

Panasonic Lithium-ion Batteries (All Sizes)

Master Instruments Pty Ltd

Chemwatch Hazard Alert Code: 1

Chemwatch: 36-8109

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Safety Data Sheet according to WHS and ADG requirements

L.GHS.AUS.EN

SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	Panasonic Lithium-ion Batteries (All Sizes)
Synonyms	Cylindrical and Prismatic Lithium-ion batteries
Other means of identification	Not Available

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

Relevant identified uses	Battery. NOTE: Chemical materials are stored in sealed metal case. The toxic properties of the electrode materials are hazardous only if the materials are released by damaging the cell or if exposed to fire. The sealed Alkaline battery is not hazardous in normal use. The MSDS Risk codes and the chemical hazards are related to the leaked battery contents.
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Details of the supplier of the safety data sheet

Registered company name	Master Instruments Pty Ltd
Address	13 Sheridan Close Milperra NSW 2214 Australia
Telephone	+61 2 9519 1200
Fax	+612 9519 4604
Website	Not Available
Email	vic@master-instruments.com.au

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	1800 039 008 (24Hrs)
Other emergency telephone numbers	+61 2 9186 1132 (24hrs)

CHEMWATCH EMERGENCY RESPONSE

Primary Number	Alternative Number 1	Alternative Number 2
1800 039 008	+61 2 9186 1132	Not Available

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

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification	Not Applicable

Label elements

Hazard pictogram(s)	Not Applicable
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SIGNAL WORD

NOT APPLICABLE

Hazard statement(s)

Not Applicable

Precautionary statement(s) Prevention

Not Applicable

Precautionary statement(s) Response

Not Applicable

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

Not Applicable

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
		sealed metal container with
12057-24-8		<u>lithium oxide</u>
1313-99-1		<u>nickel oxide</u>
1313-13-9		<u>manganese dioxide</u>
1307-96-6		<u>cobalt (II) oxide</u>
7782-42-5		<u>graphite</u>
96-49-1		<u>ethylene carbonate</u>
105-58-8		<u>diethyl carbonate</u>
21324-40-3		<u>lithium fluorophosphate</u>

SECTION 4 FIRST AID MEASURES**Description of first aid measures**

Eye Contact	<ul style="list-style-type: none"> ▸ Generally not applicable. If content come in contact with eye, flush with water for 15 minutes without rubbing and immediately contact a physician.
Skin Contact	If skin contact occurs: <ul style="list-style-type: none"> ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	Remove patient to fresh air and seek medical attention.
Ingestion	<ul style="list-style-type: none"> ▸ For advice, contact a Poisons Information Centre or a doctor.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 FIREFIGHTING MEASURES**Extinguishing media**

Use dry chemical powder, alcohol-resistant foam, carbon dioxide, or water as a fine spray.

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.
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Advice for firefighters

Fire Fighting	Slight hazard when exposed to heat, flame and oxidisers. <ul style="list-style-type: none"> ▸ Use fire fighting procedures suitable for surrounding area.
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	<ul style="list-style-type: none"> ▶ 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.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Non combustible. ▶ Not considered to be a significant fire risk. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ May emit acrid smoke. May emit corrosive and poisonous fumes.
HAZCHEM	Not Applicable

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	Clean up all spills immediately. Avoid contact with skin and eyes. Place in suitable containers 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	Use good occupational work practice. Observe manufacturer's storage and handling recommendations contained within this SDS. Avoid physical damage to containers. Do not short-circuit, crush, incinerate or disassemble battery.
Other information	<ul style="list-style-type: none"> ▶ Keep dry. ▶ Store under cover. ▶ Protect containers against physical damage. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. Keep out of reach of children. Store out of direct sunlight <ul style="list-style-type: none"> ▶ Store away from incompatible materials.

Conditions for safe storage, including any incompatibilities

Suitable container	▶ Packaging as recommended by manufacturer.
Storage incompatibility	▶ Avoid reaction with oxidising agents

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
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Australia Exposure Standards	manganese dioxide	Manganese, dust & compounds (as Mn)	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	graphite	Graphite (all forms except fibres) (respirable dust) (natural & synthetic)	3 mg/m3	Not Available	Not Available	Not Available


EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
lithium oxide	Lithium oxide	0.091 mg/m3	1 mg/m3	6 mg/m3
nickel oxide	Nickel oxide; (Nickel(II) oxide)	0.76 mg/m3	220 mg/m3	1,300 mg/m3
manganese dioxide	Manganese dioxide	4.7 mg/m3	7.9 mg/m3	690 mg/m3
manganese dioxide	Manganese oxide; (Manganese tetroxide)	4.2 mg/m3	6.9 mg/m3	41 mg/m3
cobalt (II) oxide	Cobalt(II) oxide	0.076 mg/m3	4.2 mg/m3	25 mg/m3
graphite	Graphite; (Mineral carbon)	6 mg/m3	16 mg/m3	95 mg/m3
ethylene carbonate	Glycol carbonate; (Ethylene carbonate)	30 mg/m3	330 mg/m3	2,000 mg/m3
diethyl carbonate	Diethyl carbonate	12 ppm	140 ppm	810 ppm
lithium fluorophosphate	Lithium hexafluorophosphate	7.5 mg/m3	83 mg/m3	500 mg/m3

Ingredient	Original IDLH	Revised IDLH
lithium oxide	Not Available	Not Available
nickel oxide	10 mg/m3	Not Available
manganese dioxide	500 mg/m3	Not Available
cobalt (II) oxide	Not Available	Not Available
graphite	1,250 mg/m3	Not Available
ethylene carbonate	Not Available	Not Available
diethyl carbonate	Not Available	Not Available
lithium fluorophosphate	Not Available	Not Available

MATERIAL DATA

Exposure controls

Appropriate engineering controls	None under normal operating conditions.
Personal protection	
Eye and face protection	None under normal operating conditions. OTHERWISE: ▸ Safety glasses.
Skin protection	See Hand protection below
Hands/feet protection	None under normal operating conditions. OTHERWISE: ▸ Rubber Gloves
Body protection	See Other protection below
Other protection	No special equipment needed when handling small quantities

Respiratory protection

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

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

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

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

^ - Full-face

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

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Batteries.		
Physical state	Manufactured	Relative density (Water = 1)	Not Applicable
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	Not Applicable
Melting point / freezing point (°C)	Not Applicable	Viscosity (cSt)	Not Applicable
Initial boiling point and boiling range (°C)	Not Applicable	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Applicable	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Applicable
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Applicable
Vapour pressure (kPa)	Not Applicable	Gas group	Not Available
Solubility in water (g/L)	Immiscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	Not Applicable	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	Not normally a hazard due to physical form of product. Vapors or fumes released due to burning or large number of leaking battery content may cause respiratory irritation.
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Ingestion	Considered an unlikely route of entry in commercial/industrial environments Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	Not normally a hazard due to physical form of product. Contents with battery contents may cause irritation.
Eye	Not normally a hazard due to physical form of product. Eye contact with the content of an open battery may cause irritation.
Chronic	<p>Not normally a hazard due to physical form of product.</p> <p>Large doses of lithium ion have caused dizziness and prostration and can cause kidney damage if sodium intake is limited. Dehydration, weight-loss, dermatological effects and thyroid disturbances have been reported. Central nervous system effects that include slurred speech, blurred vision, sensory loss, impaired concentration, irritability, lethargy, confusion, disorientation, drowsiness, anxiety, spasticity, delirium, stupor, ataxia (loss of muscle coordination), sedation, fine and gross tremor, giddiness, twitching and convulsions may occur. Diarrhoea, vomiting and neuromuscular effects such as tremor, clonus (rapid contraction and relaxation of muscles) and hyperactive reflexes may occur as a result of repeated exposure to lithium.</p> <p>Acute severe overexposure may affect the kidneys, resulting in renal dysfunction, albuminuria, oliguria and degenerative changes. Cardiovascular effects may also result in cardiac arrhythmias and hypotension.</p> <p>The primary target organ for lithium toxicity is the central nervous system. Lithium is therefore used therapeutically on membrane transport proteins in the central nervous system when treating manic-depression. Lithium is moderately toxic with lethal dose of LiCl in rats of 526-840 mg/kg body weight. After chronic exposure to 1 meq/L decreased brain weight was observed in male offspring. Chemically, lithium resembles sodium, but is more toxic: in humans 5 g LiCl can result in fatal poisoning. In therapeutic doses, damages on the central nervous system and the kidneys have been reported.</p> <p>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.</p> <p>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).</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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</p>

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resulted in a spectrum of inflammatory, fibrotic, and proliferative lesions in the respiratory tract of male and female rats and mice. Squamous metaplasia of the larynx occurred in rats and mice at exposure concentrations of .0.11 mg cobalt/m³, with severity of the lesion increasing with increased cobalt concentration. Hyperplastic lesions of the nasal epithelium occurred in rats at concentrations of .0.11 mg cobalt/m³, and in mice at concentrations of .0.38 mg cobalt/m³. Both sexes of rats had greatly increased incidences (>90% incidence) of alveolar lesions at all exposure levels, including inflammatory changes, fibrosis, and metaplasia. Similar changes were seen in mice at all exposure levels, though the changes in mice were less severe.

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

The chemicals in this product are contained in a sealed case and exposure does not occur during normal handling and use.

Panasonic Lithium-ion Batteries (All Sizes)	TOXICITY	IRRITATION
	Not Available	Not Available
lithium oxide	TOXICITY	IRRITATION
	Not Available	Not Available
nickel oxide	TOXICITY	IRRITATION
	Oral (rat) LD50: >5000 mg/kg ^[1]	Not Available
manganese dioxide	TOXICITY	IRRITATION
	Oral (rat) LD50: >3478 mg/kg ^[2]	Not Available
cobalt (II) oxide	TOXICITY	IRRITATION
	Oral (rat) LD50: 202 mg/kg ^[2]	Not Available
graphite	TOXICITY	IRRITATION
	Inhalation (rat) LC50: >2 mg/l4 h ^[1]	Not Available
ethylene carbonate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 20 mg - mild
diethyl carbonate	TOXICITY	IRRITATION
	Oral (rat) LD50: >2000 mg/kg ^[1]	Skin (rabbit): 660 mg - moderate
lithium fluorophosphate	TOXICITY	IRRITATION
	Oral (rat) LD50: 50-300 mg/kg ^[1]	Not Available

Legend: 1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. * Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances

LITHIUM OXIDE	TOXICITY	IRRITATION
	Oral (rat) LD50: 50-300 mg/kg ^[1]	Not Available
COBALT (II) OXIDE	TOXICITY	IRRITATION
	Oral (rat) LD50: 50-300 mg/kg ^[1]	Not Available
ETHYLENE CARBONATE	TOXICITY	IRRITATION
	Oral (rat) LD50: 50-300 mg/kg ^[1]	Not Available

The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

The material may produce respiratory tract irritation. Symptoms of pulmonary irritation may include coughing, wheezing, laryngitis, shortness of breath, headache, nausea, and a burning sensation.

Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence).

The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties.

WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. Tumorigenic by RTECS criteria

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

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for ethylene carbonate

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

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

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

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

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

For ethylene glycol:

Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol

dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO₂, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO₂, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

Respiratory Effects. Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning. The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning. Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases).

Cardiovascular Effects. Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12- 24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol. Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases. Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.

Gastrointestinal Effects. Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition.

Musculoskeletal Effects. Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tenderness and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia.

Hepatic Effects. Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol.

Renal Effects. Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, anuria, and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near normal renal function can return with adequate supportive therapy.




Metabolic Effects. One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication

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	<p>is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate).</p> <p>Neurological Effects: Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion.</p> <p>Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months.</p> <p>Reproductive Effects: Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration.</p> <p>Developmental Effects: The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embryotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight.</p> <p>Cancer: No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol.</p> <p>Genotoxic Effects: Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available <i>in vivo</i> and <i>in vitro</i> laboratory studies provide consistently negative genotoxicity results for ethylene glycol.</p>
DIETHYL CARBONATE	Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis). Equivocal tumorigen by RTECS criteria
LITHIUM OXIDE & GRAPHITE & ETHYLENE CARBONATE & DIETHYL CARBONATE & LITHIUM FLUOROPHOSPHATE	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
LITHIUM OXIDE & ETHYLENE CARBONATE	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
NICKEL OXIDE & COBALT (II) OXIDE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
MANGANESE DIOXIDE & GRAPHITE & LITHIUM FLUOROPHOSPHATE	No significant acute toxicological data identified in literature search.

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

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Legend:  – Data available but does not fill the criteria for classification
 – Data available to make classification
 – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Panasonic Lithium-ion Batteries (All Sizes)	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
lithium oxide	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
nickel oxide	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.4mg/L	2
	EC50	48	Crustacea	0.1455mg/L	2
	EC50	72	Algae or other aquatic plants	0.0407mg/L	2
	NOEC	72	Algae or other aquatic plants	0.0035mg/L	2
manganese dioxide	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	EC50	48	Crustacea	>0.0219mg/L	2
	NOEC	48	Crustacea	0.0219mg/L	2
cobalt (II) oxide	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1.406mg/L	2
	EC50	48	Crustacea	2.618mg/L	2
	EC50	72	Algae or other aquatic plants	0.144mg/L	2
	NOEC	168	Algae or other aquatic plants	0.0018mg/L	2
graphite	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
ethylene carbonate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	49000mg/L	2
diethyl carbonate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	EC50	48	Crustacea	>74.16mg/L	2
	EC50	72	Algae or other aquatic plants	>57.29mg/L	2
	NOEC	72	Algae or other aquatic plants	>57.29mg/L	2
lithium fluorophosphate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	42mg/L	2
	NOEC	168	Crustacea	2.55mg/L	2
Legend:	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data				

DO NOT discharge into sewer or waterways.

Persistence and degradability

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

Continued...

Bioaccumulative potential

Ingredient	Bioaccumulation
ethylene carbonate	LOW (LogKOW = -0.3388)
diethyl carbonate	LOW (LogKOW = 1.21)

Mobility in soil

Ingredient	Mobility
ethylene carbonate	LOW (KOC = 9.168)
diethyl carbonate	LOW (KOC = 28.08)

SECTION 13 DISPOSAL CONSIDERATIONS**Waste treatment methods**

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

Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS**Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS****Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS****Transport in bulk according to Annex II of MARPOL and the IBC code**

Not Applicable

SECTION 15 REGULATORY INFORMATION**Safety, health and environmental regulations / legislation specific for the substance or mixture****LITHIUM OXIDE(12057-24-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Inventory of Chemical Substances (AICS)

NICKEL OXIDE(1313-99-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

MANGANESE DIOXIDE(1313-13-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix B (Part 3)

COBALT (II) OXIDE(1307-96-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

GRAPHITE(7782-42-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Inventory of Chemical Substances (AICS)

ETHYLENE CARBONATE(96-49-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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Australia Inventory of Chemical Substances (AICS)

DIETHYL CARBONATE(105-58-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix B (Part 3)

LITHIUM FLUOROPHOSPHATE(21324-40-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

National Inventory Status

National Inventory	Status
Australia - AICS	Y
Canada - DSL	N (lithium fluorophosphate)
Canada - NDSL	N (nickel oxide; lithium oxide; diethyl carbonate; manganese dioxide; graphite; cobalt (II) oxide; ethylene carbonate)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	N (graphite; lithium fluorophosphate)
Korea - KECI	Y
New Zealand - NZIoC	N (lithium fluorophosphate)
Philippines - PICCS	Y
USA - TSCA	Y
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Revision Date	05/09/2018
Initial Date	03/09/2013

Other information**Ingredients with multiple cas numbers**

Name	CAS No
nickel oxide	1313-99-1, 11099-02-8
manganese dioxide	1313-13-9, 301678-04-6
cobalt (II) oxide	1307-96-6, 185461-93-2, 186373-01-3

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

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

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
 PC—STEL: Permissible Concentration-Short Term Exposure Limit
 IARC: International Agency for Research on Cancer
 ACGIH: American Conference of Governmental Industrial Hygienists
 STEL: Short Term Exposure Limit
 TEEL: Temporary Emergency Exposure Limit.
 IDLH: Immediately Dangerous to Life or Health Concentrations
 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

Continued...

Panasonic Lithium-ion Batteries (All Sizes)

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