilities of a C10 dimer PAO and a C12 trimer PAO were determined to be <1 ppb and < 1 ppt respectively. The partition coefficient for a C12 trimer PAO was determined to be log Kow of >7 . Given the very low water solubility it is extremely unlikely that PAOs will be absorbed by passive diffusion following oral administration, and the size of the molecules suggest that the extent of lymphatic absorption is likely to be very low. Although PAOs are relatively large lipophilic compounds, and molecular size may be a critical limiting determinant for absorption, there is some evidence that these substances are absorbed. However, the lack of observed toxicity in the studies with PAOs suggests that these products are absorbed poorly, if at all. Furthermore, a review of the literature regarding the absorption and metabolism of long chain alkanes indicates that alkanes with 30+ carbon atoms are unlikely to be absorbed. For example the absorption of squalane, an analogous C30 product, administered orally to male CD rats was examined - essentially all of the squalane was recovered unchanged in the faeces. At the same time, the hydrophobic properties of PAOs suggest that, should they be absorbed, they would undergo limited distribution in the aqueous systemic circulation and reach potential target organs in limited concentrations. In addition to the general considerations discussed above, the low volatility of PAOs indicates that, under normal conditions of use or transportation, exposure by the inhalation route is unlikely. In particular, the high viscosity of these substances suggests that it would be difficult to generate a high concentration of respirable particles in the air. **Acute toxicity:** PAOs (decene/dodecene copolymer, octene/decene/dodecene homo-polymer, and dodecene trimer) have been adequately tested for acute oral toxicity. There were no deaths when the test materials were administered at doses of 5,000 mg/kg (decene/dodecene copolymer and dodecene trimer) and at 2,000 mg/kg (octene/decene/dodecene copolymer) in rats. Overall, the acute oral LD50 for these substances was greater than the 2000 mg/kg limit dose, indicating a relatively low order of toxicity. PAOs (decene/dodecene copolymer, octene/decene/dodecene copolymer, and dodecene trimer) have been tested for acute dermal toxicity. No mortality was observed for any substance when administered at the limit dose of 2000 or 5000 mg/kg. Overall, the acute dermal LD50 for these substances was greater than the 2000 mg/kg limit dose, indicating a relatively low order of toxicity. 1-Decene, homopolymer, is absorbed (unexpectedly for a high molecular weight polymer) to a moderate degree in rat skin and is eliminated slowly PAOs (decene homopolymer, decene/dodecene copolymer, and decene trimer) have been tested for acute inhalation toxicity. Rats were exposed to aerosols of the substances at nominal atmospheric concentrations of 2.5, 5.0, and 5.06 mg/L, respectively, for four hours. These levels were the maximum attainable concentrations under the conditions of the tests, due to the low volatility and high viscosity of the test material. No mortality was noted, and all animals fully recovered following depuration. The lack of mortality at concentrations at or above the limit dose of 2.0 mg/L indicates a relatively low order of toxicity for these substances.

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	Repeat dose toxicity: Eight repeated-dose toxicity studies using two different animal species, rats and mice, and oral and dermal routes of administration have been conducted with three structural analogs. These data suggest that the structural analogs exhibit a low order of toxicity following repeated applications, due to their similarity in chemical structures and physicochemical properties. One 28-day oral toxicity study in rats, one 90-day dermal and two 90-day dietary studies in rats, and a dermal carcinogenicity study in mice exist for decene homopolymer. A rat oral combined reproductive toxicity and 91-day systemic toxicity study was also conducted with decene homopolymer. In addition, 28-day rat oral toxicity studies exist for two structurally analogous substances (dodecene trimer and octene/decene /dodecene copolymer); and a 90-day rat dermal toxicity study exists for octene/decene/dodecene copolymer. Results from these studies show a low order of repeated dose toxicity. The dermal NOAEL for systemic toxicity studies was equal to or greater than 2000 mg/kg/day. The oral NOAEL for 1-decene homopolymer is between 5,000 and 20,000 mg/kg/day in Sprague-Dawley rats. Rats exposed repeatedly by dermal exposure at doses of 2000 mg/kg decene/dodecene copolymer showed increased incidences of hyperplasia of the sebaceous glands, hyperplasia/hyperkeratosis of the epidermis and dermal inflammation. These symptoms generally subsided within 2 weeks. Males showed decreased body weight gain and altered serum chemistry. In a 90-day feeding study rats receiving 20000 ppm of 1-decene, homopolymer, hydrogenated did not exhibit any clinical signs of systemic toxicity. Marginal effects on clinical chemistry (glucose and ALT in males; sodium, phosphorus and calcium in females) were seen. **Reproductive toxicity:** Data are available for decene homopolymer. Results from these studies show a low order of reproductive/ developmental toxicity. The NOAEL for reproductive toxicity was 1000 mg/kg/day, the highest concentration tested. The lack of effects on fertility in this study or effects on reproductive organs in this or other subchronic studies with closely related chemicals indicates that PAOs are unlikely to exert effects on reproduction. **Developmental toxicity:** Decene homopolymer (with 10 ppm of an antioxidant) was administered once daily on gestation days 0-19 via dermal application to presumed-pregnant rats at doses of 0, 800, and 2000 mg/kg/day. Dermal administration of the test material did not adversely affect parameters of reproductive performance during gestation, nor did it adversely affect *in utero* survival and development of the offspring. The NOAEL in this study for developmental parameters was 2000 mg/kg/day. **Genotoxicity:** Information for the following PAOs (decene homopolymer, octene/decene/dodecene copolymer, dodecene trimer; and decene/dodecene copolymer [*prepared from 10% C12 and 90% C10 alpha olefins; approx. 33% trimer and 51% tetramer, 16% pentamer and higher*]) is available. Either bacterial or mammalian gene mutation assays, *in vitro* chromosomal aberration assays, or *in vivo* chromosomal aberration assays have been conducted for these substances. Neither mutagenicity nor clastogenicity were exhibited by any of these substances in the referenced *in vivo* or *in vitro* tests, with or without metabolic activation. **Carcinogenicity:** While alpha-olefin polymers have similar properties to mineral oils, they do not contain polycyclic aromatic hydrocarbons, or other known possible carcinogens. Decene homopolymer produced no treatment-related tumors in C3H mice treated with a 50 ul/application twice weekly for 104 weeks. In addition, survival (56%) was greater than in any other group, including the untreated control. Inclusion of polyethylene in the diet of rats at 8 g/kg/day did not result in treatment-related effects. Polyethylene implanted into rats and mice has reportedly caused local tumorigenic activity at doses of 33 to 2120 mg/kg, but the relevance to human exposure is not certain. The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
POLYETHYLENE TEREPHTHALATE	PET might yield endocrine disruptors under conditions of common use . Proposed mechanisms include leaching of phthalates as well as leaching of antimony (a catalyst used in its production). For polyethylene terephthalate (PET polyesters) and its derivatives No adverse effects described in animals from short exposures by inhalation, ingestion or skin contact. Animal testing indicates that this compound does not have carcinogenic mutagenic, embryotoxic, nor reproductive effects. * DuPont Acetaldehyde forms by degradation of PET through the mishandling of the material. At high temperatures, (PET decomposes above 300 C or 570 F), high pressures, extruder speeds (excessive shear flow raises temperature) and long barrel residence times all contribute to the production of acetaldehyde. When acetaldehyde is produced, some of it remains dissolved in the walls of a container and then diffuses into the product stored inside, altering the taste and aroma. For bottled water low acetaldehyde content is quite important, because if nothing masks the aroma, even extremely low concentrations (10–20 parts per billion in the water) of acetaldehyde can produce an off-taste. Is is suggested that polyethylene terephthlatae (PET) may yield endocrine disruptors under conditions of common use . Proposed mechanisms include leaching of phthalates as well as leaching of antimony (a catalyst used in its production. However phthalate ester plasticizers are not used to manufacture polyethylene terephthalate. Some reports of phthalate esters in PET bottled water containers suggest that these might originated from contamination of the bottled water, or from phthalate ester contamination of recycled PET used in manufacturing water and beverage containers. When comparing water of the same spring that is packed in glass or plastic bottles made of polyethylene terephthalate (PET), one study found estrogenic activity is three times higher in water from plastic bottles. These data support the hypothesis that PET packaging materials are a source of estrogen-like compounds. Furthermore, the findings presented here conform to previous studies and indicate that the contamination of bottled water with endocrine disruptors is a transnational phenomenon. Endocrine disruptors in bottled mineral water: Estrogenic activity in the E-Screen: Martin Wagnerand Jörg Oehlmann: The Journal of Steroid Biochemistry and Molecular Biology Volume 127, Issues 1–2, October 2011, Pages 128-135 An article published in Journal of Environmental Monitoring in April 2012 concludes that antimony concentration in deionized water stored in PET bottles stays within EU's acceptable limit even if stored briefly at temperatures up to 60 deg C (140 deg F), while bottled contents (water or soft drinks) may occasionally exceed the EU limit after less than a year of storage at room temperature
LITHIUM NICKEL OXIDE & LITHIUM COBALTATE & 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 OXIDE & GRAPHITE & LITHIUM FLUOROPHOSPHATE & 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.
LITHIUM COBALTATE & LITHIUM MANGANATE & GRAPHITE & LITHIUM FLUOROPHOSPHATE & ETHYL METHYL CARBONATE	No significant acute toxicological data identified in literature search.

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& ALUMINIUM & VINYLIDENE FLUORIDE HOMOPOLYMER & POLYETHYLENE TEREPHTHALATE	
PROPYLENE CARBONATE & NICKEL	**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.

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

XU1 Lithium-ion Battery Pack

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

lithium nickel oxide

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

lithium cobaltate

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	24h	Algae or other aquatic plants	0.025mg/l	2
EC50	48h	Crustacea	5.89mg/l	2
EC50	96h	Algae or other aquatic plants	23.8mg/l	2
LC50	96h	Fish	1.512mg/l	2

lithium manganate

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

graphite

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

lithium fluorophosphate

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

ethylene carbonate

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

ethyl methyl carbonate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>62mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	62mg/l	2
EC50	48h	Crustacea	>100mg/l	2
LC50	96h	Fish	>100mg/l	2

propylene carbonate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>900mg/l	1
NOEC(ECx)	72h	Algae or other aquatic plants	900mg/l	1

Continued...

	EC50	48h	Crustacea	>1000mg/l	1
	LC50	96h	Fish	1000mg/l	1

nickel					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50(ECx)	72h	Algae or other aquatic plants	0.18mg/l	1
	EC50	72h	Algae or other aquatic plants	0.18mg/l	1
	EC50	48h	Crustacea	>100mg/l	1
	EC50	96h	Algae or other aquatic plants	0.36mg/l	2
	LC50	96h	Fish	0.168mg/L	4

aluminium					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96h	Fish	0.078-0.108mg/l	2
	NOEC(ECx)	48h	Crustacea	>100mg/l	1
	EC50	72h	Algae or other aquatic plants	0.2mg/l	2
	EC50	48h	Crustacea	1.5mg/l	2
	EC50	96h	Algae or other aquatic plants	0.024mg/l	2

copper					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50(ECx)	24h	Algae or other aquatic plants	<0.001mg/L	4
	EC50	72h	Algae or other aquatic plants	0.011-0.017mg/L	4
	EC50	48h	Crustacea	<0.001mg/L	4
	EC50	96h	Algae or other aquatic plants	0.03-0.058mg/l	4
	LC50	96h	Fish	0.005-0.06mg/l	4

carbon, non-activated					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	NOEC(ECx)	72h	Algae or other aquatic plants	50mg/L	4

vinylidene fluoride homopolymer					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available

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

polyethylene terephthalate					
	Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available

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**
ethylene carbonate	HIGH	HIGH
ethyl methyl carbonate	HIGH	HIGH
propylene carbonate	HIGH	HIGH
vinylidene fluoride homopolymer	LOW	LOW
polyethylene	LOW	LOW

Bioaccumulative potential

Ingredient	**Bioaccumulation**
ethylene carbonate	LOW (LogKOW = -0.3388)
ethyl methyl carbonate	LOW (LogKOW = 0.7247)
propylene carbonate	LOW (LogKOW = -0.41)
vinylidene fluoride homopolymer	LOW (LogKOW = 1.24)
polyethylene	LOW (LogKOW = 1.2658)

Mobility in soil

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

Continued...

Ingredient	Mobility
propylene carbonate	LOW (KOC = 14.85)
vinylidene fluoride homopolymer	LOW (KOC = 35.04)
polyethylene	LOW (KOC = 14.3)

SECTION 13 Disposal considerations

Waste treatment methods

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

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	NO
HAZCHEM	2Y

Land transport (ADG)

UN number	3481
UN proper shipping name	LITHIUM ION BATTERIES CONTAINED IN EQUIPMENT or LITHIUM ION BATTERIES PACKED WITH EQUIPMENT
Transport hazard class(es)	Class: 9 Subrisk: Not Applicable
Packing group	Not Applicable
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: 188 230 310 348 360 376 377 384 387 Limited quantity: 0

Air transport (ICAO-IATA / DGR)

UN number	3481
UN proper shipping name	Lithium ion batteries packed with equipment (including lithium ion polymer batteries); Lithium ion batteries contained in equipment (including lithium ion polymer batteries)
Transport hazard class(es)	ICAO/IATA Class: 9 ICAO / IATA Subrisk: Not Applicable ERG Code: 12FZ
Packing group	Not Applicable
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: A48 A88 A99 A154 A164 A181 A185 A206 A213; A88 A99 A154 A164 A181 A185 A206 A213 Cargo Only Packing Instructions: 967; 966 Cargo Only Maximum Qty / Pack: 35 kg Passenger and Cargo Packing Instructions: 967; 966 Passenger and Cargo Maximum Qty / Pack: 5 kg Passenger and Cargo Limited Quantity Packing Instructions: Forbidden Passenger and Cargo Limited Maximum Qty / Pack: Forbidden

Sea transport (IMDG-Code / GGVSee)

UN number	3481

Continued...

UN proper shipping name	LITHIUM ION BATTERIES CONTAINED IN EQUIPMENT or LITHIUM ION BATTERIES PACKED WITH EQUIPMENT (including lithium ion polymer batteries)
Transport hazard class(es)	IMDG Class: 9 IMDG Subrisk: Not Applicable
Packing group	Not Applicable
Environmental hazard	Not Applicable
Special precautions for user	EMS Number: F-A, S-I Special provisions: 188 230 310 348 360 376 377 384 387 390 Limited Quantities: 0

Transport in bulk according to Annex II of MARPOL and the IBC code

Not Applicable

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

Product name	Group
lithium nickel oxide	Not Available
lithium cobaltate	Not Available
lithium manganate	Not Available
graphite	Not Available
lithium fluorophosphate	Not Available
ethylene carbonate	Not Available
ethyl methyl carbonate	Not Available
propylene carbonate	Not Available
nickel	Not Available
aluminium	Not Available
copper	Not Available
carbon, non-activated	Not Available
vinylidene fluoride homopolymer	Not Available
polyethylene	Not Available
polyethylene terephthalate	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
lithium nickel oxide	Not Available
lithium cobaltate	Not Available
lithium manganate	Not Available
graphite	Not Available
lithium fluorophosphate	Not Available
ethylene carbonate	Not Available
ethyl methyl carbonate	Not Available
propylene carbonate	Not Available
nickel	Not Available
aluminium	Not Available
copper	Not Available
carbon, non-activated	Not Available
vinylidene fluoride homopolymer	Not Available
polyethylene	Not Available
polyethylene terephthalate	Not Available

SECTION 15 Regulatory information

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

lithium nickel 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

lithium cobaltate is found on the following regulatory lists

Continued...

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

lithium manganate is found on the following regulatory lists

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)

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)

ethylene 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

propylene carbonate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

nickel is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

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)

copper is found on the following regulatory lists

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

carbon, non-activated 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)

polyethylene is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

polyethylene terephthalate 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)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (lithium nickel oxide; lithium manganate; ethyl methyl carbonate)
Canada - DSL	No (lithium nickel oxide; lithium manganate; lithium fluorophosphate; ethyl methyl carbonate)
Canada - NDSL	No (lithium nickel oxide; lithium cobaltate; lithium manganate; graphite; ethylene carbonate; propylene carbonate; nickel; aluminium; copper; carbon, non-activated; vinylidene fluoride homopolymer; polyethylene; polyethylene terephthalate)
China - IECSC	No (lithium nickel oxide; lithium manganate)
Europe - EINEC / ELINCS / NLP	No (lithium nickel oxide; lithium manganate; vinylidene fluoride homopolymer; polyethylene; polyethylene terephthalate)
Japan - ENCS	No (lithium manganate; graphite; lithium fluorophosphate; nickel; aluminium; copper; carbon, non-activated)
Korea - KECI	Yes
New Zealand - NZIoC	No (lithium fluorophosphate; ethyl methyl carbonate)
Philippines - PICCS	No (lithium nickel oxide; lithium cobaltate; lithium manganate)

Continued...

National Inventory	Status
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (lithium nickel oxide; lithium cobaltate; lithium manganate; lithium fluorophosphate; ethylene carbonate; ethyl methyl carbonate; vinylidene fluoride homopolymer)
Vietnam - NCI	No (lithium cobaltate)
Russia - FBEPH	No (lithium nickel oxide; lithium cobaltate; lithium manganate; 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	26/05/2022
Initial Date	26/05/2022

SDS Version Summary

Version	Date of Update	Sections Updated
2.2	06/06/2022	Supplier Information, Synonyms

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