

Material Safety Data Sheet Cover-Sheet – This page provides additional New Zealand specific information for this product and must be read in conjunction with the Safety Data Sheet (SDS) attached

Product Name: Lithium-ion polymer battery in LED mouthpiece

Manufacturer: SDI Limited

SDS Expiry: 10 March 2028

Supplier Details: Henry Schein New Zealand
243-249 Bush Road, Rosedale, Auckland, 0632
PO Box 101 140, North Shore, Auckland 0745
Ph. 0800 808 855
www.henryschein.co.nz

Emergency Contacts: Poisons/Hazardous Chemical Info Centre –
0800POISON/0800764766 (24 Hours)
Phone 111 for Fire, Ambulance or Police

HSNO Class/Category: 6 / 8 / 9

HSNO Group Standard: Dental Products Subsidiary Hazard Group Standard 2020
HSR002558

Statements/Pictograms: As per attached Safety Data Sheet (SDS)

Date Prepared: This coversheet was prepared – January 2026

This SDS coversheet has been produced by Henry Schein NZ and has been prepared in accordance with NZ EPA advice on making overseas SDS compliant to HSNO Act. The above information is based on the present state of our knowledge of the product at the time of publication. It is given in good faith, no warranty is implied with respect to the quality or the specifications of the product. Users must satisfy that the product is entirely suitable for their purpose. The SDS and this coversheet may be revised from time to time, please ensure you have a current copy.

Lithium-ion polymer battery in LED mouthpiece

SDI Limited

Version No: 4.1

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

Initial Date: 28/07/2020

Revision Date: 10/03/2023

Print Date: 29/09/2025

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

Product Identifier

Product name	Lithium-ion polymer battery in LED mouthpiece
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	LITHIUM ION BATTERIES PACKED WITH EQUIPMENT (including lithium ion polymer batteries)
Chemical formula	Not Applicable
Other means of identification	Not Available

Relevant identified uses of the substance or mixture and uses advised against

Relevant identified uses	NOTE: Hazard statement relates to battery contents. Potential for exposure should not exist unless the battery leaks, is exposed to high temperatures or is mechanically, physically or electrically abused. Use according to manufacturer's directions.
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Details of the manufacturer or importer of the safety data sheet

Registered company name	SDI Limited	SDI (North America) Inc.	SDI HOLDINGS PTY LTD DO
Address	3-15 Brunsdon Street Bayswater VIC 3153 Australia	1279 Hamilton Parkway Itasca IL 60143 United States	Rua Dr. Reinaldo Schmithausen 3141 – Cordeiros Itajaí – SC – CEP 88310-004 Brazil
Telephone	+61 3 8727 7111	+1 630 361 9200	+55 11 3092 7100
Fax	+61 3 8727 7222	Not Available	Not Available
Website	www.sdi.com.au	www.sdi.com.au	https://www.sdi.com.au/
Email	info@sdi.com.au	USA.Canada@sdi.com.au	Brasil@sdi.com.au

Registered company name	SDI Germany GmbH
Address	Hansestrasse 85 Cologne D-51149 Germany
Telephone	+49 0 2203 9255 0
Fax	+49 0 2203 9255 200
Website	www.sdi.com.au
Email	germany@sdi.com.au

Emergency telephone number

Association / Organisation	SDI Limited	CHEMWATCH EMERGENCY RESPONSE (24/7)
Emergency telephone number(s)	131126 Poisons Information Centre	+64 800 700 112 (ID#: 5417-91)
Other emergency telephone number(s)	+61 3 8727 7111	+61 3 9573 3188

SECTION 2 Hazards identification

Classification of the substance or mixture

Classification [1]	Acute Toxicity (Oral) Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Acute Toxicity (Inhalation) Category 2, Sensitisation (Respiratory) Category 1, Carcinogenicity Category 1, Specific Target Organ Toxicity - Repeated Exposure Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
Legend:	1. Classification by vendor; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI
Determined by using GHS/HSNO criteria	6.1B (inhalation), 6.1B (oral), 8.3A, 6.5A (respiratory), 6.5B (contact), 6.7A, 6.9B, 9.1C

Label elements

Lithium-ion polymer battery in LED mouthpiece		
Hazard pictogram(s)		
Signal word		Danger
Hazard statement(s)		
H300		Fatal if swallowed.
H317		May cause an allergic skin reaction.
H318		Causes serious eye damage.
H330		Fatal if inhaled.
H334		May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H350		May cause cancer.
H373		May cause damage to organs through prolonged or repeated exposure.
H412		Harmful to aquatic life with long lasting effects.
Precautionary statement(s) Prevention		
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.
P284		[In case of inadequate ventilation] wear respiratory protection.
P273		Avoid release to the environment.
P202		Do not handle until all safety precautions have been read and understood.
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.
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.
P330		Rinse mouth.
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		
P403+P233		Store in a well-ventilated place. Keep container tightly closed.
P405		Store locked up.
Precautionary statement(s) Disposal		
P501		Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
No further product hazard information.		
SECTION 3 Composition / information on ingredients		
Substances		
See section below for composition of Mixtures		
Mixtures		
CAS No	%[weight]	Name
Not Available		Sealed containers with electrochemical contents, typically
12190-79-3	53.6	<u>lithium cobaltate</u>
7782-42-5	17.4	<u>graphite</u>
7440-50-8	12.5	<u>copper</u>
7429-90-5	12.5	<u>aluminium</u>
96-49-1	3.17	<u>ethylene carbonate</u>
21324-40-3	0.83	<u>lithium fluorophosphate</u>
Legend:	1. Classification by vendor; 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		
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Description of first aid measures

Eye Contact	► Generally not applicable.
Skin Contact	► Generally not applicable.
Inhalation	► Generally not applicable.
Ingestion	► Generally not applicable.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Firefighting measures**Extinguishing media**

- Sand, dry powder extinguishers or other inerts should be used to smother dust fires.
- **DO NOT** use halogenated fire extinguishing agents.

Special hazards arising from the substrate or mixture

Fire Incompatibility	<ul style="list-style-type: none"> ► If leaked, forbidden to contact with strong oxidisers, mineral acids, strong alkalies, halogenated hydrocarbons. ► Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result ► Keep dry ► NOTE: May develop pressure in containers; open carefully. Vent periodically.
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ► Alert Fire Brigade and tell them location and nature of hazard. ► Wear breathing apparatus plus protective gloves in the event of a fire. ► Prevent, by any means available, spillage from entering drains or water courses. ► Use fire fighting procedures suitable for surrounding area. ► DO NOT approach containers suspected to be hot. ► Cool fire exposed containers with water spray from a protected location. ► If safe to do so, remove containers from path of fire. ► Equipment should be thoroughly decontaminated after use. <p>Slight hazard when exposed to heat, flame and oxidisers.</p>
Fire/Explosion Hazard	<p>Articles and manufactured articles may constitute a fire hazard where polymers form their outer layers or where combustible packaging remains in place.</p> <p>Certain substances, found throughout their construction, may degrade or become volatile when heated to high temperatures. This may create a secondary hazard.</p> <p>Decomposes on heating and produces toxic fumes of: carbon dioxide (CO₂) metal oxides other pyrolysis products typical of burning organic material. May form peroxides.</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	<ul style="list-style-type: none"> ► Clean up all spills immediately. ► Secure load if safe to do so. ► Bundle/collect recoverable product. ► Collect remaining material in containers with covers for disposal.
Major Spills	<ul style="list-style-type: none"> ► 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>Conditions to avoid: Heat above 70 deg C or incinerate. Deform. Mutilate. Crush. Disassemble. Overcharge. Short circuit. Expose over a long period to humid conditions.</p> <p>Graphite:</p> <ul style="list-style-type: none"> · is a good conductor of electricity; avoid contact with electrical circuitry. · is a highly lubricious material and may present a slip hazard if spilled on pedestrian surfaces. <p>NOTE:</p> <ul style="list-style-type: none"> ► Wet, activated carbon removes oxygen from the air thus producing a severe hazard to workers inside carbon vessels and in enclosed or confined spaces where activated carbons might accumulate.
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	<ul style="list-style-type: none"> Before entry to such areas, sampling and test procedures for low oxygen levels should be undertaken; control conditions should be established to ensure the availability of adequate oxygen supply. <p>For molten metals:</p> <ul style="list-style-type: none"> Molten metal and water can be an explosive combination. The risk is greatest when there is sufficient molten metal to entrap or seal off water. Water and other forms of contamination on or contained in scrap or remelt ingot are known to have caused explosions in melting operations. While the products may have minimal surface roughness and internal voids, there remains the possibility of moisture contamination or entrapment. If confined, even a few drops can lead to violent explosions. All tooling, containers, molds and ladles, which come in contact with molten metal must be preheated or specially coated, rust free and approved for such use. Any surfaces that may contact molten metal (e.g. concrete) should be specially coated Drops of molten metal in water (e.g. from plasma arc cutting), while not normally an explosion hazard, can generate enough flammable hydrogen gas to present an explosion hazard. Vigorous circulation of the water and removal of the particles minimise the hazard. <p>During melting operations, the following minimum guidelines should be observed:</p> <ul style="list-style-type: none"> Inspect all materials prior to furnace charging and completely remove surface contamination such as water, ice, snow, deposits of grease and oil or other surface contamination resulting from weather exposure, shipment, or storage. Store materials in dry, heated areas with any cracks or cavities pointed downwards. Preheat and dry large objects adequately before charging in to a furnace containing molten metal. This is typically done by the use of a drying oven or homogenising furnace. The dry cycle should bring the metal temperature of the coldest item of the batch to 200 degree C (400 deg F) and then hold at that temperature for 6 hours. Avoid all personal contact, including inhalation. 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	<ul style="list-style-type: none"> Store in original containers. Keep containers securely sealed. Store in a cool, dry, well-ventilated area. Store away from incompatible materials and foodstuff containers. Protect containers against physical damage and check regularly for leaks. Observe manufacturer's storage and handling recommendations contained within this SDS. Store away from incompatible materials.

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 practicable, reuse the original packaging or something providing a similar level of protection to both the article and the handler.
Storage incompatibility	<ul style="list-style-type: none"> Avoid reaction with oxidising agents strong alkalis water

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 cobaltate	Inhalable dust (not otherwise classified)	10 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	lithium cobaltate	Respirable dust (not otherwise classified)	3 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	graphite	Graphite, all forms except graphite fibres respirable dust	3 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	copper	Respirable dust (not otherwise classified)	3 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	copper	Copper and its inorganic compounds, as Cu respirable dust	0.01 mg/m3	Not Available	Not Available	dsen - Dermal sensitisier
New Zealand Workplace Exposure Standards (WES)	copper	Inhalable dust (not otherwise classified)	10 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	aluminium	Respirable dust (not otherwise classified)	3 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	aluminium	Aluminium metal and insoluble aluminium compounds (including pyro powder, aluminium oxide, and aluminium welding fumes), as Al respirable dust	1 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	aluminium	Inhalable dust (not otherwise classified)	10 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	lithium fluorophosphate	Respirable dust (not otherwise classified)	3 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	lithium fluorophosphate	Inhalable dust (not otherwise classified)	10 mg/m3	Not Available	Not Available	Not Available

Lithium-ion polymer battery in LED mouthpiece

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

MATERIAL DATA

Exposure controls

Appropriate engineering controls	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <ul style="list-style-type: none"> ▶ Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area. ▶ Work should be undertaken in an isolated system such as a "glove-box". Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system. ▶ Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within. ▶ Open-vessel systems are prohibited. ▶ Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation. ▶ Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system. ▶ For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▶ Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas). ▶ Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air. ▶ Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed. <p>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.</p> <p>Exhaust ventilation should be designed to prevent accumulation and recirculation in the workplace and safely remove carbon black from the air.</p> <p>Note: Wet, activated carbon removes oxygen from the air and thus presents a severe hazard to workers inside carbon vessels and enclosed or confined spaces. Before entering such areas sampling and test procedures for low oxygen levels should be undertaken and control conditions set up to ensure ample oxygen availability.[Linde]</p>
Individual protection measures, such as personal protective equipment	     
Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].
Skin protection	See Hand protection below
Hands/feet protection	<ul style="list-style-type: none"> ▶ Wear general protective gloves, eg. light weight rubber gloves. <p>NOTE:</p> <ul style="list-style-type: none"> ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent] ▶ Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent] ▶ Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely. ▶ Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. ▶ Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. <p>During repair or maintenance activities the potential exists for exposures to toxic metal particulate in excess of the occupational standards. Under these circumstances, protecting workers can require the use of specific work practices or procedures involving the combined use of</p>

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

Full face respirator with supplied air.

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	Rectangular shaped battery; insoluble in water.		
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 Applicable
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 Available	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 Available
Vapour pressure (kPa)	Not Applicable	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Heat of Combustion (kJ/g)	Not Available	Ignition Distance (cm)	Not Available
Flame Height (cm)	Not Available	Flame Duration (s)	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	Enclosed Space Ignition Deflagration Density (g/m3)	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	<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

a) Acute Toxicity	There is sufficient evidence to classify this material as acutely toxic.
b) Skin Irritation/Corrosion	Based on available data, the classification criteria are not met.

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Lithium-ion polymer battery in LED mouthpiece

c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	There is sufficient evidence to classify this material as sensitising to skin or the respiratory system
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	There is sufficient evidence to classify this material as carcinogenic
g) Reproductivity	Based on available data, the classification criteria are not met.
h) STOT - Single Exposure	Based on available data, the classification criteria are not met.
i) STOT - Repeated Exposure	There is sufficient evidence to classify this material as toxic to specific organs through repeated exposure
j) Aspiration Hazard	Based on available data, the classification criteria are not met.
Inhaled	<p>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.</p> <p>Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation of dusts, generated by the material during the course of normal handling, may be damaging to the health of the individual.</p>
Ingestion	<p>Considered an unlikely route of entry in commercial/industrial environments</p> <p>Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.</p>
Skin Contact	<p>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.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Irritation and skin reactions are possible with sensitive skin</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>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.</p>
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	<p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>Substances that can cause 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 cause 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.</p> <p>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.</p> <p>On the basis, primarily, of animal experiments, the material may be regarded as carcinogenic to humans. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in cancer on the basis of:</p> <ul style="list-style-type: none"> - appropriate long-term animal studies - other relevant information <p>Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.</p> <p>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.</p> <p>Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic.</p> <p>Because aluminium competes with calcium for absorption, increased amounts of dietary aluminium may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies, aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseous tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory. These salts' estrogen-like effects have led to their classification as a metalloestrogen. Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.</p> <p>After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus.</p>

Continued...

Aluminium may persist for a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans.

At high levels of exposure, some aluminium compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminium compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminium potassium sulphate at high levels in the diet.

Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer's disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produce lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, testes, embryotoxicity, and the developing nervous system of 52, 75, 100, and 50 mg aluminium/kg bw/day, respectively. Similarly, the lowest no-observed-adverse-effect levels (NOAELs) for effects on these endpoints were reported at 30, 27, 100, and for effects on the developing nervous system, between 10 and 42 mg aluminium/kg bw per day, respectively.

Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 mg/l aluminium compared with communities where the aluminium level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminium exposure to brain disease. Aluminium concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramid-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations.

Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of "tau" a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminium hyperphosphorylates tau in vitro. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade. Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA).

Aluminium enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminium include baking powder, antacids and aluminium products used for general food preparation and storage (over 12 months, aluminium levels in soft drink packed in aluminium cans rose from 0.05 to 0.9 mg/l). [Walton, J and Bryson-Taylor, D. - *Chemistry in Australia, August 1995*]

the main target organs of aluminum are the central nervous system and bone. Aluminum binds with dietary phosphorus and impairs gastrointestinal absorption of phosphorus. The decreased phosphate body burden results in osteomalacia (softening of the bones due to defective bone mineralization) and rickets. Aluminum's neurotoxicity is believed to involve several mechanisms. Changes in cytoskeletal protein functions as a result of altered phosphorylation, proteolysis, transport, and synthesis are believed to be one cause. Aluminum may induce neurobehavioral effects by affecting permeability of the blood-brain barrier, cholinergic activity, signal transduction pathways, lipid peroxidation, and impair neuronal glutamate nitric oxide-cyclic GMP pathway, as well as interfere with metabolism of essential trace elements because of similar coordination chemistries and consequent competitive interactions. It has been suggested that aluminum's interaction with estrogen receptors , but studies have not been able to establish a clear link between aluminum and increased risk of breast cancer). Certain aluminum salts induce immune responses by activating inflammasomes.

In general, available cohort studies in humans have not reported a significant increase in total mortality as a result of cobalt exposure. Several studies have noted increased mortality rates resulting from lung cancer following occupational exposure to cobalt, either as a mixture of cobalt compounds or as hard metal, a metal alloy with a tungsten carbide and cobalt matrix. Fatal cases of hard metal disease and cardiomyopathy believed to have resulted from occupational cobalt exposure have also been reported. However, in the majority of these and other reported occupational studies, co-exposure to other substances was common, and was unable to be corrected for in the analysis. The effects of chronic occupational exposure to cobalt and cobalt compounds on the respiratory system in humans are well-documented. These effects include respiratory irritation, diminished pulmonary function, wheezing, asthma, pneumonia, and fibrosis and occurred at exposure levels ranging from 0.007 to 0.893 mg cobalt/m³ (exposure from 2 to 17 years). These effects have been observed in workers employed in cobalt refineries, as well as hard metal workers, diamond polishers, and ceramic dish painters (painting with cobalt blue dye). Occupational asthma attributed to the inhalation of cobalt powder has been confirmed following bronchial challenge tests. Chest tightness and chronic bronchitis have been recorded in hard-metal workers exposed to cobalt. Cobalt is known to function as a hapten, resulting in the generation of antibodies against cobalt-protein complexes. Although the minimum exposure level associated with cobalt sensitisation has not been determined, sensitisation has been demonstrated in hard metal workers with work-related asthma who have experienced prolonged occupational exposure (>3 years) to levels ranging from 0.007 to 0.893 mg cobalt/m³. The sensitisation phenomenon includes the production of IgE and IgA antibodies to cobalt. Exposure to inhaled cobalt chloride aerosols can precipitate an asthmatic attack in sensitised individuals believed to be the result of an allergic reaction within the lungs.

Allergic dermatitis of an erythematous papular type may also occur following occupational exposure. Dermatitis is a common result of dermal exposure to cobalt in humans that has been verified in a large number of studies. Using patch tests and intradermal injections, it has been demonstrated that the dermatitis is probably caused by an allergic reaction to cobalt. Contact allergy was reported in 22 of 223 (9.9%) nurses who were tested with a patch test of 1.0% cobalt chloride as well as 16 of 79 (20.3%) of examined dentists. Persons with body piercings showed an increased prevalence of allergy to cobalt, with the incidence of contact allergy being proportional to number of piercings. The prevalence of sensitivity to cobalt following exposure to cobalt as a component of metal implants is low, with only 3.8% of patients developing a new sensitivity to cobalt following insertion of the implant.

Exposure levels associated with the development of dermatitis have not been identified. It appears that the allergic properties of cobalt result mainly from exposure to the metal itself, rather than a salt, as it has been demonstrated that daily repeated exposure to aqueous cobalt salts did not result in hand eczema in patients known to have cobalt allergy.

Occupational exposure to cobalt in humans has been reported to cause several effects on the nervous system, including memory loss , nerve deafness, and a decreased visual acuity. It should be noted though, that both of the studies reporting on these findings, had small numbers of subjects, and exposure characterization was not reported.

Chronic exposure to cobalt produces polycythaemia (increase in blood haemoglobin), increased production of cells of the bone marrow and thyroid gland, pericardial effusion and damage to the alpha cells of the pancreas. Chronic exposure to cobalt compounds may result in pericardial effusion, polycardial effusion, cardiac failure, vomiting, convulsions and thyroid enlargement.

Chronic administration of cobaltous chloride has produced goiter, reduced thyroid activity and lowered synthesis rates and levels of cytochrome P-450, an enzymatic system responsible for chemical detoxification, in the liver. A toxic nephritis (kidney disease) may also develop.

Epidemic cardiomyopathy (heart disease) among heavy beer drinkers in the 1960's in Canada, the USA and Belgium has been attributed to the addition of up to 1.5 ppm of cobalt as a foam restorative and stabiliser. Other factors are probably implicated as therapeutic doses of cobalt, up to 50 mg/day (in the treatment of refractory anaemias) do not produce this effect. Inadequate protein or vitamin intake amongst heavy drinkers, or the effects of alcohol in rendering the heart more susceptible to disease may be important.

Single and repeated subcutaneous or intramuscular injection of cobalt powder and salts to rats may cause sarcoma at the injection site but evidence for carcinogenicity by any other route of exposure does not exist. A number of single cases of malignant tumours, mostly sarcomas, have been reported at the site of orthopedic implants containing cobalt.

Animals, exposed to cobalt compounds also exhibit an increase in respiration, as well as tremor and convulsion. Exposure of rats and mice to aerosols of cobalt (as cobalt sulfate) at concentrations from 0.11 to 1.14 mg cobalt/m³ for 2 years resulted in a spectrum of inflammatory, fibrotic, and proliferative lesions in the respiratory tract of male and female rats and mice. Squamous metaplasia of the larynx occurred in rats and mice at exposure concentrations of .011 mg cobalt/m³, with severity of the lesion increasing with increased cobalt concentration. Hyperplastic lesions of the nasal epithelium occurred in rats at concentrations of .011 mg cobalt/m³, and in mice at concentrations of .038 mg cobalt/m³. Both sexes of rats had greatly increased incidences (>90% incidence) of alveolar lesions at all exposure levels, including inflammatory changes, fibrosis, and metaplasia. Similar changes were seen in mice at all exposure levels, though the changes in mice were less severe.

Lithium-ion polymer battery in LED mouthpiece

Cobalt metal dust inhalations by miniature swine resulted in early marked decrease in lung compliance and increases in septal collagen. After a one-week "sensitising period", followed by a 10-day lapse period, further exposures resulted in wheezing produced by hypersensitivity reactions.

Prolonged or repeated inhalation of dust may result in pneumoconiosis (lung disease caused by inhalation dust). Graphite workers have reported symptoms of headaches, coughing, depression, low appetite, dyspnoea (difficult breathing) and black sputum.

A number of studies indicate that graphitosis is a progressive and disabling disease and that the presence of crystalline silica and some silicates as graphite impurities have a pronounced synergistic effect.

Workers suffering from graphite pneumoconiosis have generally worked in the industry for long periods, i.e. 10 years or more, although some cases have been reported after as little as four years.

Data indicate the higher the crystalline silica content of graphite the greater is the severity of the pneumoconiosis.

Pre-employment and periodic examinations should be directed towards detecting significant respiratory disease through chest X-rays and pulmonary function tests

Neuromuscular effects result from chronic over-exposure to lithium compounds. These may include tremor, ataxia, clonus and hyperactive reflexes. Some animal studies have shown that exposure during pregnancy may produce birth defects. Other studies with rats, rabbits and monkeys have not shown teratogenic effects. Human data are ambiguous; it is well established that lithium can cross the human placenta. Of 225 registered pregnancies in which the mothers had received lithium (as a tranquilliser) there were 25 instances of congenital malformation. Although pharmacological doses of lithium cannot be unequivocally designated as a human teratogen, lithium therapy is contraindicated in women of childbearing potential.

Prolonged exposure may produce anorexia, weight loss and emaciation. The kidneys, behavioural/ central nervous system and peripheral nervous system may also show adverse effects.

Various types of dermatitis (psoriasis, alopecia, cutaneous ulcers, acne, follicular papules, xerosis cutis, exfoliative) may also result from chronic skin exposure.

Lithium ion can be an effective treatment for manic depression. It is thought to bind the enzyme IMPase (inositol monophosphatase) and thereby mediates its influence in producing a response to calcium-induced production of neurotransmitters and hormones thought to be responsible for the clinical picture.

Lithium ions interfere with ion transport processes (involving the "sodium pump") that relay and amplify messages carried to the cells of the brain. Mania is associated with irregular increases in protein kinase C (PKC) activity within the brain. Lithium carbonate and sodium valproate, another drug traditionally used to treat the disorder, act in the brain by inhibiting PKC's activity and help to produce other compounds that also inhibit the PKC.

Taking lithium salts has risks and side effects. Extended use of lithium to treat various mental disorders has been known to lead to acquired nephrogenic diabetes insipidus. Nephrogenic diabetes insipidus (NDI), also known as renal diabetes insipidus, is a form of diabetes insipidus primarily due to pathology of the kidney. This is in contrast to central or neurogenic diabetes insipidus, which is caused by insufficient levels of antidiuretic hormone (ADH, also called vasopressin). Nephrogenic diabetes insipidus is caused by an improper response of the kidney to ADH, leading to a decrease in the ability of the kidney to concentrate the urine by removing free water.

Lithium intoxication can affect the central nervous system and renal system and can be lethal

In subchronic studies, rats were exposed to 3 milliequivalents Li/kg/day (equivalent to 1450 mg for a 70 kg person) but did not accumulate Li whilst on a high sodium diet. However when sodium was restricted, fatal kidney toxicity developed. Dogs survived daily dose of 50 mg LiCl/kg for 150 days to the termination of the experiment on a normal sodium intake, whereas the same dose was lethal in 12 to 18 days on a low sodium diet: 20 mg LiCl/kg/day resulted in death in 18 to 30 days.

Several reports have demonstrated that lithium may impair basal ganglia activity. Lithium intoxication has been associated, severe and persistent oculogyric crises. Oculogyric crisis (OGC) is the name of a dystonic reaction to certain drugs or medical conditions characterized by a prolonged involuntary upward deviation of the eyes. The term "oculogyric" refers to the bilateral elevation of the visual gaze but several other responses are associated with the crisis.

Chronic inhalation exposure of production workers has caused decreased pulmonary function and myocardial dystrophy. There is suggestive but inconclusive evidence that carbon black containing polycyclic aromatic hydrocarbons (PAHs) has been responsible for induction of skin cancers in exposed workers.

Long term inhalation of carbon black can cause cough, phlegm, tiredness, chest pain and headache. Dermal, mucosal, or inhalation exposure can cause irritation.

Inhalation of carbon black by mice, rats and monkeys caused thickened alveolar walls, increased pulmonary collagen, right atrial and ventricular strain, hypertrophy of the right atrial and ventricular septum and increased heart weights. Although carbon black itself did not cause cancer in treated animals, carbon black containing polycyclic aromatic hydrocarbons (PAHs) did cause cancer following chronic administration by all routes tested.

Epidemiological studies of workers in the carbon black producing industries of North America and Western Europe show no significant health effect due to occupational exposure to carbon black. Several other studies provide conflicting evidence. Early studies in the former USSR and Eastern Europe report respiratory diseases amongst workers exposed to carbon black, including bronchitis, pneumonia, emphysema and rhinitis. These studies are of questionable validity due to inadequate study design and methodology, lack of appropriate controls for cigarette smoking and other confounding factors such as concurrent exposure to carbon dioxide, coal oil and petroleum vapours. Moreover, review of these studies indicates that the concentrations of carbon black were greater than current occupational standards.

Carbon black may cause adverse pulmonary changes following prolonged or repeated inhalation of the dust; these include oral mucosal lesions, bronchitis and pneumoconiosis which may lead to lung tumours.

The body of evidence of carcinogenicity in animal studies comes from two chronic inhalation studies and two intratracheal instillation studies in rats, which showed significantly elevated rates of lung cancer in exposed animals. An inhalation study was tested on mice, but did not show significantly elevated rates of lung cancer in exposed animals. Epidemiologic data comes from three different cohort studies of carbon black production workers. Two studies, from the United Kingdom and Germany, with over 1,000 workers in each study group, showed elevated mortality from lung cancer in the carbon black workers. Another study of over 5,000 workers in the United States did not show elevated mortality from lung cancer in the carbon black workers. Newer findings of increased lung cancer mortality in an update from the UK study may suggest that carbon black could be a late-stage carcinogen. However, a more recent and larger study from Germany did not confirm this hypothesis that carbon black acts as a late-stage carcinogen.

In studies employing channel and furnace black, hamsters, mice, guinea pigs, rabbits and monkeys exposed to dusts for 7 hours/day, 5 days/week, at concentrations of 87.4 mg/m³ for channel black and 56.5 mg/m³ for furnace black, no malignancies were observed in any of the animals. Channel black had little if any absorbed polycyclic aromatic hydrocarbons (PAHs) (as benzene extractables) whilst furnace black had 0.28%.

Several findings have strengthened the association between inflammation and cancer and between the particle surface area dose of carbon black and other poorly soluble low toxicity (PSLT) particles and the pulmonary inflammation response in mice and the proinflammatory effects in lung cells *in vitro*. Other evidence suggests that in addition to a cancer mechanism involving indirect genotoxicity through inflammation and oxidative stress, nanoparticles may act as direct carcinogens.

Carbon black appears to act like PSLT particles, which can elicit lung tumours in rats following prolonged exposure to sufficiently high concentrations of particles. Particle surface area dose was found to be most predictive of pulmonary inflammation and tumour response in rats when comparing the dose-response relationships for various types and sizes of PSLT including carbon black. Compared to fine PSLT, much lower concentrations of ultrafine PSLT (e.g. 2.5, 6.5 or 11.5 mg/m³ carbon black and ~10 mg/m³ ultrafine titanium dioxide) were associated with impaired clearance, persistent inflammation, and malignant lung tumours in chronic inhalation studies in rats. Most evidence suggests that carbon black and other PSLT-elicited lung tumours occurs through a secondary genotoxic mechanism, involving chronic inflammation and oxidative stress. Experimental studies have shown that when the particle lung dose reaches a sufficiently high concentration (e.g., mass dose of ~0.5 mg fine-sized PSLT/g lung in rats), the alveolar macrophage-mediated clearance process begins to be impaired (complete impairment occurs at ~10 mg/g lung). Overloading of lung clearance is accompanied by pulmonary inflammation, leading to increased production of reactive oxygen and nitrogen species, depletion of antioxidants and/or impairment of other defense mechanisms, cell injury, cell proliferation, fibrosis, and as seen in rats, induction of mutations and eventually cancer. Rats appear to be more sensitive to carbon black and other PSLT than other rodent species. Although studies in humans have not shown a direct link between inhaled PSLT and lung cancer, many of the steps in the mechanism observed in rats have also been observed in humans who work in dusty jobs, including increased particle lung retention and pulmonary inflammation in workers exposed to coal dust or crystalline silica and elevated lung cancer has been observed in some studies of workers exposed to carbon black, crystalline silica, and diesel exhaust particles. Monkeys exposed to channel black for 1000-1500 hours showed evidence of electrocardiac changes indicative of right atrial and right ventricular strain. These changes increased progressively until after 10,000 hours of exposure, when the changes were marked. The authors

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<p>of this study concluded that there was no significant effect due to prolonged exposure other than those expected from the accumulation of non-toxic dusts in the pulmonary system. Exposure to furnace black produced a similar picture although electrocardiographic change was first observed in monkeys after 2500 hours' exposure and marked atrial and right ventricular strain after 10,000 hours' exposure. The authors concluded that there was no significant effect due to prolonged exposure other than those expected from the accumulation of nontoxic dusts in the pulmonary system. Exposure to furnace black produced a similar picture although electrocardiographic change was first observed in monkeys after 2500 hours exposure and marked atrial and right ventricular strain after 10,000 hours exposure.</p> <p>Chromatographic fractions of oily material extracted from carbon black have been shown to be carcinogenic whilst the unfractionated extracts are not. The activity of some carcinogens appear to be inhibited by carbon black itself.</p> <p>For copper and its compounds (typically copper chloride):</p> <p>Acute toxicity: There are no reliable acute oral toxicity results available. Animal testing shows that skin in exposure to copper may lead to hardness of the skin, scar formation, exudation and reddish changes. Inflammation, irritation and injury of the skin were noted.</p> <p>Repeat dose toxicity: Animal testing shows that very high levels of copper monochloride may cause anaemia.</p> <p>Genetic toxicity: Copper monochloride does not appear to cause mutations in vivo, although chromosomal aberrations were seen at very high concentrations in vitro.</p> <p>Cancer-causing potential: There was insufficient information to evaluate the cancer-causing activity of copper monochloride.</p>							
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lithium fluorophosphate	<table border="1"> <tr> <td>TOXICITY</td><td>IRRITATION</td></tr> <tr> <td></td><td>Skin: adverse effect observed (corrosive)^[1]</td></tr> </table>	TOXICITY	IRRITATION		Skin: adverse effect observed (corrosive) ^[1]		
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<p>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).</p> <p>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.</p> <p>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.</p> <p>Goitrogenic:</p> <p>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</p> <p>Goitrogens include:</p> <ul style="list-style-type: none"> ▶ 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. 							
LITHIUM COBALTATE	<p>WARNING: Inhalation of high concentrations of copper fume may cause "metal fume fever", an acute industrial disease of short duration. Symptoms are tiredness, influenza like respiratory tract irritation with fever.</p> <p>for copper and its compounds (typically copper chloride):</p>						
COPPER	<p>Continued...</p>						

Acute toxicity: There are no reliable acute oral toxicity results available. In an acute dermal toxicity study (OECD TG 402), one group of 5 male rats and 5 groups of 5 female rats received doses of 1000, 1500 and 2000 mg/kg bw via dermal application for 24 hours. The LD50 values of copper monochloride were 2,000 mg/kg bw or greater for male (no deaths observed) and 1,224 mg/kg bw for female. Four females died at both 1500 and 2000 mg/kg bw, and one at 1,000 mg/kg bw. Symptom of the hardness of skin, an exudation of hardness site, the formation of scar and reddish changes were observed on application sites in all treated animals. Skin inflammation and injury were also noted. In addition, a reddish or black urine was observed in females at 2,000, 1,500 and 1,000 mg/kg bw. Female rats appeared to be more sensitive than male based on mortality and clinical signs.

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 µg/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 µg/mL without S9 mix. In the presence of the metabolic activation system, significant increases of structural aberrations were observed at 50 and 70 µg/mL and significant increases of numerical aberrations were observed at 70 µg/mL. In an in vivo mammalian erythrocyte micronucleus assay, all animals dosed (15 - 60 mg/kg bw) with copper monochloride exhibited similar PCE/(PCE+NCE) ratios and MNPCE frequencies compared to those of the negative control animals. Therefore copper monochloride is not an in vivo mutagen.

Carcinogenicity: there was insufficient information to evaluate the carcinogenic activity of copper monochloride.

Reproductive and developmental toxicity: In the combined repeated dose toxicity study with the reproduction/developmental toxicity screening test (OECD TG 422), copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39-51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL of copper monochloride for fertility toxicity was 80 mg/kg bw/day for the parental animals. No treatment-related effects were observed on the reproductive organs and the fertility parameters assessed. For developmental toxicity the NOAEL was 20 mg/kg bw/day. Three of 120 pups appeared to have icterus at birth; 4 of 120 pups appeared runted at the highest dose tested (80 mg/kg bw/day).

ETHYLENE CARBONATE

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.

for ethylene carbonate

Mammalian toxicity: Reliable acute toxicity tests are available on ethylene carbonate. Ethylene carbonate is practically nontoxic following acute oral exposure in a test that meets OECD and EPA test guidelines; the LD50 is >5000 mg/kg. The dermal LD50 is >2000 mg/kg, in a test that meets OECD and EPA test guidelines.

Ethylene carbonate is rapidly metabolized to ethylene glycol. Following gavage administration to rats, ethylene carbonate is rapidly converted into ethylene glycol; the half-life for disappearance of ethylene carbonate from blood was 0.25 hours. As a result, the mammalian toxicity of ethylene carbonate is nearly identical to that of ethylene glycol for endpoints where both have been tested.

Ethylene carbonate was mixed in the diet of 26 male and 26 female Crl: CD(SD) rats for 18 months at concentrations of 25,000 ppm for males and females and 50,000 ppm for females; males were also fed 50,000 ppm for 42 weeks, and 40,000 ppm for 16 weeks. Survivors were observed to 24 months. Compound intake (mg/kg/day) was not reported, but is estimated to be approximately 250 and 500 mg/kg/day. No toxic effects were found in females, but increased mortality was seen in males at both dose levels. No high-dose males survived week 60 and only 10 low-dose males survived to week 78. Males had severe nephrotoxicity, characteristic of ethylene glycol toxicity.

The following *in vitro* genotoxicity tests were conducted on ethylene carbonate, without indications of genotoxicity: an Ames mutagenicity assay, an unscheduled DNA synthesis assay using rat hepatocytes, and a cell transformation assay using BALB/3T3 cells. No *in vivo* genotoxicity studies on ethylene carbonate were found; however, ethylene glycol has been tested and was negative in a rat dominant lethal assay.

Gavage administration of ethylene carbonate to pregnant rats days 6-15 of gestation resulted in systemic toxicity at doses of 3000 mg/kg/day, including post-dose salivation. The NOAEL for maternal toxicity was 1500 mg/kg/day. Similar to ethylene glycol, there were increased soft tissue (hydrocephalus, umbilical herniation, gasteroschisis, cleft palate, misshapen and compressed stomach) and skeletal malformations at 3000 mg/kg/day, but not at 1500 mg/kg/day.

For ethylene glycol:

Ethylene glycol is quickly and extensively absorbed throughout the gastrointestinal tract. Limited information suggests that it is also absorbed through the airways; absorption through skin is apparently slow. Following absorption, it is distributed throughout the body. In humans, it is initially metabolized by alcohol dehydrogenase to form glycoaldehyde, which is rapidly converted to glycolic acid and glyoxal. These breakdown products are oxidized to glyoxylate, which may be further metabolized to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate carbon dioxide, which is one of the major elimination products of ethylene glycol. In addition to exhaled carbon dioxide, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination is rapid and occurs within a few hours.

Respiratory effects: Respiratory system involvement occurs 12-24 hours after swallowing sufficient amounts of ethylene glycol. Symptoms include hyperventilation, shallow rapid breathing, and generalized swelling of the lungs with calcium oxalate deposits occasionally appearing in the lungs. Respiratory system involvement appears to be dose-dependent and occurs at the same time as cardiovascular changes. Later, there may be other changes compatible with adult respiratory distress syndrome (ARDS). Swelling of the lung can be a result of heart failure, ARDS, or aspiration of stomach contents. Symptoms related to acidosis such as fast or excessive breathing are frequently observed; however, major symptoms such as swelling of the lung and inflammation of the bronchi and lungs are relatively rare, and are usually seen only in extreme poisoning.

Cardiovascular effects: Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of ethylene glycol poisoning by swallowing, which is 12-24 hours after acute exposure. The symptoms of poisoning involving the heart include increased heart rate, heart enlargement and ventricular gallop. There may also be high or low blood pressure, which may progress to cardiogenic shock. In lethal cases, inflammation of the heart muscle has been observed at autopsy. Cardiovascular involvement appears to be rare and usually seen after swallowing higher doses of ethylene glycol. In summary, acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.

Gastrointestinal effects: Common early acute effects of swallowing ethylene glycol include nausea, vomiting with or without blood, heartburn and abdominal cramping and pain. One patient showed intermittent diarrhea and pain, and after surgery, deposition of oxalate crystals was shown to have occurred.

Musculoskeletal effects: Reported musculoskeletal effects in cases of acute ethylene glycol poisoning include diffuse muscle tenderness and pain, associated with high levels of creatinine in the blood, and jerks and contractions associated with low calcium.

Liver effects: Autopsies carried out on people who died following acute ethylene glycol poisoning showed deposition of calcium oxalate in the liver as well as hydropic and fatty degeneration and cell death (necrosis) of the liver.

Kidney effects: Adverse kidney effects are seen during the third stage of ethylene glycol poisoning, 2-3 days after acute exposure. Calcium oxalate crystals are deposited in the tubules and are seen in the urine. There may also be degeneration and death of tubule cells, and inflammation of the tubule interstitium. If untreated, the degree of kidney damage progresses and leads to blood and protein in the urine, decreased kidney function, reduction in urine output and ultimately, kidney failure. With adequate supportive therapy, kidney function can return to normal or near normal.

Metabolic effects: Metabolic changes can occur within 12 hours of exposure to ethylene glycol. There may be metabolic acidosis, caused by accumulation of glycolic acid in the blood and therefore a reduction in blood pH. The anion gap is increased, due to increased unmeasured anions (mainly glycolate).

Lithium-ion polymer battery in LED mouthpiece

	<p>Effects on the nervous system: Adverse reactions involving the nervous system are among the first symptoms to appear in humans after ethylene glycol is swallowed. These early effects are also the only symptoms caused by unmetabolised ethylene glycol. Together with metabolic effects (see above), they occur from 0.5-12 hours after exposure and are considered to be part of the first stage in ethylene glycol poisoning. Inco-ordination, slurred speech, confusion and sleepiness are common in the early stages, as are irritation, restlessness and disorientation. Later, there may be effects on cranial nerves (which may be reversible over many months). Swelling of the brain (cerebrum) and crystal deposits of calcium oxalate in the walls of the small blood vessels of the brain were found at autopsy in people who died after acute ethylene glycol poisoning.</p> <p>Reproductive effects: Animal testing showed that ethylene glycol may affect fertility, survival of fetuses and the male reproductive organs.</p> <p>Effects on development: Animal studies indicate that birth defects may occur after exposure in pregnancy; there may also be reduction in foetal weight.</p> <p>Cancer: No studies are known regarding cancer effects in humans or animal, after skin exposure to ethylene glycol.</p> <p>Genetic toxicity: No human studies available, but animal testing results are consistently negative.</p>
LITHIUM COBALTATE & COPPER	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>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.</p>
LITHIUM COBALTATE & GRAPHITE & ALUMINIUM & LITHIUM FLUOROPHOSPHATE	No significant acute toxicological data identified in literature search.
GRAPHITE & ETHYLENE CARBONATE & LITHIUM FLUOROPHOSPHATE	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration 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	✓
Skin Irritation/Corrosion	✗
Serious Eye Damage/Irritation	✓
Respiratory or Skin sensitisation	✓
Mutagenicity	✗
Carcinogenicity	✓
Reproductivity	✗
STOT - Single Exposure	✗
STOT - Repeated Exposure	✓
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
	Not Available	Not Available	Not Available	Not Available	Not Available
lithium cobaltate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	0.029mg/L	2
	EC50	48h	Crustacea	0.241mg/L	2
	EC10(ECx)	168h	Crustacea	0.001mg/L	2
	EC50	96h	Algae or other aquatic plants	23.8mg/l	2
	LC50	96h	Fish	0.8mg/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
	NOEC(ECx)	96h	Fish	>=100mg/l	2
	LC50	96h	Fish	>100mg/l	2
copper	Endpoint	Test Duration (hr)	Species	Value	Source
	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
	NOEC(ECx)	48h	Fish	<0.001mg/L	4
aluminium	EC50	96h	Fish	0.003mg/L	2
	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	0.017mg/L	2

Continued...

	EC50	48h	Crustacea	0.736mg/L	2
	EC50	96h	Algae or other aquatic plants	0.005mg/L	2
	NOEC(ECx)	72h	Algae or other aquatic plants	>100mg/l	1
	LC50	96h	Fish	0.078-0.108mg/l	2
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
lithium fluorophosphate	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	528h	Fish	0.2mg/l	2
	EC50	72h	Algae or other aquatic plants	62mg/l	2
	EC50	48h	Crustacea	98mg/l	2
	EC50	96h	Algae or other aquatic plants	43mg/l	2
	LC50	96h	Fish	42mg/l	2

Legend:

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

May cause long-term adverse effects in the aquatic environment.
DO NOT discharge into sewer or waterways.

Persistence and degradability

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

Bioaccumulative potential

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

Mobility in soil

Ingredient	Mobility
ethylene carbonate	LOW (Log KOC = 9.168)

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. ► 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.
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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

Continued...

Lithium-ion polymer battery in LED mouthpiece

HAZCHEM	2Y
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Land transport (UN)

14.1. UN number or ID number	3481	
14.2. UN proper shipping name	LITHIUM ION BATTERIES PACKED WITH EQUIPMENT (including lithium ion polymer batteries)	
14.3. Transport hazard class(es)	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; 360; 376; 377; 384; 387; 390
	Limited quantity	0

Air transport (ICAO-IATA / DGR)

14.1. UN number	3481	
14.2. UN proper shipping name	Lithium ion batteries packed with equipment (including lithium ion polymer batteries)	
14.3. Transport hazard class(es)	ICAO/IATA Class	9
	ICAO / IATA Subsidiary Hazard	Not Applicable
	ERG Code	12FZ
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Special provisions	A88 A99 A154 A164 A181 A185 A213 A802
	Cargo Only Packing Instructions	966
	Cargo Only Maximum Qty / Pack	35 kg
	Passenger and Cargo Packing Instructions	966
	Passenger and Cargo Maximum Qty / Pack	5 kg
	Passenger and Cargo Limited Quantity Packing Instructions	Forbidden
	Passenger and Cargo Limited Maximum Qty / Pack	Forbidden

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	3481	
14.2. UN proper shipping name	LITHIUM ION BATTERIES PACKED WITH EQUIPMENT (including lithium ion polymer batteries)	
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	188 230 310 348 360 376 377 384 387 390
	Limited Quantities	0

14.7. Maritime transport in bulk according to IMO instruments

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

Not Applicable

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

Product name	Group
lithium cobaltate	Not Available
graphite	Not Available
copper	Not Available
aluminium	Not Available
ethylene carbonate	Not Available
lithium fluorophosphate	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
lithium cobaltate	Not Available
graphite	Not Available

Continued...

Lithium-ion polymer battery in LED mouthpiece

Product name	Ship Type
copper	Not Available
aluminium	Not Available
ethylene carbonate	Not Available
lithium fluorophosphate	Not Available

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

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

HSR Number	Group Standard
HSR002504	Additives Process Chemicals and Raw Materials Acutely Toxic Carcinogenic Group Standard 2020
HSR002613	Metal Industry Products Acutely Toxic Carcinogenic Group Standard 2020
HSR002625	N.O.S. Acutely Toxic Carcinogenic Group Standard 2020
HSR002671	Surface Coatings and Colourants Acutely Toxic Carcinogenic Group Standard 2020
HSR100425	Pharmaceutical Active Ingredients Group Standard 2020
HSR002592	Industrial and Institutional Cleaning Products Acutely Toxic Carcinogenic 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 cobaltate is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
 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)

graphite is found on the following regulatory lists

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)

copper is found on the following regulatory lists

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
 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 Land Transport Rule: Dangerous Goods 2005 - Schedule 1 Quantity limits for dangerous goods
 New Zealand Workplace Exposure Standards (WES)

aluminium is found on the following regulatory lists

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

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)

lithium fluorophosphate is found on the following regulatory lists

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
 New Zealand Workplace Exposure Standards (WES)

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)
6.1B	250 kg or 250 L	500 kg or 500 L

Certified Handler

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

Class of substance	Quantities
6.1B	Any quantity

Refer Group Standards for further information

Continued...

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
6.1B	120	0,1	0,5	
6.5A or 6.5B	120	1	3	

Tracking Requirements

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

- Refer to the regulation for more information

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (lithium fluorophosphate)
Canada - NDSL	No (lithium cobaltate; graphite; copper; aluminium; ethylene carbonate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (graphite; copper; aluminium)
Korea - KECL	Yes
New Zealand - NZIoC	No (lithium fluorophosphate)
Philippines - PICCS	No (lithium cobaltate)
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	No (lithium cobaltate; ethylene carbonate; lithium fluorophosphate)
Vietnam - NCI	Yes
Russia - FBEPH	No (lithium cobaltate; lithium fluorophosphate)
UAE - Control List (Banned/Restricted Substances)	No (lithium cobaltate; copper; ethylene carbonate; 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	10/03/2023
Initial Date	28/07/2020

SDS Version Summary

Version	Date of Update	Sections Updated
3.1	28/08/2020	Firefighting measures - Fire Fighter (fire/explosion hazard), Firefighting measures - Fire Fighter (fire incompatibility), Handling and storage - Handling Procedure, Transport Information
4.1	10/03/2023	Classification change due to full database hazard calculation/update.

Other information

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

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

Definitions and abbreviations

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

Continued...

- IBC: International Bulk Chemical Code
- AIIC: Australian Inventory of Industrial Chemicals
- DSL: Domestic Substances List
- NDSL: Non-Domestic Substances List
- IECSC: Inventory of Existing Chemical Substance in China
- EINECS: European INventory of Existing Commercial chemical Substances
- ELINCS: European List of Notified Chemical Substances
- NLP: No-Longer Polymers
- ENCS: Existing and New Chemical Substances Inventory
- KECL: 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

The information contained in the Safety Data Sheet is based on data considered to be accurate, however, no warranty is expressed or implied regarding the accuracy of the data or the results to be obtained from the use thereof.

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