Battery Pack P/N 452-0133 & 452-0222

Hawker Pacific

Chemwatch Hazard Alert Code: **3**

Chemwatch: **48-0287**
Version No: **5.1.13.9**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **11/01/2019**
Print Date: **08/16/2021**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	Battery Pack P/N 452-0133 & 452-0222
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	LITHIUM METAL BATTERIES (including lithium alloy batteries)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Lithium based battery product NOTE: Hazard statement relates to battery contents. Potential for exposure should not exist unless the battery leaks, is exposed to high temperatures or is mechanically, physically or electrically abused.

Details of the supplier of the safety data sheet

Registered company name	**Hawker Pacific**	**Procter & Gamble Australia Pty Ltd**
Address	112 Airport Avenue Bankstown NSW 2200 Australia	Level 4, 1 Innovation Road Macquarie Park NSW 2113 Australia
Telephone	+61 2 9708 8555	1800 641 820 (Gilette) |1800 028 280 (Other)
Fax	+61 2 9791 0780	+61 2 8864 5319
Website	Not Available	Not Available
Email	Not Available	Not Available

Emergency telephone number

Association / Organisation	**Hawker Pacific**	**Procter & Gamble Australia Pty Ltd**
Emergency telephone numbers	13 11 26 (Poisons Info. Centre)	+61 1800 951 288 (free)
Other emergency telephone numbers	Not Available	+61 2 9186 1132 (alternative)

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Acute Toxicity (Oral) Category 4, Acute Toxicity (Inhalation) Category 4, Skin Corrosion/Irritation Category 1B, Serious Eye Damage/Eye Irritation Category 1, Reproductive Toxicity Category 1B, Oxidizing Liquids Category 3
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

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Hazard pictogram(s)	
Signal word	**Danger**

Hazard statement(s)

H302	Harmful if swallowed.
H332	Harmful if inhaled.
H314	Causes severe skin burns and eye damage.
H360FD	May damage fertility. May damage the unborn child.
AUH014	Reacts violently with water.
AUH018	In use, may form flammable/explosive vapour/air mixture.
AUH019	May form explosive peroxides.
H272	May intensify fire; oxidiser.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P260	Do not breathe dust/fume.
P264	Wash all exposed external body areas thoroughly after handling.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P220	Keep away from clothing and other combustible materials.
P270	Do not eat, drink or smoke when using this product.

Precautionary statement(s) Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P370+P378	In case of fire: Use dry agent to extinguish.
P363	Wash contaminated clothing before reuse.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available		sealed metal containers with electrochemical contents, typically
1313-13-9	25-50	manganese dioxide

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CAS No	%[weight]	Name
108-32-7	<10	propylene carbonate
110-71-4	<10	1,2-dimethoxyethane
109-99-9	<10	tetrahydrofuran
7439-93-2	<10	lithium
1333-86-4	<10	carbon black
7791-03-9	<10	lithium perchlorate

Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available*

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If cell contents come in contact with the eye hold eyes open and wash continuously for at least 15 minutes. Seek medical attention. ▸ Generally not applicable.
Skin Contact	If skin contact with the electrolyte occurs wash thoroughly with soap and water. Do not pull solidified product off the skin. Seek medical attention. If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation. ▸ Generally not applicable.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor. ▸ Generally not applicable.
Ingestion	If battery swallowed do not induce vomiting and seek medical attention IMMEDIATELY. ▸ **IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY.** ▸ For advice, contact a Poisons Information Centre or a doctor. ▸ Urgent hospital treatment is likely to be needed. ▸ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. ▸ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist. ▸ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS. **Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:** ▸ **INDUCE** vomiting with fingers down the back of the throat, **ONLY IF CONSCIOUS**. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. **NOTE:** Wear a protective glove when inducing vomiting by mechanical means. ▸ Generally not applicable. 53a3

Indication of any immediate medical attention and special treatment needed

Published reports recommend removal from the esophagus be done endoscopically (under direct visualization). Batteries beyond the esophagus need not be retrieved unless there are signs of injury to the GI tract or a large diameter battery fails to pass the pylorus. If asymptomatic, follow-up x- rays are necessary only to confirm the passage of larger batteries. Confirmation by stool inspection is preferable under most circumstances. Various corrosive, harmful or toxic substances is possible in certain cases. These substances may include lithium and/or fluoride salts; specific antidotes may be required in cases of ingestion for lithium salts and in cases of oral/dermal/inhalation contact with fluorides. If fluoride contact is suspected, calcium salts may be of value in treatment. Do not give ipecac.
Treat symptomatically.
Clinical effects of lithium intoxication appear to relate to duration of exposure as well as to level.

- Lithium produces a generalised slowing of the electroencephalogram; the anion gap may increase in severe cases.
- Emesis (or lavage if the patient is obtunded or convulsing) is indicated for ingestions exceeding 40 mg (Li)/Kg.
- Overdose may delay absorption; decontamination measures may be more effective several hours after cathartics.
- Charcoal is not useful. No clinical data are available to guide the administration of catharsis.
- Haemodialysis significantly increases lithium clearance; indications for haemodialysis include patients with serum levels above 4 meq/L.

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- There are no antidotes.

[Ellenhorn and Barceloux: Medical Toxicology]
For acute or short term repeated exposures to ethylene glycol:

- Early treatment of ingestion is important. Ensure emesis is satisfactory.
- Test and correct for metabolic acidosis and hypocalcaemia.
- Apply sustained diuresis when possible with hypertonic mannitol.
- Evaluate renal status and begin haemodialysis if indicated. [I.L.O]
- Rapid absorption is an indication that emesis or lavage is effective only in the first few hours. Cathartics and charcoal are generally not effective.
- Correct acidosis, fluid/electrolyte balance and respiratory depression in the usual manner. Systemic acidosis (below 7.2) can be treated with intravenous sodium bicarbonate solution.
- Ethanol therapy prolongs the half-life of ethylene glycol and reduces the formation of toxic metabolites.
- Pyridoxine and thiamine are cofactors for ethylene glycol metabolism and should be given (50 to 100 mg respectively) intramuscularly, four times per day for 2 days.
- Magnesium is also a cofactor and should be replenished. The status of 4-methylpyrazole, in the treatment regime, is still uncertain. For clearance of the material and its metabolites, haemodialysis is much superior to peritoneal dialysis.

[Ellenhorn and Barceloux: Medical Toxicology]
It has been suggested that there is a need for establishing a new biological exposure limit before a workshift that is clearly below 100 mmol ethoxy-acetic acids per mole creatinine in morning urine of people occupationally exposed to ethylene glycol ethers. This arises from the finding that an increase in urinary stones may be associated with such exposures.
Laitinen J., et al: Occupational & Environmental Medicine 1996; 53, 595-600

Both dermal and oral toxicity of manganese salts is low because of limited solubility of manganese. No known permanent pulmonary sequelae develop after acute manganese exposure. Treatment is supportive.

[*Ellenhorn and Barceloux: Medical Toxicology*]

In clinical trials with miners exposed to manganese-containing dusts, L-dopa relieved extrapyramidal symptoms of both hypo kinetic and dystonic patients. For short periods of time symptoms could also be controlled with scopolamine and amphetamine. BAL and calcium EDTA prove ineffective.

[*Gosselin et al: Clinical Toxicology of Commercial Products.*]

Antithyroid effects produced by the perchlorates may be reversed with iodine. Patients should be warned to report the development of sore throat, fever or rashes since these are indicative of blood abnormalities.
For chlorates:

For severe intoxication: Empty the stomach by lavage and aspiration or by emesis, give demulcents or sweetened drinks and maintain respiration. Pethidine may be given if required. A 1% solution of sodium thiosulfate may be used for lavage and may also be given by intravenous infusion. Haemodialysis, peritoneal dialysis or exchange perfusions may be of value in removing chlorate from the blood. Forced diuresis should not be attempted if there is inadequate urine input.

MARTINDALE: The Extra Pharmacopoeia, 27th Edition

The high sensitivity of glucose-6-phosphate dehydrogenase to denaturation by chlorate explains the inefficacy of methylene blue to reduce methaemoglobin formed, as the antidotal effect of methylene blue depends on NADPH formed mainly by the oxidation of glucose-6-phosphate. The observed changes occur only in the presence of methaemoglobin which forms a destabilising complex with chlorate. Methaemoglobin thus autocatalytically increases methaemoglobin formation and destruction of the erythrocyte.

SECTION 5 Firefighting measures

Extinguishing media

Metal dust fires need to be smothered with sand, inert dry powders.
DO NOT USE WATER, CO2 or FOAM.

- Use DRY sand, graphite powder, dry sodium chloride based extinguishers, G-1 or Met L-X to smother fire.
- Confining or smothering material is preferable to applying water as chemical reaction may produce flammable and explosive hydrogen gas.
- Chemical reaction with CO2 may produce flammable and explosive methane.
- If impossible to extinguish, withdraw, protect surroundings and allow fire to burn itself out.
- **DO NOT** use halogenated fire extinguishing agents.

Special hazards arising from the substrate or mixture

Fire Incompatibility	▸ Reacts with acids producing flammable / explosive hydrogen (H2) gas ▸ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result ▸ Keep dry ▸ **NOTE:** May develop pressure in containers; open carefully. Vent periodically.

Advice for firefighters

Fire Fighting	▸ When silica dust is dispersed in air, firefighters should wear inhalation protection as hazardous substances from the fire may

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	be adsorbed on the silica particles. ▸ When heated to extreme temperatures, (>1700 deg.C) amorphous silica can fuse. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire. ▸ Equipment should be thoroughly decontaminated after use. Slight hazard when exposed to heat, flame and oxidisers.
Fire/Explosion Hazard	**WARNING:** In use may form flammable/ explosive vapour-air mixtures. ▸ **DO NOT** disturb burning dust. Explosion may result if dust is stirred into a cloud, by providing oxygen to a large surface of hot metal. ▸ **DO NOT** use water or foam as generation of explosive hydrogen may result. With the exception of the metals that burn in contact with air or water (for example, sodium), masses of combustible metals do not represent unusual fire risks because they have the ability to conduct heat away from hot spots so efficiently that the heat of combustion cannot be maintained - this means that it will require a lot of heat to ignite a mass of combustible metal. Generally, metal fire risks exist when sawdust, machine shavings and other metal 'fines' are present. Metal powders, while generally regarded as non-combustible: ▸ May burn when metal is finely divided and energy input is high. ▸ May react explosively with water. ▸ May be ignited by friction, heat, sparks or flame. ▸ May **REIGNITE** after fire is extinguished. ▸ Will burn with intense heat. Note: ▸ Metal dust fires are slow moving but intense and difficult to extinguish. ▸ Containers may explode on heating. ▸ Dusts or fumes may form explosive mixtures with air. ▸ Gases generated in fire may be poisonous, corrosive or irritating. ▸ Hot or burning metals may react violently upon contact with other materials, such as oxidising agents and extinguishing agents used on fires involving ordinary combustibles or flammable liquids. ▸ Temperatures produced by burning metals can be higher than temperatures generated by burning flammable liquids ▸ Some metals can continue to burn in carbon dioxide, nitrogen, water, or steam atmospheres in which ordinary combustibles or flammable liquids would be incapable of burning. Combustible. Will burn if ignited. Combustion products include: carbon monoxide (CO) carbon dioxide (CO2) hydrogen chloride phosgene other pyrolysis products typical of burning organic material. Articles and manufactured articles may constitute a fire hazard where polymers form their outer layers or where combustible packaging remains in place. Certain substances, found throughout their construction, may degrade or become volatile when heated to high temperatures. This may create a secondary hazard. **WARNING**: Long standing in contact with air and light may result in the formation of potentially explosive peroxides.
HAZCHEM	4Y

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	▸ Clean up all spills immediately. ▸ Secure load if safe to do so. ▸ Bundle/collect recoverable product. ▸ Collect remaining material in containers with covers for disposal.
Major Spills	· Do not use compressed air to remove metal dusts from floors, beams or equipment · Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation. · Use non-sparking handling equipment, tools and natural bristle brushes. · Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations

Continued...

	· Cover and reseal partially empty containers. · Do not allow chips, fines or dusts to contact water, particularly in enclosed areas. If molten: ▸ Contain the flow using dry sand or salt flux as a dam. ▸ All tooling (e.g., shovels or hand tools) and containers which come in contact with molten metal must be preheated or specially coated, rust free and approved for such use. ▸ Allow the spill to cool before remelting scrap. ▸ 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	DO NOT ATTEMPT TO OPEN SEALED CELLS OR BATTERIES – BATTERY CONTENTS MAY PRESENT SERIOUS SAFETY AND HEALTH HAZARDS. SHORT-CIRCUITING THE TERMINALS OF A DEVICE MAY RESULT IN DAMAGE TO DEVICE AND ANY NEARBY OBJECTS OR PERSONNEL. Accidental short circuit for a few seconds will not seriously harm this battery. Avoid prolonged short circuit as battery will lose energy and may even rupture at high temperatures. Keep away from open flames or temperatures exceeding manufacturer ratings For molten metals: · Molten metal and water can be an explosive combination. The risk is greatest when there is sufficient molten metal to entrap or seal off water. Water and other forms of contamination on or contained in scrap or remelt ingot are known to have caused explosions in melting operations. While the products may have minimal surface roughness and internal voids, there remains the possibility of moisture contamination or entrapment. If confined, even a few drops can lead to violent explosions. · All tooling, containers, molds and ladles, which come in contact with molten metal must be preheated or specially coated, rust free and approved for such use. · Any surfaces that may contact molten metal (e.g. concrete) should be specially coated · Drops of molten metal in water (e.g. from plasma arc cutting), while not normally an explosion hazard, can generate enough flammable hydrogen gas to present an explosion hazard. Vigorous circulation of the water and removal of the particles minimise the hazard. During melting operations, the following minimum guidelines should be observed: · Inspect all materials prior to furnace charging and completely remove surface contamination such as water, ice, snow, deposits of grease and oil or other surface contamination resulting from weather exposure, shipment, or storage. · Store materials in dry, heated areas with any cracks or cavities pointed downwards. · Preheat and dry large objects adequately before charging in to a furnace containing molten metal. This is typically done by the use of a drying oven or homogenising furnace. The dry cycle should bring the metal temperature of the coldest item of the batch to 200 degree C (400 deg F) and then hold at that temperature for 6 hours. ▸ May form explosive peroxides on standing or following concentration by distillation. ▸ Review of stocks and testing for peroxide content by given tested procedures at 3-monthly intervals is recommended, together with safe disposal of peroxidic samples. [Peroxide-containing residues can often be rendered innocuous by pouring into an excess of sodium carbonate solution] The substance accumulates peroxides which may become hazardous only if it evaporates or is distilled or otherwise treated to concentrate the peroxides. The substance may concentrate around the container opening for example. Purchases of peroxidisable chemicals should be restricted to ensure that the chemical is used completely before it can become peroxidised. ▸ A responsible person should maintain an inventory of peroxidisable chemicals or annotate the general chemical inventory to indicate which chemicals are subject to peroxidation. An expiration date should be determined. The chemical should either be treated to remove peroxides or disposed of before this date. ▸ The person or laboratory receiving the chemical should record a receipt date on the bottle. The individual opening the container should add an opening date. ▸ Unopened containers received from the supplier should be safe to store for 18 months. ▸ Opened containers should not be stored for more than 12 months. ▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ **DO NOT** allow material to contact humans, exposed food or food utensils. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke.

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	▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	Do not store batteries loose where terminals may contact each other. Keep battery assemblies intact. ▸ Store in original containers. ▸ Keep containers securely sealed. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Store away from incompatible materials.

Conditions for safe storage, including any incompatibilities

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

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	manganese dioxide	Manganese, dust & compounds (as Mn)	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	tetrahydrofuran	Tetrahydrofuran	100 ppm / 295 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	carbon black	Carbon black	3 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
manganese dioxide	4.7 mg/m3	7.9 mg/m3	690 mg/m3
manganese dioxide	4.2 mg/m3	6.9 mg/m3	41 mg/m3
propylene carbonate	34 mg/m3	370 mg/m3	2,200 mg/m3
1,2-dimethoxyethane	13 ppm	140 ppm	840 ppm
tetrahydrofuran	Not Available	Not Available	Not Available
lithium	3.3 mg/m3	36 mg/m3	220 mg/m3
carbon black	9 mg/m3	99 mg/m3	590 mg/m3
lithium perchlorate	1.2 mg/m3	13 mg/m3	79 mg/m3

Ingredient	Original IDLH	Revised IDLH
manganese dioxide	500 mg/m3	Not Available
propylene carbonate	Not Available	Not Available
1,2-dimethoxyethane	Not Available	Not Available
tetrahydrofuran	2,000 ppm	Not Available
lithium	Not Available	Not Available
carbon black	1,750 mg/m3	Not Available
lithium perchlorate	Not Available	Not Available

Occupational Exposure Banding

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Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
propylene carbonate	E	≤ 0.1 ppm
1,2-dimethoxyethane	E	≤ 0.1 ppm
lithium	C	> 0.1 to ≤ milligrams per cubic meter of air (mg/m³)
lithium perchlorate	E	≤ 0.01 mg/m³
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

None assigned. Refer to individual constituents.

Exposure controls

Appropriate engineering controls

Articles or manufactured items, in their original condition, generally don't require engineering controls during handling or in normal use.
Exceptions may arise following extensive use and subsequent wear, during recycling or disposal operations where substances, found in the article, may be released to the environment.

Metal dusts must be collected at the source of generation as they are potentially explosive.

- Avoid ignition sources.
- Good housekeeping practices must be maintained.
- Dust accumulation on the floor, ledges and beams can present a risk of ignition, flame propagation and secondary explosions.
- Do not use compressed air to remove settled materials from floors, beams or equipment
- Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation.
- Use non-sparking handling equipment, tools and natural bristle brushes. Cover and reseal partially empty containers. Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations.
- Do not allow chips, fines or dusts to contact water, particularly in enclosed areas.
- Metal spraying and blasting should, where possible, be conducted in separate rooms. This minimises the risk of supplying oxygen, in the form of metal oxides, to potentially reactive finely divided metals such as aluminium, zinc, magnesium or titanium.
- Work-shops designed for metal spraying should possess smooth walls and a minimum of obstructions, such as ledges, on which dust accumulation is possible.
- Wet scrubbers are preferable to dry dust collectors.
- Bag or filter-type collectors should be sited outside the workrooms and be fitted with explosion relief doors.
- Cyclones should be protected against entry of moisture as reactive metal dusts are capable of spontaneous combustion in humid or partially wetted states.
- Local exhaust systems must be designed to provide a minimum capture velocity at the fume source, away from the worker, of 0.5 metre/sec.
- Local ventilation and vacuum systems must be designed to handle explosive dusts. Dry vacuum and electrostatic precipitators must not be used, unless specifically approved for use with flammable/ explosive dusts.

Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
welding, brazing fumes (released at relatively low velocity into moderately still air)	0.5-1.0 m/s (100-200 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

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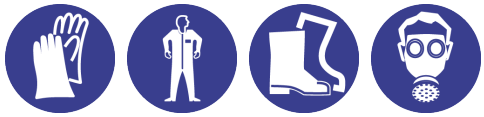
Personal protection	
Eye and face protection	No special equipment required due to the physical form of the product. ‣ Safety glasses with side shields. ‣ Chemical goggles. ‣ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	Wear general protective gloves, eg. light weight rubber gloves. ‣ Neoprene rubber gloves
Body protection	See Other protection below
Other protection	‣ Protective overalls, closely fitted at neck and wrist. ‣ Eye-wash unit. **IN CONFINED SPACES:** ‣ Non-sparking protective boots ‣ Static-free clothing. ‣ Ensure availability of lifeline. Staff should be trained in all aspects of rescue work. Rescue gear: Two sets of SCBA breathing apparatus Rescue Harness, lines etc. · During repair or maintenance activities the potential exists for exposures to toxic metal particulate in excess of the occupational standards. Under these circumstances, protecting workers can require the use of specific work practices or procedures involving the combined use of ventilation, wet and vacuum cleaning methods, respiratory protection, decontamination, special protective clothing, and when necessary, restricted work zones. · Protective over-garments or work clothing must be worn by persons who may become contaminated with particulate during activities such as machining, furnace rebuilding, air cleaning equipment filter changes, maintenance, furnace tending, etc. Contaminated work clothing and over-garments must be managed in a controlled manner to prevent secondary exposure to workers of third parties, to prevent the spread of particulate to other areas, and to prevent particulate from being taken home by workers. · Personnel who handle and work with molten metal should utilise primary protective clothing like polycarbonate face shields, fire resistant tapper's jackets, neck shades (snoods), leggings, spats and similar equipment to prevent burn injuries. In addition to primary protection, secondary or day-to-day work clothing that is fire resistant and sheds metal splash is recommended for use with molten metal. Synthetic materials should never be worn even as secondary clothing (undergarments).

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
Battery Pack P/N 452-0133 & 452-0222

Material	CPI
BUTYL	C
CPE	C
NEOPRENE	C
PE/EVAL/PE	C
PVA	C
TEFLON	C
VITON/CHLOROBUTYL	C

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice

Respiratory protection

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

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

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

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

If inhalation risk above the TLV exists, wear approved dust respirator.
Use respirators with protection factors appropriate for the exposure level.
‣ Up to 5 X TLV, use valveless mask type; up to 10 X TLV, use 1/2 mask dust respirator

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of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

- Up to 50 X TLV, use full face dust respirator or demand type C air supplied respirator
- Up to 500 X TLV, use powered air-purifying dust respirator or a Type C pressure demand supplied-air respirator
- Over 500 X TLV wear full-face self-contained breathing apparatus with positive pressure mode or a combination respirator with a Type C positive pressure supplied-air full-face respirator and an auxiliary self-contained breathing apparatus operated in pressure demand or other positive pressure mode

Respiratory protection not normally required due to the physical form of the product.
None under normal operating conditions.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Impermeable container containing odourless liquid. Electrolyte contents only exposed on crushing, puncture or destructive disassembly. Leaking devices may emit acrid or ethereal odour.

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 Available
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	Immiscible	**pH as a solution (%)**	Not Applicable
Vapour density (Air = 1)	Not Applicable	**VOC g/L**	Not Applicable

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	
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	Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the

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	recruitment and activation of many cell types, mainly derived from the vascular system. Manganese fume is toxic and produces nervous system effects characterised by tiredness. Acute poisoning is rare although acute inflammation of the lungs may occur. A chemical pneumonia may also result from frequent exposure. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure. Overexposure to tetrahydrofuran, by inhalation, may result in irritation of the mucous membranes and may produce coughing, chest pains, nausea, dizziness, headache and narcosis. Exposure to high concentrations can affect the central nervous system due to the strong narcotic effect of the material. Concentrations greater than 25000 ppm were reported to produce anaesthesia in animals. Anaesthetic properties are poor as onset is delayed and recovery is slow. Pronounced hypotension and marked respiratory hypernea accompany narcosis. Other symptoms include muscular hypotonia and disappearance of corneal reflexes, followed by coma and death. Inhalation of dusts, generated by the material, during the course of normal handling, may be harmful. Acute silicosis occurs under conditions of extremely high silica dust exposure particularly when the particle size of the dust is small. It differs greatly from classical silicosis both clinically and pathologically. The disease is rapidly progressive with diffuse pulmonary involvement developing only months after the initial exposure and causing deaths within 1 to 2 years. It is often complicated by an associated tuberculosis. The lungs of victims contain no classical silicotic nodules or only a few, microscopic abortive nodules, whereas the air spaces are diffusively filled and distended with silica-containing, lipoprotein paste in which degenerating and necrotic macrophages are sometimes discernible - the condition is sometimes described as alveolar lipoproteinosis. The uptake of silica particles by macrophages and lysosymal incorporation, is followed by rupture of the lysosomal membrane and release of lysosomal enzymes into cytoplasm of the macrophage. This causes the macrophage to be digested by its own enzymes and after lysis the free silica is released to be ingested by other macrophages thus continuing initiate collagen formation in the lung tissue producing the characteristic nodule found in classical (chronic) silicosis.
Ingestion	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. 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. 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. 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. Not normally a hazard due to physical form of product. Poisonings rarely occur after oral administration of manganese salts as they are generally poorly absorbed from the gut (generally less than 4%) and seems to be dependent, in part, on levels of dietary iron and may increase following the consumption of alcohol. A side-effect of oral manganese administration is an increase in losses of calcium in the faeces and a subsequent lowering of calcium blood levels. Absorbed manganese tends to be slowly excreted in the bile. Divalent manganese appears to be 2.5-3 times more toxic than the trivalent form. Considered an unlikely route of entry in commercial/industrial environments Ingestion of tetrahydrofuran may not, in itself, produce internal injury, however, contaminating levels of furan, present in certain grades of commercial product, may produce liver and kidney injury. The intake of alcoholic beverages may enhance the toxic effects of tetrahydrofuran.
Skin Contact	The cell contents cause severe skin irritation and corrosion on contact. Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. Skin contact with tetrahydrofuran may produce smarting and reddening of the skin and after prolonged exposures, contact (non-allergic) dermatitis may result due to the degreasing effect of the substance.

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Eye	Eye contact with the contents of an opened battery may cause severe irritation. When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	The normal handling of sealed cells or batteries is not hazardous. Exposure to cell contents may cause irritation to skin, severe eye irritation. Harmful: danger of serious damage to health by prolonged exposure through inhalation. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: - clear evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of: - clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Neuromuscular effects result from chronic over-exposure to lithium compounds. These may include tremor, ataxia, clonus and hyperactive reflexes. Some animal studies have shown that exposure during pregnancy may produce birth defects. Other studies with rats, rabbits and monkeys have not shown teratogenic effects. Human data are ambiguous; it is well established that lithium can cross the human placenta. Of 225 registered pregnancies in which the mothers had received lithium (as a tranquiliser) there were 25 instances of congenital malformation. Although pharmacological doses of lithium cannot be unequivocally designated as a human teratogen, lithium therapy is contraindicated in women of childbearing potential. Prolonged exposure may produce anorexia, weight loss and emaciation. The kidneys, behavioural/ central nervous system and peripheral nervous system may also show adverse effects. Various types of dermatitis (psoriasis, alopecia, cutaneous ulcers, acne, follicular papules, xerosis cutis, exfoliative) may also result from chronic skin exposure. Lithium ion can be an effective treatment for manic depression. It is thought to bind the enzyme IMPase (inositol monophosphatase) and thereby mediates its influence in producing a response to calcium-induced production of neurotransmitters and hormones thought to be responsible for the clinical picture. Lithium ions interfere with ion transport processes (involving the “sodium pump”) that relay and amplify messages carried to the cells of the brain. Mania is associated with irregular increases in protein kinase C (PKC) activity within the brain. Lithium carbonate and sodium valproate, another drug traditionally used to treat the disorder, act in the brain by inhibiting PKC's activity and help to produce other compounds that also inhibit the PKC. Taking lithium salts has risks and side effects. Extended use of lithium to treat various mental disorders has been known to lead to acquired nephrogenic diabetes insipidus. Nephrogenic diabetes insipidus (NDI), also known as renal diabetes insipidus, is a form of diabetes insipidus primarily due to pathology of the kidney. This is in contrast to central or neurogenic diabetes insipidus, which is caused by insufficient levels of antidiuretic hormone (ADH, also called vasopressin). Nephrogenic diabetes insipidus is caused by an improper response of the kidney to ADH, leading to a decrease in the ability of the kidney to concentrate the urine by removing free water. Lithium intoxication can affect the central nervous system and renal system and can be lethal In subchronic studies, rats were exposed to 3 milliequivalents Li/kg/day (equivalent to 1450 mg for a 70 kg person) but did not accumulate Li whilst on a high sodium diet. However when sodium was restricted, fatal kidney toxicity developed. Dogs survived daily dose of 50 mg LiCl/kg for 150 days to the termination of the experiment on a normal sodium intake, whereas the same dose was lethal in 12 to 18 days on a low sodium diet: 20 mg LiCl/kg/day resulted in death in 18 to 30 days. Several reports have demonstrated that lithium may impair basal ganglia activity. Lithium intoxication has been associated, severe and persistent oculogyric crises. Oculogyric crisis (OGC) is the name of a dystonic reaction to certain drugs or medical conditions characterized by a prolonged involuntary upward deviation of the eyes. The term "oculogyric" refers to the bilateral elevation of the visual gaze but several other responses are associated with the crisis. Chronic symptoms produced by crystalline silicas included decreased vital lung capacity and chest infections. Lengthy exposure may cause silicosis a disabling form of pneumoconiosis which may lead to fibrosis, a scarring of the lining of the air sacs in the lung. The form and severity in which silicosis manifests itself depends in part on the type and extent of exposure to silica dusts: chronic, accelerated and acute forms are all recognized. In later stages the critical condition may become disabling and potentially fatal. Restrictive and/or obstructive lung function changes may result from chronic exposure. A risk associated with silicosis is development of pulmonary tuberculosis (silico-tuberculosis). Respiratory insufficiencies due to massive fibrosis and reduced pulmonary function, possibly with accompanying heart failure, are other potential causes of death due to silicosis. Not all individuals with silicosis will exhibit symptoms (signs) of the disease. However, silicosis can be progressive, and symptoms may potentially appear years after exposures have ceased. Symptoms of silicosis may include (but are not limited to): Shortness of breath; difficulty breathing with or without exertion; coughing; diminished work capacity; diminished chest expansion; reduction of lung volume; heart enlargement and/or failure. Respirable dust containing newly broken particles has been shown to be more hazardous to animals in laboratory tests than respirable dust containing older silica particles of similar size. Respirable silica particles which had aged for sixty days or more showed less lung injury in animals than equal exposures of respirable dust containing newly broken pieces of silica. There are reports in the literature indicating that crystalline silica exposure may be associated with adverse health effects involving the kidney, scleroderma (thickening of the skin caused by swelling and thickening of fibrous tissue) and other autoimmune and immunity-related disorders. Several studies of persons with silicosis or silica exposure also indicate or suggest increased risk of

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developing lung cancer, a risk that may increase with the duration of exposure. Many of these studies of silicosis do not account for lung cancer confounders, especially smoking.
Symptoms may appear 8 to 18 months after initial exposure. Smoking increases this risk. Classic silicosis is a chronic disease characterised by the formation of scattered, rounded or stellate silica-containing nodules of scar tissue in the lungs ranging from microscopic to 1.0 cm or more. The nodules isolate the inhaled silica particles and protect the surrounding normal and functioning tissue from continuing injury. Simple silicosis (in which the nodules are less than 1.0 cm in diameter) is generally asymptomatic but may be slowly progressive even in the absence of continuing exposure. Simple silicosis can develop in complicated silicoses (in which nodules are greater than 1.0 cm in diameter) and can produce disabilities including an associated tuberculous infection (which 50 years ago accounted for 75% of the deaths among silicotic workers). Crystalline silica deposited in the lungs causes epithelial and macrophage injury and activation. Crystalline silica translocates to the interstitium and the regional lymph nodes and cause the recruitment of inflammatory cells in a dose dependent manner. In humans, a large fraction of crystalline silica persists in the lungs. The question of potential carcinogenicity associated with chronic inhalation of crystalline silica remains equivocal with some studies supporting the proposition and others finding no significant association. The results of recent epidemiological studies suggest that lung cancer risk is elevated only in those patients with overt silicosis. A relatively large number of epidemiological studies have been undertaken and in some, increased risk gradients have been observed in relation to dose surrogates - cumulative exposure, duration of exposure, the presence of radiographically defined silicosis, and peak intensity exposure. Chronic inhalation in rats by single or repeated intratracheal instillation produced a significant increase in the incidences of adenocarcinomas and squamous cell carcinomas of the lung. Lifetime inhalation of crystalline silica (87% alpha-quartz) at 1 mg/m3 (74% respirable) by rats, produced an increase in animals with keratinising cystic squamous cell tumours, adenomas, adenocarcinomas, adenosquamous cell carcinomas, squamous cell carcinoma and nodular bronchiolar alveolar hyperplasia accompanied by extensive subpleural and peribronchiolar fibrosis, increased pulmonary collagen content, focal lipoproteinosis and macrophage infiltration. Thoracic and abdominal malignant lymphomas developed in rats after single intrapleural and intraperitoneal injection of suspensions of several types of quartz.
Some studies show excess numbers of cases of schleroderma, connective tissue disorders, lupus, rheumatoid arthritis chronic kidney diseases, and end-stage kidney disease in workers
NOTE: Some jurisdictions require health surveillance be conducted on workers occupationally exposed to silica, crystalline. Such surveillance should emphasise

- demography, occupational and medical history and health advice
- standardised respiratory function tests such as FEV1, FVC and FEV1/FVC
- standardised respiratory function tests such as FV1, FVC and FEV1/FVC
- chest X-ray, full size PA view
- records of personal exposure

Chronic and/or sub-lethal exposure to inorganic chlorate may have deleterious effects on human health, such as redness of the eyes and skin (including dermatitis), sore throat, abdominal pain, blue lips or skin, diarrhea, nausea, vomiting, shortness of breath, and unconsciousness. Sodium chlorate may damage the liver, kidneys, and blood cells of humans.
Subchronic chlorate exposure was associated with smaller body and organ weights, blood abnormalities and pituitary and thyroid abnormalities in one study using Sprague-Dawley rats.
Chlorate is a thyroid toxicant producing thyroid gland follicular cell hypertrophy in rats and mice following chronic exposures, and may produce follicular cell tumors in rats. The lack of mutagenicity indicates that the thyroid tumors are induced by a non-mutagenic mechanism and are therefore not likely to be carcinogenic. The effects may be attributed to changes in levels of thyroid hormones seen after administration of high doses of sodium chlorate. In female mice there was equivocal and marginal evidence of increased pancreatic islet carcinoma. Sodium chlorate was negative in most bacterial gene mutation assays and in several cytogenetics tests, including a hypoxanthineguanine phosphoribosyl-transferase (HGPRT) assay in Chinese hamster ovaries and a micronucleus assay.
Intramuscular administration of potassium chlorate to pregnant rats resulted in a prolonged gestation period in most cases, and reduced neonatal weight relative to the controls. According to the author, newborn rats also showed a "marked" increase of haematopoietic residue and lipid deposit over controls, and occasionally, exposure resulted in the appearance of hyaline droplets and casts in newborn kidneys . The number of animals per treatment group/number affected, duration of exposure, and information on dose levels was not available.
African green monkeys (five males and seven females) were used to study the thyroid effects of sodium chlorate when administered for 30-60 days as chlorate at concentrations of 4, 7.5, 15, 30 or 58.4 mg/kg bw per day. Chlorate did not induce thyroid depression. Chlorate did not induce a dose-dependent oxidative stress, as was observed in the case of chlorite.
Female rats were exposed to 1 or 10 mg chlorate/L in their drinking water for ten weeks. Fetuses were taken on the 20th day of gestation and examined for external, visceral and skeletal malformations. No significant adverse findings were reported.
No chromosomal abnormalities were seen in either the micronucleus test or a cytogenetic assay in mouse bone marrow cells following gavage dosing with chlorate

Repeated or prolonged exposure may also damage the liver and may cause a decrease in the heart rate. Systemic poisoning may result from inhalation or chronic ingestion of manganese containing substances. Progressive and permanent disability can occur from chronic manganese poisoning if it is not treated, but it is not fatal.
Chronic exposure has been associated with two major effects; bronchitis/pneumonitis following inhalation of manganese dusts and "manganism", a neuropsychiatric disorder that may also arise from inhalation exposures. Chronic exposure to low levels may result in the accumulation of toxic concentrations in critical organs. The brain in particular appears to sustain cellular damage to the ganglion. Symptoms appear before any pathology is evident and may include a mask-like facial expression, spastic gait, tremors, slurred speech, sometimes dystonia (disordered muscle tone), fatigue, anorexia, asthenia (loss of strength and energy), apathy and the inability to concentrate. Insomnia may be an early finding. Chronic poisoning may occur over a 6-24 month period depending on exposure levels.
The onset of chronic manganese poisoning is insidious, with apathy, anorexia weakness, headache and spasms. Manganese psychosis follows with certain definitive features: unaccountable laughter, euphoria, impulsive acts, absentmindedness, mental confusion, aggressiveness and hallucinations. The final stage is characterised by speech difficulties, muscular twitching, spastic gait and other nervous system effects. Symptoms resemble those of Parkinson's disease. Rat studies indicate the gradual accumulation of brain manganese to produce lesions mimicking those found in Parkinsonism. If the disease is diagnosed whilst

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still in the early stages and the patient is removed from exposure, the course may be reversed.
Inhalation of manganese fumes may cause 'metal fume fever' characterised by flu-like symptoms: fever, chill, nausea, weakness and body aches. Manganese dust is no longer believed to be a causative factor in pneumonia. If there is any relationship at all, it appears to be as an aggravating factor to a preexisting condition.
Prolonged or repeated eye contact may result in conjunctivitis.
Manganese is an essential trace element in all living organisms with the level of tissue manganese remaining remarkably constant throughout life.
Fatal aplastic anaemia has occurred in a small percentage of patients receiving perchlorate (as the sodium salt). Other blood disorders include agranulocytosis, thrombocytopenia and leucopenia. Signs of intolerance may precede changes in blood by several days. The nephrotic syndrome occurs rarely. Nausea, vomiting and hypersensitivity reactions such as maculopapular rashes, fever and lymphadenopathies (lymph node disease) may occur. [Martindale]
The main target organ for perchlorate toxicity in humans is the thyroid gland. Perchlorate inhibits the thyroid's uptake of iodine. Iodine is required as a building block for the synthesis of thyroid hormone. Perchlorate's inhibition of iodide uptake by the thyroid may be sufficient to produce hypothyroidism. Thyroid hormones regulate certain body functions after they are released into the blood. Because thyroid hormones play a critical role in the neurological development of the fetus, there is concern that hypothyroidism (maternal and fetal) during pregnancy could result in neurodevelopmental effects. Children and developing fetuses may be more likely to be affected by perchlorate than adults because thyroid hormones are essential for normal growth and development.
Perchlorates may affect the use of iodine by the thyroid gland and chronic exposures may result in symptoms of thyroid dysfunction such as goiter. If sufficient inhibition of iodide uptake by the goiter occurs, formation of thyroid hormones is depressed. These hormones are essential to the regulation of oxygen consumption and metabolism throughout the body. Clinical manifestations of hypothyroidism (or athyrea) include low metabolic rate, a tendency to gain weight, somnolence, and myxoedema (a relatively hard oedema of the subcutaneous tissue), dryness and loss of hair, low body temperature, hoarseness, muscle weakness, a slow return of the muscle after tendon jerk, and slow mentation. When hypothyroidism occurs in women, early in pregnancy, the foetus is at risk of impaired physical and mental development, the severity of the impairment depending on the degree of hypothyroidism.

Perchlorate is a negatively charged ion (ClO4-) that can affect thyroid function through competitive inhibition of the transport of iodine into the thyroid. Iodine is an important component of thyroid hormones T4 and T3, and the transfer of iodine from the circulation into the thyroid is an essential step in the synthesis of these two hormones. Iodine transport into the thyroid is mediated by a protein molecule known as the sodium(Na+)-iodide(I-) symporter (NIS). NIS molecules bind iodide with very high affinity, but they also bind other ions that have a similar shape and electric charge, such as perchlorate. The binding of these other ions to the NIS inhibits iodide transport into the thyroid, which can result in intrathyroidal iodide deficiency and consequently decreased synthesis of T4 and T3. There is remarkable compensation for iodide deficiency, however, where the body maintains the serum concentrations of thyroid hormones within narrow limits through feedback control mechanisms. This feedback includes increased secretion of thyroid stimulating hormone (TSH) from the pituitary gland, which has among its effects the increased production of T4 and T3. Sustained changes in thyroid hormone and TSH secretion can result in thyroid hypertrophy and hyperplasia, possibly followed by hypothyroidism in people unable to compensate with an increase in thyroid iodide uptake.
Perchlorate is not likely to pose a risk of thyroid cancer in humans, at least at doses below those necessary to alter thyroid hormone homeostasis, based on the hormonally-mediated mode of action in rodent studies and species differences in thyroid function. The epidemiological evidence is insufficient to determine whether or not there is a causal association between exposure to perchlorate and thyroid cancer. Sufficient evidence is available from rodent studies to indicate that goitrogenic doses of perchlorate cause follicular cell tumors of the thyroid, both following prolonged ingestion and from a two-generation study where a low incidence of early onset adenomas was reported. Perchlorate is non-mutagenic under standard tests. Extensive data indicate that thyroid-pituitary disruption is the sole mode of action for the observed thyroid tumors caused by perchlorate in rodents.

"Agents that lead to the development of thyroid neoplasia through an adaptive physiological mechanism belong to a different category from those that lead to neoplasia through genotoxic mechanisms or through mechanisms involving pathological responses with necrosis and repair. Agents that induce thyroid follicular cell tumors in rodents by interfering with thyroid hormone homeostasis, can with some exceptions, notably the sulfonamides, also interfere with thyroid hormone homeostasis in humans if given at a sufficient dose for a sufficient time. These agents can be assumed not to be carcinogenic in humans at concentrations that do not lead to alterations in thyroid hormone homeostasis." [IARC, 2001]

The pituitary-thyroid system of rats is similar to that of humans, i.e., decreases in thyroid hormone production result in increased secretion of TSH, which then increases thyroid production and release of T4 and T3. However, there are differences in binding proteins, binding affinities of the proteins for the hormones, turnover rates of the hormones, and thyroid stimulation by placental hormones that lead to important differences between the two species. These differences mean that rats are sensitive to the development of thyroid tumors because their thyroid function is easily disrupted. The NRC (2005) concluded that humans are much less susceptible than rats to disruption of thyroid function and, therefore, are not likely to develop thyroid tumors as a result of perchlorate exposure.

Perchlorate has been evaluated in standard *in vitro* and *in vivo* assays to assess genotoxicity. The results of these assays are negative. Ammonium perchlorate was not mutagenic in the Ames assay (with or without S9 activation). Negative results were also found in the mouse lymphoma gene mutation assay with and without S9 activation. Ammonium perchlorate did not induce chromosomal anomalies when evaluated for micronuclei induction in the bone marrow of mice when administered via drinking water gavage or intraperitoneal injection. No increases in micronuclei were found in Sprague-Dawley rats when evaluated from the 90-day study at the highest dose, which produced both thyroid hormone perturbations and follicular cell hyperplasia. Because perchlorate does not have the potential to be mutagenic or clastogenic, mutagenicity is not considered a possible mode of carcinogenic action for this chemical.
On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a

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satisfactory assessment.

Repeated exposure to tetrahydrofuran (THF) and its congeners has been associated with cytolytic hepatitis and fatty degeneration of the liver. Inhalation of THF at concentrations greater than 3000 ppm, 8 hours/day for 20 days, produced irritation and evidence for hepatic and renal injury in animals. Male rats inhaling more than 5000 ppm THF for 12 weeks, 4 hours/day showed signs of systemic intoxication, skin and respiratory irritation, liver function disturbance and abnormalities in glucose function. Muscle acetylcholinesterase activity increased in a concentration-dependent manner in male rats that inhaled 200 ppm for 18 weeks, 6 hours/day. Hepatic protein and mixed function oxidase activity also increased. At 2000 ppm, liver function was inhibited. In a 13-week inhalation study, ataxia was reported in rats at 5000 ppm and narcosis in mice at 1800 ppm. Hepatocytomegaly developed in mice of both sexes at 5000 ppm while uterine atrophy and degeneration of the adrenal cortex was found in female mice. A case history suggests that interaction of THF and endoflurane (an anaesthetic) may provoke epileptic seizures following surgery. The parent compound of tetrahydrofuran, furan, is carcinogenic in rats based on an increased incidence of cholangiocarcinoma and hepatocellular neoplasms of the liver and increased incidences of mononuclear cell leukaemia. In male and female mice, furan induced hepatocellular neoplasms and benign pheochromocytomas of the of the adrenal gland. 1,4-Dioxane, another cyclic ether solvent, is carcinogenic in rats and guinea pigs, following oral administration, inducing malignant tumours of the liver in rats and malignant tumours of the liver of the gall-bladder in guinea pigs. 1,4-Dioxane is a promoter in two stage skin carcinogenic studies in mice. In a two-year inhalation study * there was evidence of carcinogenic activity of THF, in male rats, based on increased incidences of renal tube adenoma or carcinoma (combined) and in female mice based on an increased incidence of hepatocellular neoplasms. There was no evidence of carcinogenic activity in female rats or male mice exposed to 200, 600 and 1800 ppm THF by inhalation. * National Toxicology Program Technical Report Series No. 475
Cyclic ethers, including tetrahydrofuran, furan and 1,4-dioxane, produce neoplasms and carcinomas in experimental animals, typically of the liver; other target organs include the adrenal gland, nasal cavity and gall-bladder. 1,4-Dioxane was a promoter in a two-stage skin carcinogenic study in mice. Results of studies with cyclic ethers indicate that carcinogenicity is often species and sex dependent. Furan has been used to induce apoptosis (programmed cell death). Oxetanes are under investigation.
Overexposure to respirable dust may cause coughing, wheezing, difficulty in breathing and impaired lung function. Chronic symptoms may include decreased vital lung capacity, chest infections
Repeated exposures, in an occupational setting, to high levels of fine- divided dusts may produce a condition known as pneumoconiosis which is the lodgement of any inhaled dusts in the lung irrespective of the effect. This is particularly true when a significant number of particles less than 0.5 microns (1/50,000 inch), are present. Lung shadows are seen in the X-ray.
Symptoms of pneumoconiosis may include a progressive dry cough, shortness of breath on exertion (exertional dyspnea), increased chest expansion, weakness and weight loss. As the disease progresses the cough produces a stringy mucous, vital capacity decreases further and shortness of breath becomes more severe. Other signs or symptoms include altered breath sounds, diminished lung capacity, diminished oxygen uptake during exercise, emphysema and pneumothorax (air in lung cavity) as a rare complication.
Removing workers from possibility of further exposure to dust generally leads to halting the progress of the lung abnormalities. Where worker-exposure potential is high, periodic examinations with emphasis on lung dysfunctions should be undertaken
Dust inhalation over an extended number of years may produce pneumoconiosis.. Pneumoconiosis is the accumulation of dusts in the lungs and the tissue reaction in its presence. It is further classified as being of noncollagenous or collagenous types. Noncollagenous pneumoconiosis, the benign form, is identified by minimal stromal reaction, consists mainly of reticulin fibres, an intact alveolar architecture and is potentially reversible.

Battery Pack P/N 452-0133 & 452-0222

TOXICITY	IRRITATION
Not Available	Not Available

manganese dioxide

TOXICITY	IRRITATION
Oral(Rat) LD50; >3478 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Skin: no adverse effect observed (not irritating)[1]

propylene carbonate

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >=2000 mg/kg[1]	Eye (rabbit): 60 mg - moderate
Oral(Rat) LD50; >5000 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
	Skin (human): 100 mg/3d-I moderate
	Skin (rabbit): 500 mg moderate
	Skin: no adverse effect observed (not irritating)[1]

1,2-dimethoxyethane

TOXICITY	IRRITATION
dermal (guinea pig) LD50: 5 mg/kg[2]	Not Available
Inhalation(Rat) LC50; 3000 ppm4h[2]	
Oral(Rabbit) LD50; 320 mg/kg[2]	

tetrahydrofuran

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
Inhalation(Rat) LC50; 45 mg/l4h[2]	Skin: no adverse effect observed (not irritating)[1]

Continued...

	Oral(Rat) LD50; 1.65 mg/kg[1]	
lithium	**TOXICITY**	**IRRITATION**
	Not Available	Eye: adverse effect observed (irritating)[1]
		Skin: adverse effect observed (corrosive)[1]
carbon black	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral(Rat) LD50; >8000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]
lithium perchlorate	**TOXICITY**	**IRRITATION**
	Oral(Rat) LD50; >300<2000 mg/kg[1]	Eye: adverse effect observed (irreversible damage)[1]
		Skin: adverse effect observed (corrosive)[1]
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*	

PROPYLENE CARBONATE	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. for propylene carbonate: Numerous adequate and reliable acute toxicity tests are available on propylene carbonate. Oral and dermal tests meet OECD and EPA test guidelines. Propylene carbonate is practically nontoxic following acute exposures; the oral LD50 is >.5000 mg/kg and the dermal LD50 is >3000 mg/kg. No further testing is recommended. Subchronic studies (13- 14 weeks) of propylene carbonate by inhalation (aerosol) and oral (gavage) routes were conducted in rats according to current guidelines. The oral study indicated low systemic toxicity from propylene carbonate (NOAEL = 5000 mg/kg/day). In the inhalation study, no systemic toxicity was seen at concentrations up to 1000 mg/m"; however, there was periocular irritation and swelling in a few males at 500 and 1000 mg/m3. A dermal carcinogenicity study in mice did not indicate tumorigenic potential or systemic toxicity from 2 years of exposure to propylene carbonate. No further testing is recommended. There is a negative Ames in vitro mutagenicity assay of propylene carbonate. A single intraperitoneal injection of 1666 mg/kg propylene carbonate did not induce an increase in micronuclei when examined after 30,48 and 72 hours. The mutagenicity battery is satisfactorily filled; no further mutagenicity testing is recommended. Gavage administration of propylene carbonate to pregnant rats days 6-15 of gestation resulted in systemic toxicity at doses of 3000 and 5000 mg/kg/day, including mortality (not seen in 13 week study of non-pregnant rats). The NOAEL for maternal toxicity was 1000 mg/kg/day. This indicates that pregnant rats are more susceptible to propylene carbonate than are non-pregnant rats. There were no significant differences in live litter size, average fetal weight, percentage of males, or malformed fetuses. No studies of the effect of propylene carbonate on reproduction are available. However, no adverse effects on testis, ovaries, or accessory sex organs were noted in rats following oral or inhalation of propylene carbonate for 13 weeks. Therefore, reproductive effects from propylene carbonate are unlikely
1,2-DIMETHOXYETHANE	Animal testing shows material is a reproductive effector: For ethylene glycol monoalkyl ethers and their acetates (EGMAEs): Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates. EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers. **Acute Toxicity:** Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE *in vitro* than those of rats. **Repeat dose toxicity:** The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA *in vitro* and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive

Continued...

to haemolysis by BAA *in vitro*.
Mutagenicity: In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in *S. typhimurium* strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. *In vitro* cytogenicity and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and in vivo micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.
Carcinogenicity: In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity
Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes).
Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m3 and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m3), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m3), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m3) indicate that the members of the category are not teratogenic.
The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m3 (rabbit-EGPE), 100 ppm or 425 mg/m3 (rat-EGPE), 50 ppm or 241 mg/m3 (rat EGBE) and 100 ppm or 483 mg/m3 (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m3 (rat and rabbit-EGHE).
For 1,2-dimethoxyethane (monoglyme):
Monoglyme, an ethylene glycol ether, demonstrates a low order of toxicity with an oral LD50 of greater than 4000 mg/kg in rats.
The acute inhalation toxicity of monoglyme was determined in a two-dose study in which the six-hour inhalation LC50 was found to be between 20 and 63 mg/L. The vapors produced some irritation and anesthesia at the high level. All high dose animals survived the exposure but died within 72 hours post-exposure.
A great deal of information is available on the repeated-dose toxicity of the biologically indicated metabolite of monoglyme, 2-methoxyethanol (2ME). In one study testicular degeneration was a prominent finding in rats even at the lowest dose tested (750 ppm, about 70 mg/kg/day) and in females, at this level, thymic atrophy was a finding. Thus, a NOEL was not found for rats of either sex. In the case of mice, the NOAEL for testicular degeneration and increased haematopoiesis in the spleen was 2000 ppm in males. A NOAEL was not reached for female mice since adrenal gland hypertrophy and increased haematopoiesis in the spleen occurred at the lowest concentration administered (2000 ppm, about 300 mg/kg/day).
Repeated-dose studies Repeated dose exposure in the drinking water was also associated with progressive anemia in rats and mice and increased mortality in rats at the two highest doses. The target organs can be identified as testes, bone marrow, spleen (hematopoiesis), thymus and adrenal. In general, the testes is considered a sensitive, if no the most sensitive, target organ.
A complete spectrum of toxicity can be confidently predicted from monoglyme's metabolism and studies on related compounds by all routes of exposure. Toxic responses include thymic atrophy, bone marrow suppression and testicular degeneration.
Although direct chemical evidence identifying the primary and secondary metabolites of monoglyme was not found, the metabolic pathways for this class of chemicals are well known. Additionally, there is very strong biological evidence linking adverse effects of monoglyme with a common active metabolite of methoxy glycol ethers.
It has been demonstrated that most of the toxic effects of the monoalkyl glycol ethers arise as a result of the metabolic conversion of the glycol ether into a substituted acetic acid derivative. In humans it is established that 2-methoxyacetic acid is the defining toxic metabolite and studies have shown that the level of 2-methoxyacetic acid in urine is an excellent. marker for exposure Demethylation by mixed-function oxidases, a relatively slow reaction, accounts for some metabolic conversion to ethylene glycol which is converted to oxalate. Dehydrogenase enzymes initially convert the free alcohol to the aldehyde and then the carboxylic acid . This is a very rapid conversion and it is known that a teratogenic dose of 2-methoxyethanol is completely oxidised, to 2-methoxyacetic acid, in a period of one-hour in rats. The competing reaction, demethylation of 2-methoxyethanol to ethylene glycol is comparatively slow as it is accomplished by the mixed-function oxidase system.
There is general agreement that 2-methoxyacetic acid is the proximate toxin. It is also established the clearance of 2-methoxyacetic acid is relatively slow as compared to its formation and the clearance in man may be much longer than in rats.
Genotoxicity has been evaluated using multiple *in vitro* experimental procedures covering both mutation and chromosomal aberration. Results of genotoxicity studies are mixed.
In *in vitro* studies, positive results are cited for the A *S. typhimurium* reverse mutation assay . Monoglyme is known to be cytotoxic to Salmonella typhimurium (30) at concentrations as low as 500 microliters per plate.
Monoglyme produced no evidence of genotoxicity in the presence or absence of S9 metabolic activation in mammalian cell point mutation tests using the HGPRT assay in CHO cells
Clastogenic activity was assessed *in vitro* using the Sister Chromatid Exchange in Chinese Hamster Ovary Cells Test (SCE). In this test, the material produced numerous indications of statistically significant effects on the frequency of SCE over the range of concentrations tested with and without addition of an active S9 metabolic system. A high number of cells were also observed with significant types of chromosomal aberrations suggesting that material was a clastogenic agent, especially in the presence of S9 activation
DNA damage was assessed using an *in vitro* unscheduled DNA synthesis (UDS) Assay. rat hepatocytes were treated with a wide concentration range of monoglyme up to concentrations demonstrating cytotoxicity in the assay system. Treatment did not produce either statistically significant or dose-related increases in the amount of UDS activity as measured by radioactive thymidine uptake
Reproductive toxicity: Adverse effects to reproduction are based on metabolism of monoglyme to 2-methoxy acetic acid, a compound that is known to interfere with sperm production. Adverse effects on the conceptus, including embryo lethality are also caused by this metabolite. Direct specific effects on female reproduction are not known to result from 2-methoxyacetic acid and thus are not expected from monoglyme exposure
Developmental toxicity:, Monoglyme appears to be a specific developmental toxin in rats and mice. Results in rats show that 120 mg/kg/day or more was associated with 100% foetal death and doses of 30 or 60 mg/kg
were foetotoxic but did not produce major malformations . This suggests that monoglyme is a specific developmental toxin as would be anticipated based on its metabolism to 2-methoxy acetic acid.

Continued...

	Studies with some ethylene glycol ethers and their esters indicate reproductive changes, testicular atrophy, infertility and kidney function changes. The metabolic acetic acid derivatives of the glycol ethers (alkoxyacetic acids), not the ether itself, have been found to be the proximal reproductive toxin in animals. The potency of these metabolites decrease significantly as the chain length of the ether increases. Consequently glycol ethers with longer substituents (e.g diethylene glycols, triethylene glycols) have not generally been associated with reproductive effects. One of the most sensitive indicators of toxic effects observed from many of the glycol ethers is an increase in the erythrocytic osmotic fragility in rats. This appears to be related to the development of haemoglobinuria (blood in the urine) at higher exposure levels or as a result of chronic exposure. Ethylene glycol ethers and acetates are mainly metabolised to alkoxyacetic acids but there is also a minor pathway through ethylene glycol to oxalic acid. The main pathway of ethylene glycol ethers is associated with significant clinical or experimental health effects, but the minor pathway is also interesting because formation of urinary stones depends principally upon urinary concentration of oxalate and calcium. In one study (1) the tendency to form urinary stones was 2.4 times higher amongst silk-screen printers exposed to ethylene glycol ethers, than among office workers. (1) Laitinen J., et al: Occupational Environmental Medicine 1996, 53 595-600
TETRAHYDROFURAN	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may produce severe 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) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration. Oral (human) LDLo: 50 mg/kg* [CCINFO]* Nil reported
CARBON BLACK	Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
MANGANESE DIOXIDE & LITHIUM & CARBON BLACK & LITHIUM PERCHLORATE	No significant acute toxicological data identified in literature search.
TETRAHYDROFURAN & LITHIUM & LITHIUM PERCHLORATE	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.

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

Legend: ✘ *– Data either not available or does not fill the criteria for classification*
✔ *– Data available to make classification*

SECTION 12 Ecological information

Toxicity

Battery Pack P/N 452-0133 & 452-0222

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

manganese dioxide

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>0.022mg/l	2
EC50(ECx)	48h	Crustacea	>0.022mg/l	2

propylene carbonate

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

Continued...

	LC50	96h	Fish	>1000mg/l	2
	EC50	48h	Crustacea	>1000mg/l	1
	EC50(ECx)	72h	Algae or other aquatic plants	>900mg/l	1

1,2-dimethoxyethane				
Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
EC50	72h	Algae or other aquatic plants	9120mg/l	2
LC50	96h	Fish	>500mg/l	2
EC50	48h	Crustacea	4000mg/l	2
NOEC(ECx)	504h	Crustacea	320mg/l	2

tetrahydrofuran				
Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
LC50	96h	Fish	2160mg/l	2
NOEC(ECx)	24h	Fish	>=5mg/l	1

lithium				
Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
NOEC(ECx)	72h	Algae or other aquatic plants	1.65mg/l	2
EC50	72h	Algae or other aquatic plants	25.6mg/l	2
LC50	96h	Fish	18mg/l	2
EC50	48h	Crustacea	19.1mg/l	2

carbon black				
Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
EC50	72h	Algae or other aquatic plants	>0.2mg/l	2
LC50	96h	Fish	>100mg/l	2
EC50	48h	Crustacea	33.076-41.968mg/l	4
NOEC(ECx)	24h	Crustacea	3200mg/l	1

lithium perchlorate				
Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
EC50(ECx)	48h	Crustacea	>100mg/l	2
EC50	72h	Algae or other aquatic plants	>120mg/l	2
EC50	48h	Crustacea	>100mg/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*

Must not be disposed together with household garbage. Do not allow product to reach sewage system.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
propylene carbonate	HIGH	HIGH
1,2-dimethoxyethane	LOW	LOW
tetrahydrofuran	LOW	LOW
lithium perchlorate	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
propylene carbonate	LOW (LogKOW = -0.41)
1,2-dimethoxyethane	LOW (LogKOW = -0.21)
tetrahydrofuran	LOW (LogKOW = 0.46)
lithium perchlorate	LOW (LogKOW = -4.6296)

Mobility in soil

Ingredient	Mobility
propylene carbonate	LOW (KOC = 14.85)

Continued...

Ingredient	Mobility
1,2-dimethoxyethane	HIGH (KOC = 1)
tetrahydrofuran	LOW (KOC = 4.881)
lithium perchlorate	LOW (KOC = 48.64)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	· Recycle wherever possible or consult manufacturer for recycling options. · Consult State Land Waste Management Authority for disposal. **FOR DISPOSAL OF SMALL QUANTITIES**: ▸ Cautiously acidify a 3% solution or a suspension of the material to pH 2 with sulfuric acid. ▸ Gradually add a 50% excess of aqueous sodium bisulfite with stirring at room temperature. (Other reducers such as thiosulfate or ferrous salts may substitute; **do NOT** use carbon, sulfur or other strong reducing agents). An increase in temperature indicates reaction is taking place. If no reaction is observed on the addition of about 10% of the sodium bisulfite solution, initiate it by cautiously adding more acid. ▸ If manganese, chromium or molybdenum are present adjust the pH of the solution to 7 and treat with sulfide to precipitate for burial as a hazardous waste. Destroy excess sulfide, neutralise and flush the solution down the drain (subject to State and Local Regulation). [Sigma/Aldrich] ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	NO
HAZCHEM	4Y

Land transport (ADG)

UN number	3090
UN proper shipping name	LITHIUM METAL BATTERIES (including lithium alloy batteries)
Transport hazard class(es)	Class: 9 Subrisk: Not Applicable
Packing group	Not Applicable
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: 188 230 310 376 377 384 387 Limited quantity: 0

Air transport (ICAO-IATA / DGR)

UN number	3090
UN proper shipping name	Lithium metal batteries (including lithium alloy batteries)
Transport hazard class(es)	ICAO/IATA Class: 9 ICAO / IATA Subrisk: Not Applicable ERG Code: 12FZ

Continued...

Packing group	Not Applicable
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: A88 A99 A154 A164 A183 A201 A206 A213 A334 A802 Cargo Only Packing Instructions: See 968 Cargo Only Maximum Qty / Pack: See 968 Passenger and Cargo Packing Instructions: Forbidden Passenger and Cargo Maximum Qty / Pack: Forbidden Passenger and Cargo Limited Quantity Packing Instructions: Forbidden Passenger and Cargo Limited Maximum Qty / Pack: Forbidden

Sea transport (IMDG-Code / GGVSee)

UN number	3090
UN proper shipping name	LITHIUM METAL BATTERIES (including lithium alloy batteries)
Transport hazard class(es)	IMDG Class: 9 IMDG Subrisk: Not Applicable
Packing group	Not Applicable
Environmental hazard	Not Applicable
Special precautions for user	EMS Number: F-A , S-I Special provisions: 188 230 310 376 377 384 387 Limited Quantities: 0

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

Not Applicable

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

Product name	Group
manganese dioxide	Not Available
propylene carbonate	Not Available
1,2-dimethoxyethane	Not Available
tetrahydrofuran	Not Available
lithium	Not Available
carbon black	Not Available
lithium perchlorate	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
manganese dioxide	Not Available
propylene carbonate	Not Available
1,2-dimethoxyethane	Not Available
tetrahydrofuran	Not Available
lithium	Not Available
carbon black	Not Available
lithium perchlorate	Not Available

SECTION 15 Regulatory information

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

manganese dioxide is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

Continued...

propylene carbonate is found on the following regulatory lists

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

1,2-dimethoxyethane is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

tetrahydrofuran is found on the following regulatory lists

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

lithium is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australian Inventory of Industrial Chemicals (AIIC)
FEI Equine Prohibited Substances List - Banned Substances
FEI Equine Prohibited Substances List (EPSL)

carbon black is found on the following regulatory lists

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

lithium perchlorate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (manganese dioxide; propylene carbonate; 1,2-dimethoxyethane; tetrahydrofuran; lithium; carbon black; lithium perchlorate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (lithium)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (lithium perchlorate)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (lithium perchlorate)
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	11/01/2019
Initial Date	03/02/2015

Continued...

SDS Version Summary

Version	Date of Update	Sections Updated
4.1.1.1	01/13/2017	Classification, Storage (storage incompatibility)
5.1.1.1	11/01/2019	One-off system update. NOTE: This may or may not change the GHS classification
5.1.2.1	04/26/2021	Regulation Change
5.1.3.1	05/03/2021	Regulation Change
5.1.4.1	05/06/2021	Regulation Change
5.1.5.1	05/10/2021	Regulation Change
5.1.5.2	05/30/2021	Template Change
5.1.5.3	06/04/2021	Template Change
5.1.5.4	06/05/2021	Template Change
5.1.6.4	06/07/2021	Regulation Change
5.1.6.5	06/09/2021	Template Change
5.1.6.6	06/11/2021	Template Change
5.1.6.7	06/15/2021	Template Change
5.1.7.7	06/17/2021	Regulation Change
5.1.8.7	06/21/2021	Regulation Change
5.1.8.8	07/05/2021	Template Change
5.1.9.8	07/14/2021	Regulation Change
5.1.10.8	07/19/2021	Regulation Change
5.1.10.9	08/01/2021	Template Change
5.1.11.9	08/02/2021	Regulation Change
5.1.12.9	08/05/2021	Regulation Change
5.1.13.9	08/09/2021	Regulation Change

Other information

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

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

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
ES: Exposure Standard
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index
AIIC: Australian Inventory of Industrial Chemicals
DSL: Domestic Substances List
NDSL: Non-Domestic Substances List
IECSC: Inventory of Existing Chemical Substance in China
EINECS: European INventory of Existing Commercial chemical Substances
ELINCS: European List of Notified Chemical Substances
NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory

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KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals
PICCS: Philippine Inventory of Chemicals and Chemical Substances
TSCA: Toxic Substances Control Act
TCSI: Taiwan Chemical Substance Inventory
INSQ: Inventario Nacional de Sustancias Químicas
NCI: National Chemical Inventory
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

end of SDS