Lithium-ion battery in equipment – Radii Plus and Radii Cal

SDI Limited

Version No: **3.1.1.1**
Safety Data Sheet according to WHS and ADG requirements

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

Product Identifier

Product name	Lithium-ion battery in equipment – Radii Plus and Radii Cal
Synonyms	Lithium-ion (Li-ion) battery pack. Nominal voltage: 7.4V, Rated Capacity: 1550mAh, Wh rating: 11.47 Wh
Proper shipping name	LITHIUM ION BATTERIES CONTAINED IN EQUIPMENT or LITHIUM ION BATTERIES PACKED WITH EQUIPMENT
Other means of identification	Not Available

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

Relevant identified uses	Battery in Radii Plus and Radii Cal, to be used as dental curing lights. Potentially hazardous materials are sealed and contained in equipment. Equipment is packed in strong outer packaging to withstand normal handling and use. Exposure could occur if the equipment has been exposed to high temperatures (>125°C), battery or cells have been opened, crushed, dissembled or burned.

Details of the supplier of the safety data sheet

Registered company name	SDI Limited	SDI Brazil Industria E Comercio Ltda	SDI Germany GmbH
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Emergency telephone number

Association / Organisation	SDI Limited	Not Available	Not Available
Emergency telephone numbers	+61 3 8727 7111	Not Available	Not Available
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Association / Organisation	Not Available
Emergency telephone numbers	+61 3 8727 7111
Other emergency telephone numbers	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

Poisons Schedule	Not Applicable
Classification	Not Applicable

Label elements

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GHS label elements	Not Applicable
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
		Battery Cell contains
12190-79-3	<38	lithium cobaltate
21324-40-3	<3	lithium fluorophosphate
96-49-1	<6	ethylene carbonate
Not Available	<8	chain carbonate
7782-42-5	<20	graphite
7439-92-1	<0.1	lead
7439-97-6	<0.0005	mercury (elemental)
		Note: other 25% includes the below meterials:
		Al (Positive Base Film, Cap, Can, Tab)
		Cu (Negative film base)
		Ni (Tab, Terminal)
		Fe (Terminal)
		Resin (PP, PE, PET) (Separator, Plastic, Parts, Insulator)
		Circuit Module contains
7439-92-1	<0.1	lead
7439-97-6		mercury (elemental)
7440-47-3		chromium
7440-43-9		cadmium
		plastic case and Si2O
		Plastic Parts and Paints contains
25971-63-5	>81	bisphenol A/ phosgene polymer
Not Available	<12	flame retardant
Not Available	<7	elastomer

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs: ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Seek medical attention.

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Ingestion	▸ Not considered a normal route of entry. ▸ For advice, contact a Poisons Information Centre or a doctor at once. ▸ Urgent hospital treatment is likely to be needed. ▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Transport to hospital or doctor without delay.

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.

Advice for firefighters

Fire Fighting	Slight hazard when exposed to heat, flame and oxidisers. ▸ Use fire fighting procedures suitable for surrounding area. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire. ▸ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	▸ The material is not readily combustible under normal conditions. ▸ However, it will break down under fire conditions and the organic component may burn. ▸ Not considered to be a significant fire risk. ▸ Heat may cause expansion or decomposition with violent rupture of containers. ▸ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke.

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

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

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.
Other information	▸ Store away from incompatible materials. ▸ Keep dry. ▸ Store under cover. ▸ Protect containers against physical damage. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. Store out of direct sunlight Keep away from heat and naked flames.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ **DO NOT** repack. Use containers supplied by manufacturer only.
Storage incompatibility	▸ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

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OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	graphite	Graphite (all forms except fibres) (respirable dust)(natural & synthetic)	3 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	lead	Lead, inorganic dusts & fumes (as Pb)	0.15 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	lead	Cadmium and compounds (as Cd)	0.01 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	mercury (elemental)	Mercury, elemental vapour (as Hg)	0.025 mg/m3 / 0.003 ppm	Not Available	Not Available	Not Available
Australia Exposure Standards	lead	Lead, inorganic dusts & fumes (as Pb)	0.15 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	lead	Cadmium and compounds (as Cd)	0.01 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	mercury (elemental)	Mercury, elemental vapour (as Hg)	0.025 mg/m3 / 0.003 ppm	Not Available	Not Available	Not Available
Australia Exposure Standards	chromium	Chromium (metal)	0.5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	cadmium	Cadmium and compounds (as Cd)	0.01 mg/m3	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
ethylene carbonate	Glycol carbonate; (Ethylene carbonate)	30 mg/m3	330 mg/m3	2000 mg/m3
graphite	Graphite; (Mineral carbon)	2 mg/m3	2 mg/m3	95 mg/m3
lead	Lead	0.15 mg/m3	120 mg/m3	700 mg/m3
mercury (elemental)	Mercury vapor	0.15 mg/m3	Not Available	Not Available
lead	Lead	0.15 mg/m3	120 mg/m3	700 mg/m3
mercury (elemental)	Mercury vapor	0.15 mg/m3	Not Available	Not Available
chromium	Chromium	1.5 mg/m3	17 mg/m3	99 mg/m3
cadmium	Cadmium	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
lithium cobaltate	Not Available	Not Available
lithium fluorophosphate	Not Available	Not Available
ethylene carbonate	Not Available	Not Available
chain carbonate	Not Available	Not Available
graphite	N.E. mg/m3 / N.E. ppm	1,250 mg/m3
lead	700 mg/m3	100 mg/m3
mercury (elemental)	10 mg/m3 / 28 mg/m3	2 mg/m3 / 10 mg/m3
lead	700 mg/m3	100 mg/m3
mercury (elemental)	10 mg/m3 / 28 mg/m3	2 mg/m3 / 10 mg/m3
chromium	N.E. mg/m3 / N.E. ppm	250 mg/m3
cadmium	50 mg/m3 / 9 mg/m3	9 mg/m3 / 9 [Unch] mg/m3
bisphenol A/ phosgene polymer	Not Available	Not Available
flame retardant	Not Available	Not Available
elastomer	Not Available	Not Available

MATERIAL DATA

Exposure controls

Appropriate engineering controls	None under normal operating conditions. Provide adequate ventilation in warehouse or closed storage areas.
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

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Body protection	See Other protection below
Other protection	None under normal operating conditions. **OTHERWISE:** ▸ Overalls. ▸ PVC Apron. ▸ PVC protective suit may be required if exposure severe. ▸ Eyewash unit. ▸ Ensure there is ready access to a safety shower.
Thermal hazards	Not Available

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.
Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	AHG-AUS P2	-	AHG-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AHG-AUS / Class 1 P2	-
up to 100 x ES	-	AHG-2 P2	AHG-PAPR-2 P2 ^

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Solid articles, insoluble in water.

Physical state	Solid	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Applicable
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water (g/L)	Immiscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**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.
Ingestion	Considered an unlikely route of entry in commercial/industrial environments Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. Ingestion may result in nausea, abdominal irritation, pain and vomiting

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Skin Contact	Not normally a hazard due to physical form of product.
Eye	Not normally a hazard due to physical form of product.
Chronic	Not normally a hazard due to physical form of product.

	TOXICITY	IRRITATION
Lithium-ion battery in equipment – Radii Plus and Radii Cal	Not Available	Not Available
lithium cobaltate	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
lithium fluorophosphate	**TOXICITY**	**IRRITATION**
	Oral (rat) LD50: 50-300 mg/kg[1]	Not Available
ethylene carbonate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	[CCInfo]*
	Oral (rat) LD50: >2000 mg/kg[1]	Eye (rabbit): 20 mg - mild
		Skin (rabbit): 660 mg - moderate
graphite	**TOXICITY**	**IRRITATION**
	Inhalation (rat) LC50: >2 mg/L4 h[1]	Not Available
	Oral (rat) LD50: >2000 mg/kg**[2]	
lead	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Nil Reported
	Inhalation (rat) LC50: >5.05 mg/l4 h[1]	
	Oral (rat) LD50: >2000 mg/kg[1]	
mercury (elemental)	**TOXICITY**	**IRRITATION**
	Oral (rat) LD50: >9.2 mg/kg[1]	(Source: RTECS)
		Nil reported
lead	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Nil Reported
	Inhalation (rat) LC50: >5.05 mg/l4 h[1]	
	Oral (rat) LD50: >2000 mg/kg[1]	
mercury (elemental)	**TOXICITY**	**IRRITATION**
	Oral (rat) LD50: >9.2 mg/kg[1]	(Source: RTECS)
		Nil reported
chromium	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
cadmium	**TOXICITY**	**IRRITATION**
	Inhalation (monkey) LC50: 0.03 mg/L15 min[1]	Nil reported
	Inhalation (monkey) LC50: 0.0467 mg/L15 min[1]	
	Inhalation (monkey) LC50: 0.204 mg/L15 min[1]	
	Inhalation (monkey) LC50: 0.23 mg/L15 min[1]	
	Inhalation (monkey) LC50: 0.94 mg/L15 min[1]	
	Inhalation (mouse) LC50: >0.00902 mg/L15 min[1]	
	Inhalation (rabbit) LC50: >0.0224 mg/L15 min[1]	
	Inhalation (rat) LC50: 0.025 mg/L/30m[2]	
	Oral (rat) LD50: >63-<259 mg/kg[1]	
bisphenol A/ phosgene polymer	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*	

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LITHIUM COBALTATE	No significant acute toxicological data identified in literature search.
ETHYLENE CARBONATE	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. The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. for ethylene carbonate **Mammalian toxicity:** Reliable acute toxicity tests are available on ethylene carbonate. Ethylene carbonate is practically nontoxic following acute oral exposure in a test that meets OECD and EPA test guidelines; the LD50 is >5000 mg/kg. The dermal LD50 is >2000 mg/kg, in a test that meets OECD and EPA test guidelines. Ethylene carbonate is rapidly metabolized to ethylene glycol. Following gavage administration to rats, ethylene carbonate is rapidly converted into ethylene glycol; the half-life for disappearance of ethylene carbonate from blood was 0.25 hours. As a result, the mammalian toxicity of ethylene carbonate is nearly identical to that of ethylene glycol for endpoints where both have been tested Ethylene carbonate was mixed in the diet of 26 male and 26 female Crl: CD(SD) rats for 18 months at concentrations of 25,000 ppm for males and females and 50,000 ppm for females; males were also fed 50,000 ppm for 42 weeks, and 40,000 ppm for 16 weeks. Survivors were observed to 24 months. Compound intake (mg/kg/day) was not reported, but is estimated to be approximately 250 and 500 mg/kg/day. No toxic effects were found in females, but increased mortality was seen in males at both dose levels. No high-dose males survived week 60 and only 10 low-dose males survived to week 78. Males had severe nephrotoxicity, characteristic of ethylene glycol toxicity. The following *in vitro* genotoxicity tests were conducted on ethylene carbonate, without indications of genotoxicity: an Ames mutagenicity assay, an unscheduled DNA synthesis assay using rat hepatocytes, and a cell transformation assay using BALB/3T3 cells. No *in vivo* genotoxicity studies on ethylene carbonate were found; however, ethylene glycol has been tested and was negative in a rat dominant lethal assay. Gavage administration of ethylene carbonate to pregnant rats days 6-15 of gestation resulted in systemic toxicity at doses of 3000 mg/kg/day, including post-dose salivation. The NOAEL for maternal toxicity was 1500 mg/kg/day. Similar to ethylene glycol, there were increased soft tissue (hydrocephalus, umbilical herniation, gastroschisis, cleft palate, misshapen and compressed stomach) and skeletal malformations at 3000 mg/kg/day, but not at 1500 mg/kg/day. For ethylene glycol: Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol. dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO_2, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO_2, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested. **Respiratory Effects.** Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases). **Cardiovascular Effects.** Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12- 24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol. Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases.Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown. **Gastrointestinal Effects.** Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition. **Musculoskeletal Effects.** Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tenderness and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia. **Hepatic Effects.** Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. **Renal Effects.** Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, anuria , and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near normal renal function can return with adequate supportive therapy. **Metabolic Effects.** One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate). **Neurological Effects:** Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion. Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar

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	nerves and are reversible over many months. **Reproductive Effects:** Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration. **Developmental Effects:** The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embyrotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight. **Cancer:** No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol. **Genotoxic Effects**: Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available *in vivo* and *in vitro* laboratory studies provide consistently negative genotoxicity results for ethylene glycol.
CHROMIUM	For chrome(III) and other valence states (except hexavalent): For inhalation exposure, all trivalent and other chromium compounds are treated as particulates, not gases. The mechanisms of chromium toxicity are very complex, and although many studies on chromium are available, there is a great deal of uncertainty about how chromium exerts its toxic influence. Much more is known about the mechanisms of hexavalent chromium toxicity than trivalent chromium toxicity. There is an abundance of information available on the carcinogenic potential of chromium compounds and on the genotoxicity and mutagenicity of chromium compounds in experimental systems. The consensus from various reviews and agencies is that evidence of carcinogenicity of elemental, divalent, or trivalent chromium compounds is lacking. Epidemiological studies of workers in a number of industries (chromate production, chromate pigment production and use, and chrome plating) conclude that while occupational exposure to hexavalent chromium compounds is associated with an increased risk of respiratory system cancers (primarily bronchogenic and nasal), results from occupational exposure studies to mixtures that were mainly elemental and trivalent (ferrochromium alloy worker) were inconclusive. Studies in leather tanners, who were exposed to trivalent chromium were consistently negative. In addition to the lack of direct evidence of carcinogenicity of trivalent or elemental chromium and its compounds, the genotoxic evidence is overwhelmingly negative. The lesser potency of trivalent chromium relative to hexavalent chromium is likely related to the higher redox potential of hexavalent chromium and its greater ability to enter cells. enter cells The general inability of trivalent chromium to traverse membranes and thus be absorbed or reach peripheral tissue in significant amounts is generally accepted as a probable explanation for the overall absence of systemic trivalent chromium toxicity. Elemental and divalent forms of chromium are not able to traverse membranes readily either. This is not to say that elemental, divalent, or trivalent chromium compounds cannot traverse membranes and reach peripheral tissue, the mechanism of absorption is simply less efficient in comparison to absorption of hexavalent chromium compounds. Hexavalent chromium compounds exist as tetrahedral chromate anions, resembling the forms of other natural anions like sulfate and phosphate which are permeable across nonselective membranes. Trivalent chromium forms octahedral complexes which cannot easily enter though these channels, instead being absorbed via passive diffusion and phagocytosis. Although trivalent chromium is less well absorbed than hexavalent chromium, workers exposed to trivalent compounds have had detectable levels of chromium in the urine at the end of a workday. Absorbed chromium is widely distributed throughout the body via the bloodstream, and can reach the foetus. Although there is ample in vivo evidence that hexavalent chromium is efficiently reduced to trivalent chromium in the gastrointestinal tract and can be reduced to the trivalent form by ascorbate and glutathione in the lungs, there is no evidence that trivalent chromium is converted to hexavalent chromium in biological systems. In general, trivalent chromium compounds are cleared rapidly from the blood and more slowly from the tissues. Although not fully characterized, the biologically active trivalent chromium molecule appears to be chromodulin, also referred to as (GTF). Chromodulin is an oligopeptide complex containing four chromic ions. Chromodulin may facilitate interactions of insulin with its receptor site, influencing protein, glucose, and lipid metabolism. Inorganic trivalent chromium compounds, which do not appear to have insulin-potentiating properties, are capable of being converted into biologically active forms by humans and animals Chromium can be a potent sensitiser in a small minority of humans, both from dermal and inhalation exposures. The most sensitive endpoint identified in animal studies of acute exposure to trivalent chromium appears to involve the respiratory system. Specifically, acute exposure to trivalent chromium is associated with impaired lung function and lung damage. Based on what is known about absorption of chromium in the human body, its potential mechanism of action in cells, and occupational data indicating that valence states other than hexavalent exhibit a relative lack of toxicity the toxicity of elemental and divalent chromium compounds is expected to be similar to or less than common trivalent forms. No significant acute toxicological data identified in literature search. The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing. Tenth Annual Report on Carcinogens: Substance known to be Carcinogenic [*National Toxicology Program: U.S. Dep. of Health and Human Services 2002*] Gastrointestinal tumours, lymphoma, musculoskeletal tumours and tumours at site of application recorded.
BISPHENOL A/ PHOSGENE POLYMER	No significant acute toxicological data identified in literature search. The chemical structure of hydroxylated diphenylalkanes or bisphenols consists of two phenolic rings joined together through a bridging carbon. This class of endocrine disruptors that mimic oestrogens is widely used in industry, particularly in plastics Bisphenol A (BPA) and some related compounds exhibit oestrogenic activity in human breast cancer cell line MCF-7, but there were remarkable differences in activity. Several derivatives of BPA exhibited significant thyroid hormonal activity towards rat pituitary cell line GH3, which releases growth hormone in a thyroid hormone-dependent manner. However, BPA and several other derivatives did not show such activity. Results suggest that the 4-hydroxyl group of the A-phenyl ring and the B-phenyl ring of BPA derivatives are required for these hormonal activities, and substituents at the 3,5-positions of the phenyl rings and the bridging alkyl moiety markedly influence the activities. Bisphenols promoted cell proliferation and increased the synthesis and secretion of cell type-specific proteins. When ranked by proliferative potency, the longer the alkyl substituent at the bridging carbon, the lower the concentration needed for maximal cell yield; the most active compound contained two propyl chains at the bridging carbon. Bisphenols with two hydroxyl groups in the para position and an angular configuration are suitable for appropriate hydrogen bonding to the acceptor site of the oestrogen receptor.
LITHIUM FLUOROPHOSPHATE & GRAPHITE	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. No significant acute toxicological data identified in literature search.
LEAD	WARNING: Lead is a cumulative poison and has the potential to cause
LEAD	abortion and intellectual impairment to unborn children of
LEAD	pregnant workers.
MERCURY (ELEMENTAL)	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

Continued...

	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.
MERCURY (ELEMENTAL)	Animal studies have shown that mercury may be a reproductive effector.

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

Legend: ✗ *– Data available but does not fill the criteria for classification*
✓ *– Data required to make classification available*
⊘ *– Data Not Available to make classification*

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
lithium cobaltate	LC50	96	Fish	1.406mg/L	2
lithium cobaltate	EC50	48	Crustacea	2.618mg/L	2
lithium cobaltate	EC50	504	Crustacea	0.012mg/L	2
lithium cobaltate	EC50	72	Algae or other aquatic plants	0.144mg/L	2
lithium cobaltate	NOEC	168	Algae or other aquatic plants	0.0018mg/L	2
lithium fluorophosphate	LC50	96	Fish	42mg/L	2
lithium fluorophosphate	EC50	528	Fish	1mg/L	2
lithium fluorophosphate	NOEC	528	Fish	0.2mg/L	2
lithium fluorophosphate	EC50	48	Crustacea	98mg/L	2
lithium fluorophosphate	EC50	96	Algae or other aquatic plants	43mg/L	2
ethylene carbonate	EC50	96	Algae or other aquatic plants	17.388mg/L	3
ethylene carbonate	LC50	96	Fish	238.065mg/L	3
graphite	LC50	96	Fish	>100mg/L	2
graphite	EC50	48	Crustacea	>=38.4- <=67.6mg/L	2
graphite	NOEC	672	Crustacea	>=0.58- <=10mg/L	2
graphite	EC50	72	Algae or other aquatic plants	19mg/L	2
graphite	EC50	72	Algae or other aquatic plants	7.2mg/L	2
lead	BCFD	8	Fish	4.324mg/L	4
lead	NOEC	672	Fish	0.00003mg/L	4
lead	LC50	96	Fish	0.0079mg/L	2
lead	EC50	48	Crustacea	0.029mg/L	2
lead	EC50	48	Algae or other aquatic plants	0.0217mg/L	2
lead	EC50	72	Algae or other aquatic plants	0.0205mg/L	2
mercury (elemental)	BCF	720	Fish	0.001mg/L	4
mercury (elemental)	EC50	72	Algae or other aquatic plants	0.0025mg/L	4
mercury (elemental)	LC50	96	Fish	0.004mg/L	4
mercury (elemental)	EC50	240	Fish	0.0003mg/L	5
mercury (elemental)	EC50	48	Crustacea	0.0003mg/L	2
mercury (elemental)	NOEC	2688	Crustacea	0.00025mg/L	2
lead	BCFD	8	Fish	4.324mg/L	4
lead	NOEC	672	Fish	0.00003mg/L	4
lead	LC50	96	Fish	0.0079mg/L	2
lead	EC50	48	Crustacea	0.029mg/L	2
lead	EC50	48	Algae or other aquatic plants	0.0217mg/L	2
lead	EC50	72	Algae or other aquatic plants	0.0205mg/L	2
mercury (elemental)	BCF	720	Fish	0.001mg/L	4
mercury (elemental)	EC50	72	Algae or other aquatic plants	0.0025mg/L	4
mercury (elemental)	LC50	96	Fish	0.004mg/L	4
mercury (elemental)	EC50	240	Fish	0.0003mg/L	5
mercury (elemental)	EC50	48	Crustacea	0.0003mg/L	2
mercury (elemental)	NOEC	2688	Crustacea	0.00025mg/L	2
chromium	BCF	1440	Algae or other aquatic plants	0.0495mg/L	4

Continued...

chromium	EC50	72	Algae or other aquatic plants	0.104mg/L	4
chromium	LC50	96	Fish	13.9mg/L	4
chromium	NOEC	672	Fish	0.00019mg/L	4
chromium	EC50	48	Crustacea	0.0225mg/L	5
chromium	EC50	48	Crustacea	0.0245mg/L	5
cadmium	BCF	960	Fish	500mg/L	4
cadmium	LC50	96	Fish	0.001mg/L	4
cadmium	NOEC	168	Fish	0.00001821mg/L	4
cadmium	EC50	336	Crustacea	0.00065mg/L	5
cadmium	EC50	48	Crustacea	0.0033mg/L	5
cadmium	EC50	72	Algae or other aquatic plants	0.018mg/L	2
Legend:	*Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 - 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

Bioaccumulative potential

Ingredient	Bioaccumulation
ethylene carbonate	LOW (LogKOW = -0.3388)

Mobility in soil

Ingredient	Mobility
ethylene carbonate	LOW (KOC = 9.168)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	Consult State Land Waste Management Authority for disposal. Bury residue in an authorised landfill.

SECTION 14 TRANSPORT INFORMATION

Labels Required

	
Marine Pollutant	NO
HAZCHEM	4W

Land transport (ADG)

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

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee)

UN number	3481
Packing group	II

Continued...

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

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 COBALTATE(12190-79-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

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

Australia Inventory of Chemical Substances (AICS)

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

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)
Australia Hazardous Substances Information System - Consolidated Lists

LEAD(7439-92-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

MERCURY (ELEMENTAL)(7439-97-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

LEAD(7439-92-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

MERCURY (ELEMENTAL)(7439-97-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

CHROMIUM(7440-47-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

CADMIUM(7440-43-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

BISPHENOL A/ PHOSGENE POLYMER(25971-63-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

National Inventory	Status
Australia - AICS	Y
Canada - DSL	N (lithium fluorophosphate)
Canada - NDSL	N (lead; graphite; bisphenol A/ phosgene polymer; ethylene carbonate; mercury (elemental); lithium cobaltate; chromium; cadmium)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	N (bisphenol A/ phosgene polymer)
Japan - ENCS	N (graphite; mercury (elemental); chromium; lithium fluorophosphate; cadmium)
Korea - KECI	Y

Continued...

New Zealand - NZIoC	N (lithium fluorophosphate)
Philippines - PICCS	N (lithium cobaltate)
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

Other information

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

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

Definitions and abbreviations

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

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

Other information:

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Department issuing SDS: Research and Development

Contact: Technical Director

end of SDS