# Panasonic Lithium-ion Batteries (All Sizes)

**Master Instruments Pty Ltd**

Chemwatch Hazard Alert Code: **1**

Chemwatch: **36-8109**
Version No: **3.1.1.1**
Safety Data Sheet according to WHS and ADG requirements

Issue Date: **05/09/2018**
Print Date: **06/09/2018**
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## 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. |

### 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 prefered language then please dial 01**

## SECTION 2 HAZARDS IDENTIFICATION

### Classification of the substance or mixture

| | |
|---|---|
| **Poisons Schedule** | Not Applicable |
| **Classification** | Not Applicable |

### Label elements

| | |
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| **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 | | lithium oxide |
| 1313-99-1 | | nickel oxide |
| 1313-13-9 | | manganese dioxide |
| 1307-96-6 | | cobalt (II) oxide |
| 7782-42-5 | | graphite |
| 96-49-1 | | ethylene carbonate |
| 105-58-8 | | diethyl carbonate |
| 21324-40-3 | | lithium fluorophosphate |

## SECTION 4 FIRST AID MEASURES

### Description of first aid measures

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|---|---|
| **Eye Contact** | ▸ Generally not applicable.<br>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:<br>▸ Immediately remove all contaminated clothing, including footwear.<br>▸ Flush skin and hair with running water (and soap if available).<br>▸ Seek medical attention in event of irritation. |
| **Inhalation** | Remove patient to fresh air and seek medical attention. |
| **Ingestion** | ▸ For advice, contact a Poisons Information Centre or a doctor. |

### Indication of any immediate medical attention and special treatment needed

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

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|---|---|
| **Fire Incompatibility** | None known. |

### Advice for firefighters

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| **Fire Fighting** | Slight hazard when exposed to heat, flame and oxidisers.<br>▸ Use fire fighting procedures suitable for surrounding area. |

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|---|---|
| | ▸ **DO NOT** approach containers suspected to be hot.<br>▸ Cool fire exposed containers with water spray from a protected location.<br>▸ If safe to do so, remove containers from path of fire.<br>▸ Equipment should be thoroughly decontaminated after use. |
| **Fire/Explosion Hazard** | ▸ Non combustible.<br>▸ Not considered to be a significant fire risk.<br>▸ Heating may cause expansion or decomposition leading to violent rupture of containers.<br>▸ 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

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|---|---|
| **Minor Spills** | Clean up all spills immediately.<br>Avoid contact with skin and eyes.<br>Place in suitable containers for disposal. |
| **Major Spills** | ▸ Clean up all spills immediately.<br>▸ Wear protective clothing, safety glasses, dust mask, gloves.<br>▸ Secure load if safe to do so. Bundle/collect recoverable product.<br>▸ Use dry clean up procedures and avoid generating dust.<br>▸ Vacuum up (consider explosion-proof machines designed to be grounded during storage and use).<br>▸ Water may be used to prevent dusting.<br>▸ Collect remaining material in containers with covers for disposal.<br>▸ 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

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|---|---|
| **Safe handling** | Use good occupational work practice. Observe manufacturer's storage and handling recommendations contained within this SDS.<br>Avoid physical damage to containers.<br>\|Do not short-circuit, crush, incinerate or disassemble battery. |
| **Other information** | ▸ Keep dry.<br>▸ Store under cover.<br>▸ Protect containers against physical damage.<br>▸ Observe manufacturer's storage and handling recommendations contained within this SDS.<br>Keep out of reach of children.<br>Store out of direct sunlight<br>▸ Store away from incompatible materials. |

### Conditions for safe storage, including any incompatibilities

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|---|---|
| **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.<br>**OTHERWISE:**<br>‣ Safety glasses. |
| **Skin protection** | See Hand protection below |
| **Hands/feet protection** | None under normal operating conditions.<br>**OTHERWISE:**<br>‣ 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

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| **Appearance** | Batteries. |

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|---|---|---|---|
| **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

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|---|---|
| **Inhaled** | Not normally a hazard due to physical form of product.<br>\|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<br>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.<br>|Contents with battery contents may cause irritation. |
| **Eye** | Not normally a hazard due to physical form of product.<br>|Eye contact with the content of an open battery may cause irritation. |
| **Chronic** | Not normally a hazard due to physical form of product.<br>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.<br>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.<br>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.<br>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.<br>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).<br>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.<br>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<br>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.<br>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.<br>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.<br>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.<br>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.<br>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.<br>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 |

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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/3, and in mice at concentrations of .0.38 mg cobalt/m3. Both sexes of rats had greatly increased incidences (>90% incidence) of alveolar lesions at all exposure levels, including inflammatory changes, fibrosis, and metaplasia. Similar changes were seen in mice at all exposure levels, though the changes in mice were less severe.<br>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.<br>\|The chemicals in this product are contained in a sealed case and exposure does not occur during normal handling and use. |

| | TOXICITY | IRRITATION |
|---|---|---|
| **Panasonic Lithium-ion Batteries (All Sizes)** | 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 |
| | Oral (rat) LD50: >2000 mg/kg[2] | |
| **ethylene carbonate** | TOXICITY | IRRITATION |
| | dermal (rat) LD50: >2000 mg/kg[1] | Eye (rabbit): 20 mg - mild |
| | Oral (rat) LD50: >2000 mg/kg[1] | Skin (rabbit): 660 mg - moderate |
| **diethyl carbonate** | TOXICITY | IRRITATION |
| | Not Available | Not Available |
| **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* | |

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| **LITHIUM OXIDE** | The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.<br>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.<br>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).<br><br>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. |
| **COBALT (II) OXIDE** | **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.<br>Tumorigenic by RTECS criteria |
| **ETHYLENE CARBONATE** | 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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| | |
|---|---|
| | 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).<br>**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.<br>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.<br>**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.<br>**Developmental Effects:** The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embyrotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight.<br>**Cancer:** No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol.<br>**Genotoxic Effects**: Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available *in vivo* and *in vitro* laboratory studies provide consistently negative genotoxicity results for ethylene glycol. |
| **DIETHYL CARBONATE** | Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).<br>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.<br>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.<br>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** | ⊘ |

**Continued...**

***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** | ▸ Recycle wherever possible or consult manufacturer for recycling options.<br>▸ Consult State Land Waste Management Authority for disposal.<br>▸ Bury residue in an authorised landfill.<br>▸ Recycle containers if possible, or dispose of in an authorised landfill. |

## 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
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
Australia Inventory of Chemical Substances (AICS)

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

Australia Exposure Standards
Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
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
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
Australia Inventory of Chemical Substances (AICS)

**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**

**Continued...**

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*<br>*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...**

**end of SDS**