

ACR Electronics Battery Pack 452-6499 & 455-0012

ACR Electronics

Chemwatch: 33-67150

Version No: 3.1

Safety Data Sheet according to OSHA HazCom Standard (2012) requirements

Chemwatch Hazard Alert Code: 3

Issue Date: 03/10/2023

Print Date: 04/04/2024

L.GHS.USA.EN

SECTION 1 Identification

Product Identifier

Product name	ACR Electronics Battery Pack 452-6499 & 455-0012
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

Recommended use of the chemical and restrictions on use

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.
--------------------------	---

Name, address, and telephone number of the chemical manufacturer, importer, or other responsible party

Registered company name	ACR Electronics
Address	5757 Ravenswood Road Fort Lauderdale FL 33312 United States
Telephone	+1 954 981 3333 +1 800 432 0227
Fax	+1 954 983 5087
Website	http://www.acrartex.com/
Email	msds@acrartex.com

Emergency phone number

Association / Organisation	ChemTel Inc.	CHEMWATCH EMERGENCY RESPONSE (24/7)
Emergency telephone numbers	+ 800-255-3924 (24 hours)	+1 855-237-5573
Other emergency telephone numbers	Not Available	+61 3 9573 3188

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

Una vez conectado y si el mensaje no está en su idioma preferido, por favor marque 02

SECTION 2 Hazard(s) identification

Classification of the substance or mixture

NFPA 704 diamond



Note: The hazard category numbers found in GHS classification in section 2 of this SDSs are NOT to be used to fill in the NFPA 704 diamond. Blue = Health Red = Fire Yellow = Reactivity White = Special (Oxidizer or water reactive substances)

Classification	Substances and Mixtures which in Contact with Water Emit Flammable Gases Category 2, Oxidizing Solids Category 3, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Acute Toxicity (Inhalation) Category 4, Carcinogenicity Category 1A, Reproductive Toxicity Category 1B
-----------------------	--

Label elements

Hazard pictogram(s)	
----------------------------	--

Signal word	Danger
--------------------	---------------

Hazard statement(s)

H261	In contact with water releases flammable gases.
H272	May intensify fire; oxidiser.
H302	Harmful if swallowed.
H315	Causes skin irritation.
H318	Causes serious eye damage.
H332	Harmful if inhaled.
H350	May cause cancer.
H360	May damage fertility or the unborn child.

Hazard(s) not otherwise classified

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat.
P221	Take any precaution to avoid mixing with combustibles/organic material.
P231+P232	Handle under inert gas. Protect from moisture.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P220	Keep/Store away from clothing/organic material/combustible materials.
P261	Avoid breathing dust/fumes.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P202	Do not handle until all safety precautions have been read and understood.
P223	Do not allow contact with water.

Precautionary statement(s) Response

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.
P335+P334	Brush off loose particles from skin. Immerse in cool water/wrap in wet bandages.
P370+P378	In case of fire: Use dry agent to extinguish.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P330	Rinse mouth.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

P405	Store locked up.
P402+P404	Store in a dry place. Store in a closed container.

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	<u>manganese dioxide</u>
7439-93-2	<10	<u>lithium</u>
1333-86-4	<10	<u>carbon black</u>
110-71-4	<10	<u>1,2-dimethoxyethane</u>
109-99-9	<10	<u>tetrahydrofuran</u>
108-32-7	<10	<u>propylene carbonate</u>
7791-03-9	<10	<u>lithium perchlorate</u>

The specific chemical identity and/or exact percentage (concentration) of composition has been withheld as a trade secret.

SECTION 4 First-aid measures**Description of first aid measures**

Eye Contact	If this product comes in contact with the eyes: ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. ▶ DO NOT attempt to remove particles attached to or embedded in eye . ▶ Lay victim down, on stretcher if available and pad BOTH eyes, make sure dressing does not press on the injured eye by placing thick pads under dressing, above and below the eye. ▶ Seek urgent medical assistance, or transport to hospital.
Skin Contact	If skin or hair contact occurs: ▶ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▶ Quickly remove all contaminated clothing, including footwear. ▶ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▶ Transport to hospital, or doctor.
Inhalation	▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prosthesis 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, without delay.
Ingestion	▶ 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.

Continued...

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.

Most important symptoms and effects, both acute and delayed

See Section 11

Indication of any immediate medical attention and special treatment needed

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.
- ▶ 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 Fire-fighting 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	▸ 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.
-----------------------------	---

Special protective equipment and precautions for fire-fighters

Fire Fighting	▸ When silica dust is dispersed in air, firefighters should wear inhalation protection as hazardous substances from the fire may 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.
Fire/Explosion Hazard	▸ 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. Combustion products include: carbon monoxide (CO) carbon dioxide (CO2) hydrogen chloride phosgene metal oxides other pyrolysis products typical of burning organic material. WARNING: Long standing in contact with air and light may result in the formation of potentially explosive peroxides.

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

Continued...

See section 12

Methods and material for containment and cleaning up

Minor Spills	▶ Clean up waste regularly and abnormal spills immediately. ▶ Avoid breathing dust and contact with skin and eyes. ▶ Wear protective clothing, gloves, safety glasses and dust respirator. ▶ Use dry clean up procedures and avoid generating dust. ▶ Vacuum up or sweep up. NOTE: Vacuum cleaner must be fitted with an exhaust micro filter (H-Class HEPA type) (consider explosion-proof machines designed to be grounded during storage and use). H-Class HEPA filtered industrial vacuum cleaners should NOT be used on wet materials or surfaces. ▶ Dampen with water to prevent dusting before sweeping. ▶ Place in suitable containers 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 · 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.

Personal Protective Equipment advice is contained in Section 8 of the SDS.

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	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 tendency of many ethers to form explosive peroxides is well documented. Ethers lacking non-methyl hydrogen atoms adjacent to the ether link are thought to be relatively safe ▶ DO NOT concentrate by evaporation, or evaporate extracts to dryness, as residues may contain explosive peroxides with DETONATION potential. ▶ Any static discharge is also a source of hazard. ▶ Before any distillation process remove trace peroxides by shaking with excess 5% aqueous ferrous sulfate solution or by percolation through a column of activated alumina. ▶ Distillation results in uninhibited ether distillate with considerably increased hazard because of risk of peroxide formation on storage. ▶ Add inhibitor to any distillate as required. ▶ When solvents have been freed from peroxides by percolation through columns of activated alumina, the absorbed peroxides must promptly be desorbed by treatment with polar solvents such as methanol or water, which should then be disposed of safely. 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.
---------------	---

Continued...

- ▶ 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.**
 - ▶ 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.
 - ▶ Organic powders when finely divided over a range of concentrations regardless of particulate size or shape and suspended in air or some other oxidizing medium may form explosive dust-air mixtures and result in a fire or dust explosion (including secondary explosions)
 - ▶ Minimise airborne dust and eliminate all ignition sources. Keep away from heat, hot surfaces, sparks, and flame.
 - ▶ Establish good housekeeping practices.
 - ▶ Remove dust accumulations on a regular basis by vacuuming or gentle sweeping to avoid creating dust clouds.
 - ▶ Use continuous suction at points of dust generation to capture and minimise the accumulation of dusts. Particular attention should be given to overhead and hidden horizontal surfaces to minimise the probability of a "secondary" explosion. According to NFPA Standard 654, dust layers 1/32 in. (0.8 mm) thick can be sufficient to warrant immediate cleaning of the area.
 - ▶ Do not use air hoses for cleaning.
 - ▶ Minimise dry sweeping to avoid generation of dust clouds. Vacuum dust-accumulating surfaces and remove to a chemical disposal area. Vacuums with explosion-proof motors should be used.
 - ▶ Control sources of static electricity. Dusts or their packages may accumulate static charges, and static discharge can be a source of ignition.
 - ▶ Solids handling systems must be designed in accordance with applicable standards (e.g. NFPA including 654 and 77) and other national guidance.
 - ▶ Do not empty directly into flammable solvents or in the presence of flammable vapors.
 - ▶ The operator, the packaging container and all equipment must be grounded with electrical bonding and grounding systems. Plastic bags and plastics cannot be grounded, and antistatic bags do not completely protect against development of static charges.
- Empty containers may contain residual dust which has the potential to accumulate following settling. Such dusts may explode in the presence of an appropriate ignition source.
- ▶ **Do NOT cut, drill, grind or weld such containers.**
 - ▶ In addition ensure such activity is not performed near full, partially empty or empty containers without appropriate workplace safety authorisation or permit.

Other information

- ▶ Store in original containers.
- ▶ Keep containers securely sealed.
- ▶ Store in a cool, dry area protected from environmental extremes.
- ▶ 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.

For major quantities:

- ▶ Consider storage in bunded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams).
- ▶ Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities.

Conditions for safe storage, including any incompatibilities**Suitable container**

- ▶ Bulk bags: Reinforced bags required for dense materials.
- ▶ Polyethylene or polypropylene container.
- ▶ Check all containers are clearly labelled and free from leaks.

Storage incompatibility

Lithium metal:

- ▶ may ignite spontaneously in air when it exists as finely divided particles or powders
- ▶ forms corrosive fumes of lithium hydroxide when in contact with water
- ▶ reacts violently with water, releasing heat, flammable hydrogen gas, corrosive fumes and forming strong caustic solutions
- ▶ is air sensitive and reacts with nitrogen gas at high temperatures

- ▶ reacts violently with oxidisers, acetonitrile, arsenic, alcohols, beryllium, bromine pentafluoride, bromobenzene, carbides, carbon dioxide, carbon tetrachloride, chlorine, chlorine trifluoride, chlorine pentafluoride, chromium, chromium trichloride, cobalt and its alloys, diazomethane, diborane, glycols, iron alloys, magnesium alloys, maleic anhydride, mercury, metal oxides, nickel alloys, nitric acid, nitrogen, organic matter, oxygen, phosphorus, silicates, sulfur, vanadium and many other substances
- ▶ in moist conditions readily undergoes an exothermic reaction with nitrogen at room temperature to form the nitride
- ▶ forms friction- and impact-sensitive explosive mixtures with halogens, halocarbons, diborane, methyl dichloride, methyl diiodide, and other substances
- ▶ is incompatible with atmospheric gases, diazomethane, hydrogen, iron(II) sulfide, manganese telluride, metal chlorides, nonmetal oxides, platinum, trimethyl hypofluorite and other substances
- ▶ attacks plastics, rubber
- ▶ when molten attacks ceramic materials, concrete, sand
- ▶ attacks metal alloys, cobalt, iron manganese, nickel
- ▶ reacts violently with most standard fire extinguishers: water, carbon dioxide, foam, halogenated agents, sodium carbonate, sodium chloride and other dry powders (in case of fire Class D extinguishers or smothering quantities of dry sand, crushed limestone or clay recommended).

NOTE: . Argon should be used as an inert atmosphere for lithium metal reactions

For tetrahydrofuran (THF)

- ▶ Avoid contact with oxygen, air, light and heat
- ▶ Contact with lithium aluminium hydride or with sodium or potassium hydroxide can be hazardous when peroxides are present. THF may polymerise in the presence of cationic initiators such as Lewis acids or strong proton acids.
- ▶ Segregate from lithium aluminium hydride, sodium or potassium hydroxide, cationic initiators such as Lewis acids or strong proton acids.
- ▶ In the absence of inhibitors tetrahydrofuran is subject to auto-oxidation with the formation of 2-tetrahydrofuryl hydroperoxide . When heated this tends to decompose smoothly but if allowed to accumulate over a considerable period it transforms to other peroxidic species, such as unstable and explosive polyalkylidene peroxide, which violently decompose.
- ▶ Copper(I) chloride has been recommended to remove trace amounts of peroxide. An attempt to remove peroxides by shaking with solid ferrous sulfate, prior to distillation, did not prevent explosion of the distillation residue. Alkali treatment does not appear to be safe.
- ▶ Peroxides may be destroyed by passage through activated carbon at 20-66 C with contact time in excess of 2 min.
- ▶ is incompatible with borane, calcium hydride, lithium tetrahydroaluminate, sodium aluminium tetrahydride
- ▶ reacts violently with strong oxidisers, bromine, oxygen, magnesium tetrahydroaluminate, metal halides, peroxyacetic acid, potassium hydride
- ▶ attacks some plastics and coatings
- ▶ may accumulate static charges that can result in ignition of its vapours

Storage tanks and other equipment should be absolutely dry and free from air, ammonia, acetylene, hydrogen sulfide, rust and other contaminants.

1,2-Dimethoxyethane (monoglyme):

- ▶ reacts violently with strong oxidisers, lithium tetrahydroaluminate
- ▶ is incompatible with sulfuric acid, isocyanates
- ▶ may form unstable peroxides on standing
- ▶ may generate electrostatic charges due to low conductivity

Silicas:

- ▶ react with hydrofluoric acid to produce silicon tetrafluoride gas
- ▶ react with xenon hexafluoride to produce explosive xenon trioxide
- ▶ reacts exothermically with oxygen difluoride, and explosively with chlorine trifluoride (these halogenated materials are not commonplace industrial materials) and other fluorine-containing compounds
- ▶ may react with fluorine, chlorates
- ▶ are incompatible with strong oxidisers, manganese trioxide, chlorine trioxide, strong alkalis, metal oxides, concentrated orthophosphoric acid, vinyl acetate
- ▶ may react vigorously when heated with alkali carbonates.
- ▶ Inorganic peroxy compounds are potent oxidisers that pose fire or explosive hazards when in contact with ordinary combustible materials.
- ▶ Inorganic peroxides react with organic compounds to generate organic peroxide and hydroperoxide products that react violently with reducing agents.
- ▶ Inorganic oxidising agents can react with reducing agents to generate heat and products that may be gaseous (causing pressurization of closed containers). The products may themselves be capable of further reactions (such as combustion in the air).
- ▶ Organic compounds in general have some reducing power and can in principle react with compounds in this class. Actual reactivity varies greatly with the identity of the organic compound.
- ▶ Inorganic oxidising agents can react violently with active metals, cyanides, esters, and thiocyanates.
- ▶ Peroxides, in contact with inorganic cobalt and copper compounds, iron and iron compounds, acetone, metal oxide salts and acids and bases can react with rapid, uncontrolled decomposition, leading to fires and explosions.
- ▶ Inorganic reducing agents react with oxidizing agents to generate heat and products that may be flammable, combustible, or otherwise reactive. Their reactions with oxidizing agents may be violent.
- ▶ Incidents involving interaction of active oxidants and reducing agents, either by design or accident, are usually very energetic and examples of so-called redox reactions.
- ▶ WARNING: Avoid or control reaction with peroxides. All **transition metal** peroxides should be considered as potentially explosive. For example transition metal complexes of alkyl hydroperoxides may decompose explosively.

- ▶ The pi-complexes formed between chromium(0), vanadium(0) and other transition metals (haloarene-metal complexes) and mono- or poly-fluorobenzene show extreme sensitivity to heat and are explosive.
- ▶ Avoid reaction with borohydrides or cyanoborohydrides
- ▶ Contact with water liberates highly flammable gases
- ▶ Many metals may incandesce, react violently, ignite or react explosively upon addition of concentrated nitric acid.

NOTE: May contain traces of perchloric acid or may, on contact with acids, produce an anhydrous perchloric acid an extremely reactive and explosive species. Many of the reported explosions involving perchlorate may result its ability to form unstable perchlorate esters or salts of the anhydrous acid.

WARNING:

- ▶ On the basis of experience with cobalt(III) perchlorate, attention is drawn to the possibility of stable metal perchlorates being converted by unintentional dehydration to unstable (endothermic) lower hydrates capable of explosive decomposition in the absence of impurities. Great care must be taken to avoid dehydration or desolvation of perchlorates.
- ▶ Metal perchlorates may be explosively reactive with finely divided aluminium, magnesium and zinc and other metals, calcium and strontium hydrides, glycol (on heating), sulfuric acid (with the formation of unstable perchloric acid), and trifluoromethanesulfonic acid.
- ▶ The perchlorate salts of the complexes of divalent cobalt, nickel and particularly iron ([tetramethyl[14]-N8 complexes) are potentially explosive and storage for more than 4 weeks is inadvisable
- ▶ Avoid any contamination of this material as it is very reactive and any contamination is potentially hazardous
- ▶ Glycol ethers may form peroxides under certain conditions; the potential for peroxide formation is enhanced when these substances are used in processes such as distillation where they are concentrated or even evaporated to near-dryness or dryness; storage under a nitrogen atmosphere is recommended to minimise the possible formation of highly reactive peroxides
- ▶ Nitrogen blanketing is recommended if transported in containers at temperatures within 15 deg C of the flash-point and at or above the flash-point - large containers may first need to be purged and inerted with nitrogen prior to loading
- ▶ In the presence of strong bases or the salts of strong bases, at elevated temperatures, the potential exists for runaway reactions.
- ▶ Contact with aluminium should be avoided; release of hydrogen gas may result- glycol ethers will corrode scratched aluminium surfaces.
- ▶ May discolour in mild steel/ copper; lined containers, glass or stainless steel is preferred
- ▶ Glycols and their ethers undergo violent decomposition in contact with 70% perchloric acid. This seems likely to involve formation of the glycol perchlorate esters (after scission of ethers) which are explosive, those of ethylene glycol and 3-chloro-1,2-propanediol being more powerful than glyceryl nitrate, and the former so sensitive that it explodes on addition of water . Investigation of the hazards associated with use of 2-butoxyethanol for alloy electropolishing showed that mixtures with 50-95% of acid at 20 deg C, or 40-90% at 75 C, were explosive and initiable by sparks. Sparking caused mixtures with 40-50% of acid to become explosive, but 30% solutions appeared safe under static conditions of temperature and concentration.
- ▶ Segregate chlorates from organic matter, acids, poisonous gases, flammables, corrosives, aluminium and ammonium salts and any other combustible material.
- ▶ Mixtures of chlorates with fibrous and absorbent organic materials such as wood, paper, leather, flour, sawdust, sugar, shellac, may be ignited or caused to explode by static sparks, friction or shock.
- ▶ The extreme hazardous nature of mixtures of metal chlorates with phosphorus or sulfur, apart from being powerful explosives, are dangerously sensitive to friction or shock; spontaneous ignition occasionally occurs.
- ▶ Mixtures with sucrose, lactose, chromium, sulfur dioxide, sodium amide, zirconium, germanium and titanium explode on heating.
- ▶ Forms incompatible sometimes explosive mixtures with thorium dicarbide, strontium hydride, hydrogen iodide, fluorine, cyanoguanidine, cyanides, dinickel trioxide, powdered carbon, aqua regia and ruthenium, nitric acid, manganese dioxide and potassium hydroxide or boron.
- ▶ Chlorates should not be allowed to come into contact with ammonium salts, aluminium and other powdered metals, phosphorous, silicon, sulfur, sulfides, sulfuric acid, nitrobenzene, iodides and tartaric acid
- ▶ Mixtures with hydrocarbons, metal phosphides (Zn, Ag, Al, Cu, Hg, Mg, etc), metal thiocyanates, metal sulfides, arsenic, carbon, phosphorous, sulfur, ammonium salts, powdered metals, arsenic trioxide, phosphorous, silicon, sulfur, sulfides, sulfites and hyposulfites are easily ignited (by friction impact or heat) and are potentially explosive.
- ▶ Metal chlorates in contact with strong acids liberate explosive chlorine dioxide gas. With concentrated sulfuric acid a violent explosion can occur unless effective cooling is used. Mixing potassium chlorate and concentrated sulfuric acid results in an explosion with optimum temperature range being 120-130. Heating a moist mixture of metal chlorate and a dibasic organic acid (tartaric or citric acid) liberates chlorine dioxide diluted with carbon dioxide.
- ▶ Fusion of chlorates with metal cyanides may lead to an explosion.
- ▶ Chlorates containing 1-2% bromate or sulfur are liable to spontaneous explosion.
- ▶ Chlorates releases oxygen, chlorine and chlorine dioxide when heated above 300 deg. C..
- ▶ In presence of moisture may release oxygen and ozone

Metals exhibit varying degrees of activity. Reaction is reduced in the massive form (sheet, rod, or drop), compared with finely divided forms. The less active metals will not burn in air but:

- ▶ can react exothermically with oxidising acids to form noxious gases.
- ▶ catalyse polymerisation and other reactions, particularly when finely divided
- ▶ react with halogenated hydrocarbons (for example, copper dissolves when heated in carbon tetrachloride), sometimes forming explosive compounds.
- ▶ Many metals in elemental form react exothermically with compounds having active hydrogen atoms (such as acids and water) to form flammable hydrogen gas and caustic products.
- ▶ Elemental metals may react with azo/diazo compounds to form explosive products.
- ▶ Some elemental metals form explosive products with halogenated hydrocarbons.

- ▶ The unhindered oxygen atom found on cyclic ethers such as the epoxides, oxetanes, furans, dioxanes and pyrans, carries two unshared pairs of electrons - a structure which favors the formation of coordination complexes and the solvation of cations.
- ▶ Cyclic ethers are used as important solvents, as chemical intermediate and as monomers for ring-opening polymerization.
- ▶ They are unstable at room temperature due to possibility of peroxide formation; stabiliser is sometimes needed for storage and transportation.

NOTE: Ethers lacking non-methyl hydrogen atoms adjacent to the ether link are thought to be relatively safe

- ▶ Intimate mixtures of chlorates, bromates or iodates of barium, cadmium, calcium, magnesium, potassium, sodium or zinc, with finely divided aluminium, arsenic, copper, carbon, phosphorus, sulfur, hydrides of alkali- and alkaline earth-metals; sulfides of antimony, arsenic, copper or tin; metal cyanides, thiocyanates; or impure manganese dioxide may react explosively or violently, either spontaneously (especially in the presence of moisture) or on initiation by heat, impact or friction, sparks or addition of sulfuric acid.

BRETHERRICKS HANDBOOK OF REACTIVE CHEMICAL HAZARDS, 4th Edition

- ▶ Finely divided metal powders develop pyrophoricity when a critical specific surface area is exceeded; this is ascribed to high heat of oxide formation on exposure to air.
- ▶ Safe handling is possible in relatively low concentrations of oxygen in an inert gas.
- ▶ Several pyrophoric metals, stored in glass bottles have ignited when the container is broken on impact. Storage of these materials moist and in metal containers is recommended.
- ▶ The reaction residues from various metal syntheses (involving vacuum evaporation and co-deposition with a ligand) are often pyrophoric.

Factors influencing the pyrophoricity of metals are particle size, presence of moisture, nature of the surface of the particle, heat of formation of the oxide, or nitride, mass, hydrogen content, stress, purity and presence of oxide, among others.

- ▶ Keep dry
- ▶ **NOTE:** May develop pressure in containers; open carefully. Vent periodically.
- ▶ Avoid storage with reducing agents.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US OSHA Permissible Exposure Limits (PELs) Table Z-1	manganese dioxide	Manganese compounds (as Mn)	Not Available	Not Available	5 mg/m3	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	manganese dioxide	Particulates Not Otherwise Regulated (PNOR)- Respirable fraction	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	manganese dioxide	Particulates Not Otherwise Regulated (PNOR)- Total dust	15 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	manganese dioxide	Inert or Nuisance Dust: Respirable fraction	5 mg/m3 / 15 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	manganese dioxide	Inert or Nuisance Dust: Total Dust	15 mg/m3 / 50 mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	manganese dioxide	Manganese tetroxide (as Mn)	Not Available	Not Available	Not Available	See Appendix D
US OSHA Permissible Exposure Limits (PELs) Table Z-1	lithium	Particulates Not Otherwise Regulated (PNOR)- Total dust	15 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	lithium	Particulates Not Otherwise Regulated (PNOR)- Respirable fraction	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	lithium	Inert or Nuisance Dust: Total Dust	15 mg/m3 / 50 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	lithium	Inert or Nuisance Dust: Respirable fraction	5 mg/m3 / 15 mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	lithium	Particulates not otherwise regulated	Not Available	Not Available	Not Available	See Appendix D

Continued...

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US OSHA Permissible Exposure Limits (PELs) Table Z-1	carbon black	Carbon black	3.5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	carbon black	Inert or Nuisance Dust: Respirable fraction	5 mg/m3 / 15 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	carbon black	Inert or Nuisance Dust: Total Dust	15 mg/m3 / 50 mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	carbon black	Carbon black	3.5 mg/m3	Not Available	Not Available	Ca; TWA 0.1 mg PAHs/m3 [Carbon black in presence of polycyclic aromatic hydrocarbons (PAHs)] See Appendix A See Appendix C
US OSHA Permissible Exposure Limits (PELs) Table Z-1	tetrahydrofuran	Tetrahydrofuran	200 ppm / 590 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	tetrahydrofuran	Tetrahydrofuran	200 ppm / 590 mg/m3	735 mg/m3 / 250 ppm	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
lithium	3.3 mg/m3	36 mg/m3	220 mg/m3
carbon black	9 mg/m3	99 mg/m3	590 mg/m3
1,2-dimethoxyethane	13 ppm	140 ppm	840 ppm
tetrahydrofuran	Not Available	Not Available	Not Available
propylene carbonate	34 mg/m3	370 mg/m3	2,200 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
lithium	Not Available	Not Available
carbon black	1,750 mg/m3	Not Available
1,2-dimethoxyethane	Not Available	Not Available
tetrahydrofuran	2,000 ppm	Not Available
propylene carbonate	Not Available	Not Available
lithium perchlorate	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
1,2-dimethoxyethane	E	≤ 0.1 ppm
propylene carbonate	E	≤ 0.1 ppm
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

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if
----------------------------------	--

designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.

- ▶ Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area.
- ▶ Work should be undertaken in an isolated system such as a "glove-box". Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system.
- ▶ Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within.
- ▶ Open-vessel systems are prohibited.
- ▶ Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation.
- ▶ Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system.
- ▶ For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood.
- ▶ Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas).
- ▶ Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air.
- ▶ Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed.

Individual protection measures, such as personal protective equipment



Eye and face protection

- ▶ Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure.
- ▶ Chemical goggles. Whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted. [AS/NZS 1337.1, EN166 or national equivalent]
- ▶ Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection.
- ▶ Alternatively a gas mask may replace splash goggles and face shields.
- ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].

Skin protection

See Hand protection below

Hands/feet protection

- ▶ Elbow length PVC gloves

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.

The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.

Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

- frequency and duration of contact,
- chemical resistance of glove material,
- glove thickness and
- dexterity

Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).

- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
 - When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
 - Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
 - Contaminated gloves should be replaced.
- As defined in ASTM F-739-96 in any application, gloves are rated as:
- Excellent when breakthrough time > 480 min
 - Good when breakthrough time > 20 min
 - Fair when breakthrough time < 20 min

Continued...

	· Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. ▸ Protective gloves eg. Leather gloves or gloves with Leather facing Experience indicates that the following polymers are suitable as glove materials for protection against undissolved, dry solids, where abrasive particles are not present. ▸ polychloroprene. ▸ nitrile rubber. ▸ butyl rubber. ▸ fluorocautchouc. ▸ polyvinyl chloride. Gloves should be examined for wear and/ or degradation constantly.
Body protection	See Other protection below
Other protection	▸ Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent] ▸ Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent] ▸ Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely. ▸ Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. ▸ Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▸ 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 <u>molten metal</u> should utilise primary protective clothing like polycarbonate face shields, fire resistant tapper's jackets, neck shades (snoods), leggings, spats and similar equipment to prevent burn injuries. In addition to primary protection, secondary or day-to-day work clothing that is fire resistant and sheds metal splash is recommended for use with molten metal. Synthetic materials should never be worn even as secondary clothing (undergarments).

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:

ACR Electronics Battery Pack 452-6499 & 455-0012

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.

Continued...

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

Ansell Glove Selection

Glove — In order of recommendation
MICROFLEX® MidKnight® XTRA 93-862
MICROFLEX® LifeStar EC™ 93-868
BioClean™ Emerald BENS
BioClean™ Extra BLAS
BioClean™ Fusion (Sterile) S-BFAP
BioClean™ N-Plus BNPS
BioClean™ Ultimate BUPS
MICROFLEX® 93-732
MICROFLEX® SafeGrip™ SG-375

The suggested gloves for use should be confirmed with the glove supplier.

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
 ▶ 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
· Respirators may be necessary when engineering and administrative controls do not adequately prevent exposures.
· The decision to use respiratory protection should be based on professional judgment that takes into account toxicity information, exposure measurement data, and frequency and likelihood of the worker's exposure - ensure users are not subject to high thermal loads which may result in heat stress or distress due to personal protective equipment (powered, positive flow, full face apparatus may be an option).
· Published occupational exposure limits, where they exist, will assist in determining the adequacy of the selected respiratory protection. These may be government mandated or vendor recommended.
· Certified respirators will be useful for protecting workers from inhalation of particulates when properly selected and fit tested as part of a complete respiratory protection program.
· Where protection from nuisance levels of dusts are desired, use type N95 (US) or type P1 (EN143) dust masks. Use respirators and components tested and approved under appropriate government standards such as NIOSH (US) or CEN (EU)
· Use approved positive flow mask if significant quantities of dust becomes airborne.
· Try to avoid creating dust conditions.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Dark grey battery with no odour; insoluble in water.		
Physical state	Manufactured	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable
pH (as supplied)	Not Applicable	Decomposition temperature (°C)	Not Available
Melting point / freezing	Not Available	Viscosity (cSt)	Not Applicable

point (°C)			
Initial boiling point and boiling range (°C)	Not Available	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 Available
Vapour pressure (kPa)	Not Applicable	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	Not Applicable	VOC g/L	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation. 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 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.
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. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing. 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,

Continued...

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.

Ingestion of perchlorates produces symptoms resembling chlorate intoxication. Ingestion of material causes gastro-intestinal upset. Perchlorates appears to be readily absorbed by the digestive system after oral exposure and enter the bloodstream within a few hours of ingestion; they are rapidly taken up into the thyroid gland by an active transport mechanism. Perchlorates do not appear to be modified in the body, either by degradation or covalent binding. Perchlorate is rapidly eliminated from the body in the urine with half-times of approximately 8-12 hours in humans.

Toxicity is due to strong oxidant effect and possible destruction of red blood cells. Perchlorates have only a limited ability to produce methaemoglobinaemia which reduces the oxygen-carrying capabilities of blood. Symptoms include shortness of breath, difficulty in breathing and a bluish discolouration of the skin (cyanosis). These effects may be delayed for several hours following exposure. Fatal aplastic anaemia has occurred in a small percentage of patients receiving therapeutic sodium perchlorate. Other blood disorders include agranulocytosis, thrombocytopenia and leucopenia. Signs of intolerance may precede changes in blood for several days. A nephrotic syndrome seldom occurs. Nausea, vomiting and hypersensitivity reactions such as maculopapular rashes, fever and lymphadenopathies (lymph node disease) may occur.

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.

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.

Overexposure to chlorates may result in methaemoglobinemia (lack of oxygen in blood) and cyanosis (bluish discolouration of skin and mucous membranes). Effects may be delayed. Coma, convulsions, liver and kidney damage and eventual kidney failure may also result. As sodium chlorate is used as a weed killer, several cases of chlorate poisoning in humans have been reported. Nausea and vomiting are almost always apparent after chlorate poisonings usually with epigastric pain (gastritis). Diarrhoea may also occur. The mean lethal dose of a chlorate salt taken by mouth has been established as 20-30 gms in human adults although others use a figure as low as 8 gm. Chlorates are nephrotoxic (cause kidney damage) and death in man and animals appears to be due to acute renal failure. Animal studies on chlorate suggest an increase in the utilisation or metabolism of thyroid hormones. Chlorates are readily absorbed by the gastrointestinal tract following ingestion. The chlorate ion is not metabolised to another compound, and elimination is exclusively by the kidneys. The acute toxic effects of potassium chlorate appear to be cumulative because of slow excretion of the chlorate ion.

Albuminuria and other evidence of impaired kidney function may persist for several days or weeks after. Severe intravascular haemolysis (blood cell damage), haemoglobinuria and methaemoglobinaemia are prominent features of chlorate poisonings although they may not be apparent until several hours have elapsed. Renal insufficiency may also result. Potassium chlorate has produced renal tubular necrosis in animals.

High levels can interfere with the ability of the blood to carry oxygen (methaemoglobinaemia), causing headache, dizziness. Ingestion may produce gastrointestinal distress, and may damage the kidneys. The primary mechanism of chlorate toxicity is rupture of the red blood cell membranes with intravascular haemolysis. The formation of methaemoglobin is secondary to lysis of red blood cells, and is caused by autooxidation of the free hemoglobin. The formation of methaemoglobin from free hemoglobin is irreversible, and may cause life-threatening effects. (Within the red blood cells, methaemoglobin is rapidly reduced by methaemoglobin reductase, but this activity is lost with cell lysis). Signs of chlorate intoxication may also include dark urine secondary to methaemoglobin formation.

Chlorates induce the formation of Heinz Bodies in the erythrocyte. Gross deformation of this cell may occur with premature splenic phagocytosis or haemolysis.

Central nervous system effects may also be secondary to hypoxia. Potassium chlorate is also a relatively powerful irreversible inhibitor of catalase.

Infants (and presumably the fetus) are much more sensitive than adults to intracellular methemoglobin inducers. This is due to a relative deficiency in methaemoglobin reductase in red blood cells of newborns, because the fetal form of haemoglobin is more sensitive to reducing agents, and because the fetus has a greater oxygen demand. However, it is not clear whether the newborn or the fetus may be more sensitive to the haemolytic effect of chlorates than adults. The extracellular autooxidative formation of methaemoglobin from lysed cells is irreversible and complete in both adults and fetuses, so there would be no difference in sensitivity in this step.

Persons with pre-existing blood conditions, especially anemia, or those with kidney diseases, might be more sensitive. Persons with genetic diseases such as hereditary methaemoglobinemia and glucose-6-phosphate dehydrogenase deficiency (which increases the haemolytic susceptibility of humans to oxidising agents), and other persons who may be unusually susceptible to oxidants may also be at greater risk than the general population.

Several studies have reported adverse effects (significant organ and body weight reductions, haematological effects, changes in pituitary and thyroid glands) in rats exposed via drinking water to sodium and/or potassium chlorate.

High chlorate levels were found in the testes of rats after oral administration of potassium chlorate, but it is not known if chlorate can affect male fertility.

	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	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. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact. 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.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	On the basis of epidemiological data, the material is regarded as carcinogenic to humans. There is sufficient data to establish a causal association between human exposure to the material and the development of cancer. Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. 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. Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. 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. Studies with some glycol ethers (principally the monoethylene glycols) and their esters indicate reproductive changes, testicular atrophy, infertility and kidney function changes. The metabolic acetic acid derivatives of glycol ethers (alkoxyacetic acids), not the ether itself, have been found to be the proximal reproductive toxin in animals. The potency of these metabolites decreases 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 Which produces haemolytic anaemia). 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. Glycol ethers based on propylene oxides, propylene glycol ethers, dipropylene glycol ethers and tripropylene glycol ethers are mainly available, commercially, as alpha-isomers (because of thermodynamic considerations); these are incapable of forming alkoxyacetic or alkoxypropionic acids as metabolites and therefore do not produce erythrocyte fragility unless contaminated by ethylene glycol ethers or to a significant degree by the beta-isomer . beta-Isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). 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 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 scleroderma, 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 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 (ClO_4^-) 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.

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 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

Overexposure to the breathable dust may cause coughing, wheezing, difficulty in breathing and impaired lung function. Chronic symptoms may include decreased vital lung capacity and chest infections. Repeated exposures in the workplace 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 (1/50000 inch) are present. Lung shadows are seen in the X-ray. Symptoms of pneumoconiosis may include a progressive dry cough, shortness of

ACR Electronics Battery Pack 452-6499 & 455-0012

breath on exertion, increased chest expansion, weakness and weight loss. As the disease progresses, the cough produces stringy phlegm, vital capacity decreases further, and shortness of breath becomes more severe. Other signs or symptoms include changed breath sounds, reduced oxygen uptake during exercise, emphysema and rarely, pneumothorax (air in the lung cavity).

Removing workers from the possibility of further exposure to dust generally stops the progress of lung abnormalities. When there is high potential for worker exposure, examinations at regular period with emphasis on lung function should be performed.

Inhaling dust over an extended number of years may cause pneumoconiosis, which is the accumulation of dusts in the lungs and the subsequent tissue reaction. This may or may not be reversible.

ACR Electronics Battery Pack 452-6499 & 455-0012	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]
lithium	TOXICITY	IRRITATION
	Not Available	Eye: adverse effect observed (irritating) ^[1] Skin: adverse effect observed (corrosive) ^[1]
carbon black	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[1] Oral (Rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1] 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] Oral (Rat) LD50: 2816 mg/kg ^[2]	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 ^[2]	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]
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	

CARBON BLACK	Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported
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

Continued...

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 sensitizers 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 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* cytogenetic 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 not 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 <i>in vitro</i> studies, positive results are cited for the A <i>S. typhimurium</i> reverse mutation assay. Monoglyme is known to be cytotoxic to <i>Salmonella typhimurium</i> (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 <i>in vitro</i> 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 <i>in vitro</i> 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. 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	Oral (human) LDLo: 50 mg/kg* [CCINFO]* Nil reported 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.
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/m³; 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
MANGANESE DIOXIDE & LITHIUM & CARBON BLACK & LITHIUM PERCHLORATE	No significant acute toxicological data identified in literature search.
LITHIUM & TETRAHYDROFURAN &	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of

LITHIUM PERCHLORATE	highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
CARBON BLACK & PROPYLENE CARBONATE	WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.

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

ACR Electronics Battery Pack 452-6499 & 455-0012	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
	NOEC(ECx)	1560h	Fish	0.55mg/l	2
lithium	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	10mg/l	2
	EC50	72h	Algae or other aquatic plants	25.6mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	1.65mg/l	2
	LC50	96h	Fish	18mg/l	2
carbon black	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	33.076-41.968mg/l	4
	EC50	72h	Algae or other aquatic plants	>0.2mg/l	2
	NOEC(ECx)	24h	Crustacea	3200mg/l	1
	LC50	96h	Fish	>100mg/l	2
1,2-dimethoxyethane	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	4000mg/l	2
	EC50	72h	Algae or other aquatic plants	9120mg/l	2
	NOEC(ECx)	504h	Crustacea	320mg/l	2
	LC50	96h	Fish	>500mg/l	2
tetrahydrofuran	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	24h	Fish	>=5mg/l	1
	LC50	96h	Fish	1970-2360mg/l	4
propylene carbonate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	>1000mg/l	1
	EC50	72h	Algae or other aquatic plants	>900mg/l	1

	NOEC(ECx)	72h	Algae or other aquatic plants	900mg/l	1
	LC50	96h	Fish	1000mg/l	1
lithium perchlorate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	>100mg/l	2
	EC50	72h	Algae or other aquatic plants	>120mg/l	2
	EC50(ECx)	48h	Crustacea	>100mg/l	2
Legend:	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data				

For Metal:

Atmospheric Fate - Metal-containing inorganic substances generally have negligible vapour pressure and are not expected to partition to air.

Environmental Fate: Environmental processes, such as oxidation, the presence of acids or bases and microbiological processes, may transform insoluble metals to more soluble ionic forms. Environmental processes may enhance bioavailability and may also be important in changing solubilities.

Aquatic/Terrestrial Fate: When released to dry soil, most metals will exhibit limited mobility and remain in the upper layer; some will leach locally into ground water and/ or surface water ecosystems when soaked by rain or melt ice. A metal ion is considered infinitely persistent because it cannot degrade further. Once released to surface waters and moist soils their fate depends on solubility and dissociation in water. A significant proportion of dissolved/ sorbed metals will end up in sediments through the settling of suspended particles. The remaining metal ions can then be taken up by aquatic organisms. Ionic species may bind to dissolved ligands or sorb to solid particles in water.

Ecotoxicity: Even though many metals show few toxic effects at physiological pH levels, transformation may introduce new or magnified effects.

For Silica:

Environmental Fate: Most documentation on the fate of silica in the environment concerns dissolved silica, in the aquatic environment, regardless of origin, (man-made or natural), or structure, (crystalline or amorphous).

Terrestrial Fate: Silicon makes up 25.7% of the Earth's crust, by weight, and is the second most abundant element, being exceeded only by oxygen. Silicon is not found free in nature, but occurs chiefly as the oxide and as silicates. Once released into the environment, no distinction can be made between the initial forms of silica.

Aquatic Fate: At normal environmental pH, dissolved silica exists exclusively as monosilicic acid. At pH 9.4, amorphous silica is highly soluble in water. Crystalline silica, in the form of quartz, has low solubility in water. Silicic acid plays an important role in the biological/geological/chemical cycle of silicon, especially in the ocean. Marine organisms such as diatoms, silicoflagellates and radiolarians use silicic acid in their skeletal structures and their skeletal remains leave silica in sea sediment

Ecotoxicity: Silicon is important to plant and animal life and is practically non-toxic to fish including zebrafish, and Daphnia magna water fleas.

For Ethelene Glycol Monoalkyl Ethers and their Acetates:

log BCF: 0.463 to 0.732;

LC50 : 94 to > 5000 mg/L. (aquatic species).

Members of this category include ethylene glycol propyl ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE).

Environmental Fate: Aquatic Fate - The ethers possess no functional groups that are readily subject to hydrolysis in the presence of waters. The acetates possess an ester group that hydrolyses in neutral ambient water under abiotic conditions. Will partition predominately to water and, to a lesser extent, to air and soil. Soil - Highly mobile in soil.

Ecotoxicity: Ethelene glycol monoalkyl ethers and their acetates are readily biodegradable. The physical chemistry and environmental fate properties indicate that category members will not persist or bioconcentrate in the environment. Glycol ether acetates do not hydrolyze rapidly into their corresponding glycol ethers in water under environmental conditions. Glycol ether acetates are not acutely toxic to fish, specifically, zebra fish, rainbow trout and water fleas. Population changes were noted in freshwater and green algae species.

For 1,2-dimethoxyethane (monoglyme):

Monoglyme, an ethylene glycol ether, is a volatile liquid with high water solubility. The value of the partition coefficient suggests that Monoglyme will partition into water and has little potential for bioaccumulation.

Environmental fate:

Distribution: Using Fugacity models monoglyme is expected to distribute primarily to water after release to the environment.

Photodegradation: Environmental degradation to carbon dioxide will likely occur by a combination of slow biodegradation and reaction with atmospheric hydroxyl radicals after volatilisation. Using the measured rate constant for reaction of Monoglyme with hydroxyl radical, vapours have an estimated photolytic half-life in air of approximately 8 hours, and predicted values for fugacity have been calculated with the Mackay Level III model.

Water Stability: Ethers are established to be relatively or completely inert in water as are glycols .

Experience with monoglyme in various aqueous preparations supports its resistance to hydrolysis under conditions that would occur in the environment. It has high hydrolytic stability in water at pH 4 to 9 with an estimated hydrolytic half-life at 25 C greater than one year.

Biodegradation: Monoglyme was recalcitrant to biodegradation, has questionable biodegradation, or is not assimilated as a substrate by bacteria. Monoglyme is poorly biodegradable in a waste water treatment facility and not considered readily biodegradable.

Ecotoxicity:

Fish, daphnia and green algae are estimated to be acutely affected by monoglyme only at concentrations far in excess of 1000 mg/l. Modeling and comparison with related compounds indicate that monoglyme is of low concern to aquatic environmental species. Given that the estimates and surrogate compounds show acute toxicity parameters several fold the OECD recommended maximum concentration for these studies, there is a high degree of confidence that monoglyme is of similar low hazard.

Fish LC50 (96 h): 8984 mg/l (estimated)

Daphnia EC50 (48 h): 7344 mg/l (estimated)

Algae EC50 (96 h): 4042 mg/l (estimated)

For manganese and its compounds:

Environmental fate:

It has been established that while lower organisms (e.g., plankton, aquatic plants, and some fish) can significantly bioconcentrate manganese, higher organisms (including humans) tend to maintain manganese homeostasis. This indicates that the potential for biomagnification of manganese from lower trophic levels to higher ones is low.

There were two mechanisms involved in explaining the retention of manganese and other metals in the environment by soil. First, through cation exchange reactions, manganese ions and the charged surface of soil particles form manganese oxides, hydroxides, and oxyhydroxides which in turn form absorption sites for other metals. Secondly, manganese can be adsorbed to other oxides, hydroxides, and oxyhydroxides through ligand exchange reactions. When the soil solution becomes saturated, these manganese oxides, hydroxides, and oxyhydroxides can precipitate into a new mineral phase and act as a new surface to which other substances can absorb. The tendency of soluble manganese compounds to adsorb to soils and sediments depends mainly on the cation exchange capacity and the organic composition of the soil. The soil adsorption constants (the ratio of the concentration in soil to the concentration in water) for Mn(II) span five orders of magnitude, ranging from 0.2 to 10,000 mL/g, increasing as a function of the organic content and the ion exchange capacity of the soil; thus, adsorption may be highly variable. In some cases, adsorption of manganese to soils may not be a readily reversible process. At low concentrations, manganese may be "fixed" by clays and will not be released into solution readily. At higher concentrations, manganese may be desorbed by ion exchange mechanisms with other ions in solution. For example, the discharge of waste water effluent into estuarine environments resulted in the mobilization of manganese from the bottom sediments. The metals in the effluent may have been preferentially adsorbed resulting in the release of manganese. The oxidation state of manganese in soil and sediments may be altered by microbial activity; oxidation may lead to the precipitation of manganese. Bacteria and microflora can increase the mobility of manganese. The transport and partitioning of manganese in water is controlled by the solubility of the specific chemical form present, which in turn is determined by pH, Eh (oxidation-reduction potential), and the characteristics of the available anions. The metal may exist in water in any of four oxidation states.

Manganese(II) predominates in most waters (pH 4-7) but may become oxidized at a pH >8 or 9. The principal anion associated with Mn(II) in water is usually carbonate (CO₃²⁻), and the concentration of manganese is limited by the relatively low solubility (65 mg/L) of MnCO₃. In relatively oxidized water, the solubility of Mn(II) may be controlled by manganese oxide equilibria, with manganese being converted to the Mn(II) or Mn(IV) oxidation states. In extremely reduced water, the fate of manganese tends to be controlled by formation of a poorly soluble sulfide. Manganese in water may undergo oxidation at high pH or Eh and is also subject to microbial activity. For example, Mn(II) in a lake was oxidized during the summer months, but this was inhibited by a microbial poison, indicating that the oxidation was mediated by bacteria. The microbial metabolism of manganese is presumed to be a function of pH, temperature, and other factors.

Manganese in water may be significantly bioconcentrated at lower trophic levels. A bioconcentration factor (BCF) relates the concentration of a chemical in plant and animal tissues to the concentration of the chemical in the water in which they live. The BCF of manganese was estimated as 2,500 - 6,300 for phytoplankton, 300 - 5,500 for marine algae, 80 - 830 for intertidal mussels, and 35 - 930 for coastal fish. Similarly, the BCF of manganese was estimated to be 10,000 - 20,000 for marine and freshwater plants, 10,000 - 40,000 for invertebrates, and 10 - 600 for fish. In general, these data indicate that lower organisms such as algae have larger BCFs than higher organisms. In order to protect consumers from the risk of manganese bioaccumulation in marine mollusks, the U.S. EPA has set a criterion for manganese at 0.1 mg/L for marine waters.

Elemental manganese and inorganic manganese compounds have negligible vapor pressures but may exist in air as suspended particulate matter derived from industrial emissions or the erosion of soils. Manganese-containing particles are mainly removed from the atmosphere by gravitational settling, with large particles tending to fall out faster than small particles. The half-life of airborne particles is usually on the order of days, depending on the size of the particle and atmospheric conditions. Some removal by washout mechanisms such as rain may also occur, although it is of minor significance in comparison to dry deposition.

Ecotoxicity:

Manganese ion is toxic to aqueous organisms

Fish LC50 (28 d): orfe 2490 mg/l, trout 2.91 mg/l

Daphnia magna LC50: 50 mg/l

Pseudomonas putida LC50: 10.6 mg/l

Photobacterium phosphoreum LC50: 14.7 mg/l

Turbellarian worms (EC0): Polycelis nigra 660 mg/l (interference threshold); microregma 31 mg/l

Perchlorate poses a human health concern because this contaminant has the same ionic size as iodide, and can compete with iodide for uptake into the thyroid gland, causing changes in thyroid hormone levels and possibly thyroid disorders. Although the long-term effects on humans are currently being debated, public concerns have generated considerable legislation designed to minimise potential damage. The environmental impacts of perchlorate have been less well studied, but the pollutant is clearly being transferred between abiotic and biotic ecosystem components.

Perchlorate consists of four-double covalent bonds between chlorine and oxygen (tetrahedral coordination), making this highly soluble oxyanion very stable and nonreactive in aqueous environments. Perchlorate salts also have a very low volatility. Although a strong oxidizing agent, the perchlorate anion is stable in the environment, due to the high activation energy associated with its abiotic reduction to chlorate (ClO₃⁻). Moreover, given its relatively low charge density, perchlorate does not form complexes with metals in the same manner as other anions, and it does not readily sorb to environmental media. This combination of perchlorate solubility, stability, and mobility creates the potential for both localised and area-wide potential effects of ecotoxicological interest. As such, it is a relatively non-reactive and kinetically a very stable contaminant, with very low biodegradation rates under many natural conditions. Biodegradation of perchlorate in the natural environment (e.g., sediments) will not occur unless significant levels of organic carbon are present, oxygen and nitrate are depleted, and perchlorate-degrading anaerobic bacteria are present.

Perchlorate (ClO₄⁻) is a highly toxic compound that is stable and persistent in the environment. Once perchlorate gets into the food chain or water supply, it does not break down easily. It has been detected in surface and ground water, soils and food (lettuce, bottled water, milk, meat, kelp, animal feed). It is stable, water soluble and persistent; it is not volatile and does not readily adhere to soil. It is also found in plants because its solubility in water allows it to be taken up by their roots. The populations considered to be most sensitive to perchlorate exposure are nursing infants, children, post-menopausal women, and people with hypothyroidism. Perchlorate interferes with thyroid functioning and is especially dangerous to fetuses, babies, and children. It causes thyroid iodine deficiency that in turn limits the gland's ability to produce a hormone essential to neurological development. "On average, children of iodine-deficient mothers have 5-to-13 fewer IQ points compared to children of mothers with iodine-sufficient diets," according to a report issued by the Environment California Research and Policy Center. "Reduced thyroid levels in the first few weeks of life for pre-term and low birth-weight babies are associated with increased risk of neurological disorders, including the need for special education by age nine."

Little is known about how perchlorate affects other species and the environment.

Until perchlorate effects on the human body are better understood a human reference dose of 0.0007 mg/kg per day has been suggested by the US EPA. The human reference dose is the daily exposure level below which EPA believes there would be no serious negative effects to a human over their lifetime. The EPA added that it considers drinking water safe if it contains no more than 24.5 parts per billion (ppb) of perchlorate.

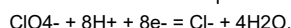
Perchlorate has contaminated ground and surface waters that are currently being used for irrigation in food production. Physical processes including mixing and dispersion control the distribution of perchlorate in groundwater.

Soil is not expected to naturally contain perchlorate. Perchlorate does not adsorb onto inorganic surfaces, including hydrous ferric oxide, smectite, and manganese oxide. These are characterised by net-negative surface charges at pH of 6.5 to 9.0. The inorganic perchlorate compounds introduced into soils are readily soluble and are not strongly adsorbed by other soil components. In soils, perchlorate is chemically stable and is expected to behave in a manner similar to dissolved minerals. While perchlorate is subject to leaching, its content in the soil is expected to approach steady state equilibrium in irrigated crop production system.

Perchlorate has been shown to be absorbed into crops from irrigation water or other sources. Reports show that raw materials and ingredients used to formulate fertilizers may contain significant amounts of perchlorate. Sodium nitrate imported from Chile is manufactured from Chilean caliche ores that are known to contain considerable amounts of perchlorate. Plants absorb nutrients and pollutants from solutions in the soil. Evidence shows that plants take up perchlorate from soils by processes similar to those of pertechnetate (TcO_4^-) and nitrate. Based on the basic principles of ion transport processes, plants exposed to perchlorate in the growing media are expected to accumulate perchlorate. The extent of the accumulation, however, may be influenced by external factors such as plant species and maturity, nutrient concentrations, and consumptive use of water. perchlorate uptake increased linearly in proportion to the perchlorate concentration in the growth media and the plant biomass. While there is evidence that a portion of the absorbed perchlorate may be reduced inside the plant through biochemical pathways similar to reductive microbial metabolic mechanisms, the majority of the absorbed perchlorate will translocate to and concentrate in the leaf tissue. Since oxygen is always present in the leaf tissue, plant mediated perchlorate reductions are not likely to be efficient or complete. The slow rate of phytodegradation of the perchlorate fraction taken up by plants during the growing season explains the detection of higher perchlorate concentrations in leaves collected later in the growing season (autumn) and in senesced leaves compared to younger, live leaves. This proves that the leaves of senesced plant potentially recycle perchlorate back into the soil on which plant litter collects. To minimise the potential recycling of perchlorate during phytoremediation, it is recommended that senesced leaves be collected and composted or phytoremediation be designed to enhance rapid rhizodegradation (rhizoremediation)

The fate of perchlorate (ClO_4^-) in streambed sediments is becoming a concern due to the increasing number of groundwater and surface water contamination sites. Results indicated that the spatial and temporal ClO_4^- penetration into sediments could be affected by numerous factors, such as temperature, microbial degradation, ClO_4^- surface water concentration, and sediment physico-geological properties. In general, maximum ClO_4^- penetration into sediments at the studied sites was 30 cm below the sediment-water surface. The vertical sequential depletion of electron acceptors in sediments suggested that microbial reduction was responsible for ClO_4^- depletion in stream sediments. Biodegradation of ClO_4^- occurred over a seasonally variable active depth zone of 1-10 cm. Results implied that there was a rapid natural attenuation potential of perchlorate in near-surface sediments. Perchlorate may be rapidly attenuated in saturated near-surface sediments.

The overall reduction of perchlorate to chloride is given by the following half reaction:



This reaction has relevance to natural systems such as wetlands and other microbially-active environments with plentiful and diverse electron donors. Perchlorate is reduced to intermediate compounds (chlorate [Cl(V)] and chlorite [Cl(III)]) and eventually to chloride in anaerobic environments by serving as a terminal electron acceptor during oxidation of acetate and other forms of reactive organic carbon. Perchlorate reduction is both thermodynamically and microbially enhanced under denitrifying conditions. Perchlorate has been shown to serve as a terminal electron acceptor for energy and growth. Reductase enzymes catalyse the reduction of perchlorate to chlorate and then to chlorite (ClO_2^-). A fairly unique enzyme, a dismutase, then facilitates the disproportionation of ClO_2^- to Cl^- and O_2 . There are numerous strains of micro-organisms capable of reducing both chlorate and perchlorate under anoxic conditions. The chlorite dismutase enzyme is present in all dissimilatory perchlorate- and chlorate reducing bacteria. Under anaerobic conditions, perchlorate and chlorate- are first converted to chlorite (ClO_2^-) by the enzyme chlorate reductase, which is present in perchlorate- and chlorate-reducing bacteria. In the second step, chlorite is disproportionated to chloride (Cl^-) and molecular oxygen (O_2) by the non-respiratory enzyme chlorite dismutase, which catalyses the reaction. The presence of chlorite dismutase is a prerequisite for the growth of perchlorate- and chlorate reducing bacteria as chlorite is toxic due to its high reactivity.

Perchlorate is not likely to come out of solution given its low vapor pressure. Droplet size during showering would likely preclude significant inhalation of perchlorate-contaminated water as an aerosol.

In perchlorate-contaminated lakes and streams, perchlorate is detected infrequently in fish heads, fillets, and whole bodies, but may be detected more often depending on species and seasonal trends, and always at concentrations higher in the fish than in the water. Perchlorates may perturb thyroid-hormone concentration in fish; this may affect growth and neurological development. Data from fish indicates that perchlorate can also disrupt sexual development. Certain have been so dramatic that female fish have been mistaken for males. Several females displayed male-courtship behavior and produced sperm. This is suggestive of the fact that perchlorate may act as an androgen (male sex hormone). The concentration of perchlorate used in these studies were at least a 1000 times the US EPA limit (24.5 parts per billion in natural bodies of water).

For Glycol Ethers:

Environmental Fate: Several glycol ethers have been shown to biodegrade however; biodegradation slows as molecular weight increases. No glycol ethers that have been tested demonstrate marked resistance to biodegradative processes. No glycol ethers that have been tested demonstrate marked resistance to biodegradative processes.

Atmospheric Fate: Upon release to the atmosphere by evaporation, high boiling glycol ethers are estimated to undergo photo-degradation (atmospheric half lives = 2.4-2.5 hr). **Aquatic Fate:** In water, glycol ethers undergo biodegradation (typically 47-92% after 8-21 days) and have a low potential for bioaccumulation (log Kow ranges from -1.73 to +0.51).

Ecotoxicity: Tri- and tetra ethylene glycol ethers are "practically non-toxic" to aquatic species. No major differences are observed in the order of toxicity going from the methyl- to the butyl ethers. Glycols exert a high oxygen demand for decomposition and once released to the environment death of aquatic organisms occurs if dissolved oxygen is depleted.

For lithium (anion):

Environmental fate:

Experiments with experimental animals have shown that lithium can have reprotoxic effects, and increasing consumption might therefore result in adverse effects on health and environment. Lithium has significant bioavailability only when administered as a partially soluble salt such as lithium carbonate. Lithium is not a dietary mineral for plants but it does stimulate plant growth.

Ecotoxicity:

Fish LC50 (28, 35 days) rainbow trout 9.28, 1.4 mg/l (salt)

Fish LC50 (96 h): fathead minnow 42 mg/l; NOEC 13 mg/l (salt)

Daphnia magna EC50 (48 h): 24 mg/l; NOEC 11 mg/l

Lithium is not expected to bioaccumulate in mammals and its human and environmental toxicity are low. Lithium does accumulate in several species of fish, molluscs and crustaceans where it stored in the digestive tract and exoskeleton

Methanogenesis of granular anaerobic sludge (initial COD 5750 mg/l O₂, pH 7.2) was stimulated at lithium ion concentration 10-20 mg/l, slightly inhibited at lithium ion concentration 350 mg/l and seriously inhibited at lithium ion concentration > 500 mg/l.

Microinjection of lithium chloride into prospective ventral blastomeres of a 32-cell *Xenopus laevis* embryo gives rise to duplication of dorsoanterior structures such as the notochord, neural tube and eyes.

for propylene carbonate:

Environmental fate:

Data on the transport of propylene carbonate between environmental compartments has been estimated using EPIWIN; propylene carbonate will partition mostly to water and soil; 1% to air; 46% to water; 53% to soil.

Does not absorb UV and therefore is unlikely to be photodegradable.

Biodegradation: OECD Test Guideline 301B (Modified Sturm Test)

Propylene carbonate was readily biodegradable by activated domestic sludge under aerobic conditions. More than 85% degraded after 29 days

Ecotoxicity

Invertebrate EC₅₀: >1000 mg/l

Aquatic plant EC₅₀: >=929 mg/l

Acute toxicity studies on fish and daphnia on a propylene carbonate analog, butylene carbonate, were conducted according to OECD Guidelines, following GLP guidelines. Butylene carbonate (CAS # 4437-85-8) is considered an acceptable surrogate for propylene carbonate because of similar physical-chemical properties.

In rainbow trout the LC₅₀ for butylene carbonate was 480 mg/l and in *Daphnia* the EC₅₀ was >1000 mg/l. Based on similarity to butylene carbonate, propylene carbonate is not expected to be significantly more toxic to aquatic organisms. The low toxicity of butylene carbonate provides adequate information to conclude that propylene carbonate is not likely to be more than slightly toxic to aquatic organisms.

Toxicity to Aquatic plants (e.g., Algae): OECD Test Guideline 201 Propylene carbonate did not affect cell density, area under the growth curve, or growth rate of unicellular green alga, *Selenastrum capricornutum*, after 72 and 96 hours of exposure at levels up to 929 mg/l, measured concentration.

Biodegradation: OECD Test Guideline 301B (Modified Sturm Test)

Propylene carbonate was readily biodegradable by activated domestic sludge under aerobic conditions. More than 85% degraded after 29 days.

For tetrahydrofuran (THF):

Ecotoxicity: Acute short term toxic environmental effects of THF may include the death of animals, birds, fish and death or low growth rate in plants. Acute effects are seen 2 to 4 days after animals or plants are exposed to tetrahydrofuran.

Chronic toxic effects include shortened life span, reproductive problems, lowered fertility, and changes in appearance or behaviour in exposed animals. These effects have been seen long after the first exposure(s).

Environmental fate: Tetrahydrofuran is slightly persistent in water with a half-life of between 2 to 20 days. Tetrahydrofuran is not expected to bioaccumulate in aquatic organisms. The concentration of tetrahydrofuran in edible fish tissue is expected to be negligible as compared to the levels found in the water from which the fish was taken.

If tetrahydrofuran should contact the water table, aquifer or navigable waterway, time is of the essence. It is highly soluble in water and total remediation may not be possible. A comprehensive emergency response or disaster preparedness/ recovery plan should be in place prior to use.

Volatilisation from water surfaces is expected to be an important fate process based upon this compound's Henry's Law constant. Estimated volatilisation half-lives for a model river and model lake are 13.1 hrs and 6.6 days, respectively. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to occur due to the lack of hydrolysable functional groups

If released to air, a vapor pressure of 162 mm Hg at 25 deg C indicates tetrahydrofuran will exist solely as a vapor in the ambient atmosphere. Vapor-phase tetrahydrofuran will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl and nitrate radicals; the half-life for these reactions in air are about 1 and 3 days, respectively. Tetrahydrofuran is moderately reactive in photochemical smog conditions where nitrogen oxides are present; reactions occur in time scales of hours. Acrolein and formaldehyde have been reported as reaction products.

If released to soil, tetrahydrofuran is expected to have very high mobility based upon K_{oc} values of 23 and 18. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry's Law constant of 7.1X10⁻⁵ atm-m³/mole. Tetrahydrofuran may volatilise from dry soil surfaces based upon its vapor pressure. Tetrahydrofuran added to surface soil had an abiotic half-life of 5.7 days. If released into water, tetrahydrofuran is not expected to adsorb to suspended solids and sediment based upon the K_{oc}. Tetrahydrofuran is expected to biodegrade under aerobic conditions but may be resistant to biodegradation in anaerobic environments. In the modified MITI screening test, tetrahydrofuran at 30 mg/l was completely biodegraded in 14 days using an activated sludge inoculum. Tetrahydrofuran at 50 mg C/l was resistant to anaerobic biodegradation with a lag period of greater than 60 days using a primary digesting sludge as an inoculum.

DO NOT discharge into sewer or waterways.

Persistence and degradability

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

Bioaccumulative potential

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

Mobility in soil

Ingredient	Mobility
1,2-dimethoxyethane	HIGH (Log KOC = 1)
tetrahydrofuran	LOW (Log KOC = 4.881)
propylene carbonate	LOW (Log KOC = 14.85)
lithium perchlorate	LOW (Log KOC = 48.64)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	- Containers may still present a chemical hazard/ danger when empty. - Return to supplier for reuse/ recycling if possible. Otherwise: - If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. - Where possible retain label warnings and SDS and observe all notices pertaining to the product. 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.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	NO

Shipping container, transport vehicle placarding, and labeling may vary from the below information. This depends on the quantity shipped, the applicability of excepted quantity requirements, limited quantity requirements, and/or special provisions according to US DOT, IATA and IMDG regulations. In case of reshipment, it is the responsibility of the shipper to determine the appropriate labels and markings in accordance with applicable transport regulations.

Land transport (DOT)

14.1. UN number or ID number	3090		
14.2. UN proper shipping name	Lithium metal batteries including lithium alloy batteries		
14.3. Transport hazard class(es)	Class	9	
	Subsidiary Hazard	Not Applicable	
14.4. Packing group	Not Applicable		
14.5. Environmental hazard	Not Applicable		
14.6. Special precautions for user	Hazard Label	9	
	Special provisions	388, 422, A54	

Air transport (ICAO-IATA / DGR)

14.1. UN number	3090	
14.2. UN proper shipping name	Lithium metal batteries (including lithium alloy batteries)	
14.3. Transport hazard class(es)	ICAO/IATA Class	9
	ICAO / IATA Subsidiary Hazard	Not Applicable
	ERG Code	12FZ
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Special provisions	A88 A99 A154 A164 A183 A201 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)

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

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

Not Applicable

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

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

14.7.3. Transport in bulk in accordance with the IGC Code

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

Product name	Ship Type
tetrahydrofuran	Not Available
propylene carbonate	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

- International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
- US - Alaska Air Quality Control - Concentrations Triggering an Air Quality Episode for Air Pollutants Other Than PM-2.5
- US - California Hazardous Air Pollutants Identified as Toxic Air Contaminants
- US - Massachusetts - Right To Know Listed Chemicals
- US Clean Air Act - Hazardous Air Pollutants
- US DOE Temporary Emergency Exposure Limits (TEELs)
- US EPCRA Section 313 Chemical List
- US NIOSH Recommended Exposure Limits (RELs)
- US OSHA Permissible Exposure Limits (PELs) Table Z-1
- US OSHA Permissible Exposure Limits (PELs) Table Z-3
- US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

lithium is found on the following regulatory lists

- FEI Equine Prohibited Substances List - Banned Substances
- FEI Equine Prohibited Substances List (EPSSL)
- International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
- US - Alaska Air Quality Control - Concentrations Triggering an Air Quality Episode for Air Pollutants Other Than PM-2.5
- US - Massachusetts - Right To Know Listed Chemicals
- US DOE Temporary Emergency Exposure Limits (TEELs)
- US NIOSH Recommended Exposure Limits (RELs)
- US OSHA Permissible Exposure Limits (PELs) Table Z-1
- US OSHA Permissible Exposure Limits (PELs) Table Z-3
- US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

carbon black is found on the following regulatory lists

- 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)
- US - Alaska Air Quality Control - Concentrations Triggering an Air Quality Episode for Air Pollutants Other Than PM-2.5
- US - California Proposition 65 - Carcinogens
- US - California Safe Drinking Water and Toxic Enforcement Act of 1986 - Proposition 65 List
- US - Massachusetts - Right To Know Listed Chemicals
- US DOE Temporary Emergency Exposure Limits (TEELs)
- US NIOSH Carcinogen List
- US NIOSH Recommended Exposure Limits (RELs)
- US OSHA Permissible Exposure Limits (PELs) Table Z-1
- US OSHA Permissible Exposure Limits (PELs) Table Z-3
- US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

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

- Chemical Footprint Project - Chemicals of High Concern List
- US - California Hazardous Air Pollutants Identified as Toxic Air Contaminants
- US - Massachusetts - Right To Know Listed Chemicals
- US DOE Temporary Emergency Exposure Limits (TEELs)
- US EPCRA Section 313 Chemical List
- US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
- US TSCA Section 12(b) - List of Chemical Substances Subject to Export Notification Requirements
- US TSCA Section 5(a)(2) - Significant New Use Rules (SNURs)

tetrahydrofuran is found on the following regulatory lists

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
- US - California Proposition 65 - Carcinogens
- US - California Safe Drinking Water and Toxic Enforcement Act of 1986 - Proposition 65 List
- US - Massachusetts - Right To Know Listed Chemicals
- US DOE Temporary Emergency Exposure Limits (TEELs)
- US EPA Integrated Risk Information System (IRIS)
- US NIOSH Recommended Exposure Limits (RELs)
- US OSHA Permissible Exposure Limits (PELs) Table Z-1
- US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
- US TSCA Section 4/12 (b) - Sunset Dates/Status

propylene carbonate is found on the following regulatory lists

- US DOE Temporary Emergency Exposure Limits (TEELs)
- US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

lithium perchlorate is found on the following regulatory lists

- US - California - Biomonitoring - Priority Chemicals
- US DOE Temporary Emergency Exposure Limits (TEELs)
- US EPA Integrated Risk Information System (IRIS)
- US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

Additional Regulatory Information

Not Applicable

Federal Regulations

Superfund Amendments and Reauthorization Act of 1986 (SARA)

Section 311/312 hazard categories

Flammable (Gases, Aerosols, Liquids, or Solids)	No
Gas under pressure	No
Explosive	No
Self-heating	No
Pyrophoric (Liquid or Solid)	No
Pyrophoric Gas	No
Corrosive to metal	No
Oxidizer (Liquid, Solid or Gas)	Yes
Organic Peroxide	No
Self-reactive	No
In contact with water emits flammable gas	Yes
Combustible Dust	No
Carcinogenicity	Yes
Acute toxicity (any route of exposure)	Yes
Reproductive toxicity	Yes
Skin Corrosion or Irritation	Yes
Respiratory or Skin Sensitization	No
Serious eye damage or eye irritation	Yes
Specific target organ toxicity (single or repeated exposure)	No
Aspiration Hazard	No
Germ cell mutagenicity	No
Simple Asphyxiant	No
Hazards Not Otherwise Classified	No

US. EPA CERCLA Hazardous Substances and Reportable Quantities (40 CFR 302.4)

Name	Reportable Quantity in Pounds (lb)	Reportable Quantity in kg
tetrahydrofuran	1000	454

US. EPCRA Section 313 Toxic Release Inventory (TRI) (40 CFR 372)

This product contains the following EPCRA section 313 chemicals subject to the reporting requirements of section 313 of the Emergency Planning and Community Right-To-Know-Act of 1986 (40 CFR 372):

CAS No	%[weight]	Name
1313-13-9	25-50	manganese dioxide
110-71-4	<10	1,2-dimethoxyethane

This information must be included in all SDSs that are copied and distributed for this material.

Additional Federal Regulatory Information

Not Applicable

State Regulations

US. California Proposition 65

 **WARNING:** This product can expose you to chemicals including **carbon black, tetrahydrofuran**, which are known to the State of California to cause cancer. For more information, go to www.P65Warnings.ca.gov

Additional State Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (manganese dioxide; lithium; carbon black; 1,2-dimethoxyethane; tetrahydrofuran; propylene carbonate; 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	03/10/2023
Initial Date	10/21/2020

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	11/02/2020	Toxicological information - Acute Health (eye), Toxicological information - Acute Health (inhaled), Toxicological information - Acute Health (skin), Toxicological information - Acute Health (swallowed), First Aid measures - Advice to Doctor, Toxicological information - Chronic Health, Hazards identification - Classification, Disposal considerations - Disposal, Exposure controls / personal protection - Engineering Control, Ecological Information -

Version	Date of Update	Sections Updated
		Environmental, Firefighting measures - Fire Fighter (fire/explosion hazard), Firefighting measures - Fire Fighter (fire fighting), First Aid measures - First Aid (inhaled), First Aid measures - First Aid (skin), First Aid measures - First Aid (swallowed), Handling and storage - Handling Procedure, Composition / information on ingredients - Ingredients, Stability and reactivity - Instability Condition, Exposure controls / personal protection - Personal Protection (other), Exposure controls / personal protection - Personal Protection (Respirator), Exposure controls / personal protection - Personal Protection (eye), Exposure controls / personal protection - Personal Protection (hands/feet), Accidental release measures - Spills (major), Accidental release measures - Spills (minor), Handling and storage - Storage (storage incompatibility), Handling and storage - Storage (storage requirement), Handling and storage - Storage (suitable container), Transport information - Transport, Transport Information
3.1	03/10/2023	Classification change due to full database hazard calculation/update.

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by 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
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration

- 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
- 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

This document is copyright.

Apart from any fair dealing for the purposes of private study, research, review or criticism, as permitted under the Copyright Act, no part may be reproduced by any process without written permission from CHEMWATCH.

TEL (+61 3) 9572 4700.

ACR Electronics Battery Pack 452-6499 & 455-0012