

# 24V battery pack

## Leitner Pty Ltd

Part Number: D10 D12

Version No: 1.2

Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: 30/09/2022

Print Date: 30/09/2022

L.GHS.AUS.EN

### SECTION 1 Identification of the substance / mixture and of the company / undertaking

#### Product Identifier

|                               |   |
|-------------------------------|---|
| Product name                  | 24V battery pack  |
| Synonyms                      | Not Available   |
| Proper shipping name          | LITHIUM ION BATTERIES (including lithium ion polymer batteries) |
| Other means of identification | D10 D12   |

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

|                          |                                 |
|--------------------------|---------------------------------|
| Relevant identified uses | Battery for electric wheelchair |
|--------------------------|---------------------------------|

#### Details of the manufacturer or supplier of the safety data sheet

|                         |  |
|-------------------------|--|
| Registered company name | Leitner Pty Ltd  |
| Address                 | U11, 69 Acacia Rd Ferntree Gully VIC 3156 Australia        |
| Telephone               | 1300 856 725   |
| Fax                     | Not Available  |
| Website                 | <a href="http://www.leitner.com.au">www.leitner.com.au</a> |
| Email                   | ops@leitner.com.au   |

#### Emergency telephone number

|                                   |                 |
|-----------------------------------|-----------------|
| Association / Organisation        | Not Available   |
| Emergency telephone numbers       | 1300 856 725    |
| Other emergency telephone numbers | +61 455 210 194 |

### SECTION 2 Hazards identification

#### Classification of the substance or mixture

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

|                    |  |
|--------------------|--|
| Poisons Schedule   | Not Applicable   |
| Classification [1] | Germ Cell Mutagenicity Category 1A, Acute Toxicity (Oral) Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 2, Serious Eye Damage/Eye Irritation Category 1, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Carcinogenicity Category 2 |
| Legend:            | 1. Classification by vendor; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI   |

#### Label elements

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

**24V battery pack**

Signal word **Danger**

**Hazard statement(s)**

|             |  |
|-------------|--|
| <b>H340</b> | May cause genetic defects.   |
| <b>H300</b> | Fatal if swallowed.  |
| <b>H411</b> | Toxic to aquatic life with long lasting effects.                   |
| <b>H373</b> | May cause damage to organs through prolonged or repeated exposure. |
| <b>H318</b> | Causes serious eye damage.   |
| <b>H315</b> | Causes skin irritation.  |
| <b>H317</b> | May cause an allergic skin reaction.                               |
| <b>H351</b> | Suspected of causing cancer.                                       |

**Supplementary statement(s)**

Not Applicable

**Precautionary statement(s) Prevention**

|             |  |
|-------------|--|
| <b>P201</b> | Obtain special instructions before use.  |
| <b>P260</b> | Do not breathe dust/fume.  |
| <b>P264</b> | Wash all exposed external body areas thoroughly after handling.                  |
| <b>P270</b> | Do not eat, drink or smoke when using this product.                              |
| <b>P280</b> | Wear protective gloves, protective clothing, eye protection and face protection. |
| <b>P273</b> | Avoid release to the environment.  |
| <b>P272</b> | Contaminated work clothing should not be allowed out of the workplace.           |

**Precautionary statement(s) Response**

|                       |  |
|-----------------------|--|
| <b>P301+P310</b>      | IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.   |
| <b>P305+P351+P338</b> | IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing. |
| <b>P308+P313</b>      | IF exposed or concerned: Get medical advice/ attention.  |
| <b>P330</b>           | Rinse mouth.   |
| <b>P302+P352</b>      | IF ON SKIN: Wash with plenty of water.   |
| <b>P333+P313</b>      | If skin irritation or rash occurs: Get medical advice/attention.   |
| <b>P362+P364</b>      | Take off contaminated clothing and wash it before reuse.   |
| <b>P391</b>           | Collect spillage.  |

**Precautionary statement(s) Storage**

|             |                  |
|-------------|------------------|
| <b>P405</b> | Store locked up. |
|-------------|------------------|

**Precautionary statement(s) Disposal**

|             |  |
|-------------|--|
| <b>P501</b> | 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                                      |
|-------------|-----------|---|
| 182442-95-1 | 58-62     | <u>cobalt lithium manganese nickelate</u> |
| 7440-44-0   | 12-15     | <u>carbon, non-activated</u>              |
| 7440-50-8   | 7-10      | <u>copper</u>                             |
| 7782-42-5   | 8         | <u>graphite</u>                           |
| 7429-90-5   | 5         | <u>aluminium</u>                          |
| 96-49-1     | 5         | <u>ethylene carbonate</u>                 |

## 24V battery pack

| CAS No    | %[weight] | Name  |
|-----------|-----------|---|
| 7440-02-0 | 2-5       | <u>nickel catalyst, finely divided, &gt;= 40% water</u> |
| 7439-93-2 | 2-3       | <u>lithium</u>  |

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

## SECTION 4 First aid measures

## Description of first aid measures

|                     |  |
|---------------------|--|
| <b>Eye Contact</b>  | <p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> <li>▶ Immediately hold eyelids apart and flush the eye continuously with running water.</li> <li>▶ 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.</li> <li>▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.</li> <li>▶ Transport to hospital or doctor without delay.</li> <li>▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> <li>▶ <b>DO NOT attempt to remove particles attached to or embedded in eye .</b></li> <li>▶ Lay victim down, on stretcher if available and pad <b>BOTH</b> eyes, make sure dressing does not press on the injured eye by placing thick pads under dressing, above and below the eye.</li> <li>▶ Seek urgent medical assistance, or transport to hospital.</li> </ul>  |
| <b>Skin Contact</b> | <p>If skin contact occurs:</p> <ul style="list-style-type: none"> <li>▶ Immediately remove all contaminated clothing, including footwear.</li> <li>▶ Flush skin and hair with running water (and soap if available).</li> <li>▶ Seek medical attention in event of irritation.</li> </ul> <p>For thermal burns:</p> <ul style="list-style-type: none"> <li>▶ Decontaminate area around burn.</li> <li>▶ Consider the use of cold packs and topical antibiotics.</li> </ul> <p>For first-degree burns (affecting top layer of skin)</p> <ul style="list-style-type: none"> <li>▶ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides.</li> <li>▶ Use compresses if running water is not available.</li> <li>▶ Cover with sterile non-adhesive bandage or clean cloth.</li> <li>▶ Do NOT apply butter or ointments; this may cause infection.</li> <li>▶ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur.</li> </ul> <p>For second-degree burns (affecting top two layers of skin)</p> <ul style="list-style-type: none"> <li>▶ Cool the burn by immerse in cold running water for 10-15 minutes.</li> <li>▶ Use compresses if running water is not available.</li> <li>▶ Do NOT apply ice as this may lower body temperature and cause further damage.</li> <li>▶ Do NOT break blisters or apply butter or ointments; this may cause infection.</li> <li>▶ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape.</li> </ul> <p>To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort):</p> <ul style="list-style-type: none"> <li>▶ Lay the person flat.</li> <li>▶ Elevate feet about 12 inches.</li> <li>▶ Elevate burn area above heart level, if possible.</li> <li>▶ Cover the person with coat or blanket.</li> <li>▶ Seek medical assistance.</li> </ul> <p>For third-degree burns</p> <p>Seek immediate medical or emergency assistance.</p> <p>In the mean time:</p> <ul style="list-style-type: none"> <li>▶ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound.</li> <li>▶ Separate burned toes and fingers with dry, sterile dressings.</li> <li>▶ Do not soak burn in water or apply ointments or butter; this may cause infection.</li> <li>▶ To prevent shock see above.</li> <li>▶ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway.</li> <li>▶ Have a person with a facial burn sit up.</li> <li>▶ Check pulse and breathing to monitor for shock until emergency help arrives.</li> </ul> |
| <b>Inhalation</b>   | <ul style="list-style-type: none"> <li>▶ If fumes or combustion products are inhaled remove from contaminated area.</li> <li>▶ Lay patient down. Keep warm and rested.</li> <li>▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.</li> <li>▶ 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.</li> <li>▶ Transport to hospital, or doctor, without delay.</li> </ul>   |
| <b>Ingestion</b>    | <ul style="list-style-type: none"> <li>▶ <b>IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY.</b></li> <li>▶ For advice, contact a Poisons Information Centre or a doctor.</li> <li>▶ Urgent hospital treatment is likely to be needed.</li> </ul>   |

## 24V battery pack

- ▶ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition.
- ▶ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist.
- ▶ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS.

**Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:**

- ▶ **INDUCE** vomiting with fingers down the back of the throat, **ONLY IF CONSCIOUS**. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.

**NOTE:** Wear a protective glove when inducing vomiting by mechanical means.

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

Treat symptomatically.

for copper intoxication:

- ▶ Unless extensive vomiting has occurred empty the stomach by lavage with water, milk, sodium bicarbonate solution or a 0.1% solution of potassium ferrocyanide (the resulting copper ferrocyanide is insoluble).
- ▶ Administer egg white and other demulcents.
- ▶ Maintain electrolyte and fluid balances.
- ▶ Morphine or meperidine (Demerol) may be necessary for control of pain.
- ▶ If symptoms persist or intensify (especially circulatory collapse or cerebral disturbances, try BAL intramuscularly or penicillamine in accordance with the supplier's recommendations.
- ▶ Treat shock vigorously with blood transfusions and perhaps vasopressor amines.
- ▶ If intravascular haemolysis becomes evident protect the kidneys by maintaining a diuresis with mannitol and perhaps by alkalinising the urine with sodium bicarbonate.
- ▶ It is unlikely that methylene blue would be effective against the occasional methaemoglobinemia and it might exacerbate the subsequent haemolytic episode.
- ▶ Institute measures for impending renal and hepatic failure.

[GOSSELIN, SMITH & HODGE: Commercial Toxicology of Commercial Products]

- ▶ A role for activated charcoals for emesis is, as yet, unproven.
- ▶ In severe poisoning CaNa<sub>2</sub>EDTA has been proposed.

[ELLENHORN & BARCELOUX: Medical Toxicology]

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 cathartics.
- ▶ 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]

- ▶ In cases of nickel poisoning, dimercaptol delivered by deep intramuscular injection may be a suitable antidote. (Patients should not exhibit renal or hepatic dysfunction.) The use of diethyldithiocarbamate is the subject of ongoing research.
- ▶ Irritant contact dermatoses or eczemas may respond to applications of weak antiseptic packs, antibiotic ointments (tetracycline or erythromycin) or inert pastes and ointments. Systemic antibiotics are advisable in the presence of lymphangitis or lymphadenitis.

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

## 24V battery pack

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.*]

- Chronic exposures to cobalt and its compounds results in the so-called "hard metal pneumoconiosis" amongst industrial workers. The lesions consist of nodular conglomerate shadows in the lungs, together with peribronchial infiltration. The disease may be reversible. The acute form of the disease resembles a hypersensitivity reaction with malaise, cough and wheezing; the chronic form progresses to cor pulmonale.
- Chronic therapeutic administration may cause goiter and reduced thyroid activity.
- An allergic dermatitis, usually confined to elbow flexures, the ankles and sides of the neck, has been described.
- Cobalt cardiomyopathy may be diagnosed early by changes in the final part of the ventricular ECG (repolarisation). In the presence of such disturbances, the changes in carbohydrate metabolism (revealed by the glucose test) are of important diagnostic value.
- Treatment generally consists of a combination of Retabolil (1 injection per week over 4 weeks) and beta-blockers (average dose 60-80 mg Obsidan/24 hr). Potassium salts and diuretics have also proved useful.

## BIOLOGICAL EXPOSURE INDEX (BEI)

| Determinant     | Sampling time                   | Index   | Comments |
|-----------------|---------------------------------|---------|----------|
| Cobalt in urine | End of shift at end of workweek | 15 ug/L | B        |
| Cobalt in blood | End of shift at end of workweek | 1 ug/L  | B, SQ    |

B: Background levels occur in specimens collected from subjects NOT exposed

SQ: Semi-quantitative determinant - Interpretation may be ambiguous; should be used as a screening test or confirmatory test.

## SECTION 5 Firefighting measures

## Extinguishing media

Metal dust fires need to be smothered with sand, inert dry powders.

**DO NOT USE WATER, CO2 or FOAM.**

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

## Special hazards arising from the substrate or mixture

|                             |   |
|-----------------------------|---|
| <b>Fire Incompatibility</b> | <ul style="list-style-type: none"> <li>▸ Reacts with acids producing flammable / explosive hydrogen (H2) gas</li> <li>▸ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result</li> </ul> |
|-----------------------------|---|

## Advice for firefighters

|                              |   |
|------------------------------|---|
| <b>Fire Fighting</b>         | <ul style="list-style-type: none"> <li>▸ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▸ Wear breathing apparatus plus protective gloves in the event of a fire.</li> <li>▸ Prevent, by any means available, spillage from entering drains or water courses.</li> <li>▸ Use fire fighting procedures suitable for surrounding area.</li> <li>▸ <b>DO NOT</b> approach containers suspected to be hot.</li> <li>▸ Cool fire exposed containers with water spray from a protected location.</li> <li>▸ If safe to do so, remove containers from path of fire.</li> <li>▸ Equipment should be thoroughly decontaminated after use.</li> </ul>   |
| <b>Fire/Explosion Hazard</b> | <ul style="list-style-type: none"> <li>▸ <b>DO NOT</b> disturb burning dust. Explosion may result if dust is stirred into a cloud, by providing oxygen to a large surface of hot metal.</li> <li>▸ <b>DO NOT</b> use water or foam as generation of explosive hydrogen may result.</li> </ul> <p>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.</p> <p>Metal powders, while generally regarded as non-combustible:</p> <ul style="list-style-type: none"> <li>▸ May burn when metal is finely divided and energy input is high.</li> <li>▸ May react explosively with water.</li> <li>▸ May be ignited by friction, heat, sparks or flame.</li> <li>▸ May <b>REIGNITE</b> after fire is extinguished.</li> <li>▸ Will burn with intense heat.</li> </ul> <p>Note:</p> <ul style="list-style-type: none"> <li>▸ Metal dust fires are slow moving but intense and difficult to extinguish.</li> <li>▸ Containers may explode on heating.</li> <li>▸ Dusts or fumes may form explosive mixtures with air.</li> <li>▸ Gases generated in fire may be poisonous, corrosive or irritating.</li> <li>▸ 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.</li> </ul> |

## 24V battery pack

- ▶ 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 (CO<sub>2</sub>)

metal oxides

other pyrolysis products typical of burning organic material.

When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles.

May emit poisonous fumes.

A fire in bulk finely divided carbon may not be obviously visible unless the material is disturbed and sparks appear. A straw broom may be useful to produce the disturbance.

Explosion and Ignition Behaviour of Carbon Black with Air

|                                |   |
|--------------------------------|---|
| Lower Limit for Explosion:     | 50 g/m <sup>3</sup> (carbon black in air) |
| Maximum Explosion Pressure:    | 10 bar                                    |
| Maximum Rate of Pressure Rise: | 30-100 bar/sec                            |
| Minimum Ignition Temperature:  | 315 deg. C.                               |
| Ignition Energy:               | >1 kJ                                     |
| Glow Temperature:              | 500 deg. C. (approx.)                     |

Notes on Test Methods:

Tests 1, 2 and 3 were conducted by Bergwerkeschaftliche Versuchsstrecke, Dortmunde-Derne, using a 1 m<sup>3</sup> vessel with two chemical igniters having an intensity of 5000 W.S.

Tests 1 and 2 results are confirmed by information in the Handbook of Powder Technology, Vol. 4 (P. Field)

In Test 4, a modified Godbert-Greenwald furnace was used. See U.S. Bureau of Mines, Report 5624, 1960, p.5, "Lab Equipment and Test Procedures".

Test 5 used a 1 m<sup>3</sup> vessel with chemical igniters of variable intensity.

Test 6 was conducted in a laboratory oven. Active glowing appeared after 3 minutes exposure.

(European Committee for Biological Effects of Carbon Black) (2/84)

HAZCHEM

2Y

## SECTION 6 Accidental release measures

## Personal precautions, protective equipment and emergency procedures

See section 8

## Environmental precautions

See section 12

## Methods and material for containment and cleaning up

|                     |   |
|---------------------|---|
| <b>Minor Spills</b> | <p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> <li>▶ Clean up waste regularly and abnormal spills immediately.</li> <li>▶ Avoid breathing dust and contact with skin and eyes.</li> <li>▶ Wear protective clothing, gloves, safety glasses and dust respirator.</li> <li>▶ Use dry clean up procedures and avoid generating dust.</li> <li>▶ Vacuum up or sweep up. <b>NOTE:</b> Vacuum cleaner must be fitted with an exhaust micro filter (HEPA type) (consider explosion-proof machines designed to be grounded during storage and use).</li> <li>▶ Dampen with water to prevent dusting before sweeping.</li> <li>▶ Place in suitable containers for disposal.</li> </ul>   |
| <b>Major Spills</b> | <p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> <li>· Do not use compressed air to remove metal dusts from floors, beams or equipment</li> <li>· Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation.</li> <li>· Use non-sparking handling equipment, tools and natural bristle brushes.</li> <li>· Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations</li> <li>· Cover and reseal partially empty containers.</li> <li>· Do not allow chips, fines or dusts to contact water, particularly in enclosed areas.</li> </ul> <p>If molten:</p> <ul style="list-style-type: none"> <li>▶ Contain the flow using dry sand or salt flux as a dam.</li> <li>▶ 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.</li> <li>▶ Allow the spill to cool before remelting scrap.</li> </ul> |

## 24V battery pack

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

## SECTION 7 Handling and storage

### Precautions for safe handling

#### Safe handling

#### NOTE:

- Wet, activated carbon removes oxygen from the air thus producing a severe hazard to workers inside carbon vessels and in enclosed or confined spaces where activated carbons might accumulate.
- Before entry to such areas, sampling and test procedures for low oxygen levels should be undertaken; control conditions should be established to ensure the availability of adequate oxygen supply.

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.
    - 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.

## 24V battery pack

|                          |   |
|--------------------------|---|
| <b>Other information</b> | <p>Carbon and charcoal may be stabilised for storage and transport, without moistening, by treatment with hot air at 50 deg. C.. Use of oxygen-impermeable bags to limit oxygen and moisture uptake has been proposed. Surface contamination with oxygenated volatiles may generate a heat of reaction (spontaneous heating). Should stored product reach 110 deg. C., stacked bags should be pulled apart with each bag separated by an air space to permit cooling away from other combustible materials.</p> <ul style="list-style-type: none"> <li>▸ Store in original containers.</li> <li>▸ Keep containers securely sealed.</li> <li>▸ Store in a cool, dry, well-ventilated area.</li> <li>▸ Store away from incompatible materials and foodstuff containers.</li> <li>▸ Protect containers against physical damage and check regularly for leaks.</li> <li>▸ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> </ul> |
|--------------------------|---|

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

|                                |  |
|--------------------------------|--|
| <b>Suitable container</b>      | <p>For low viscosity materials</p> <ul style="list-style-type: none"> <li>▸ Drums and jerricans must be of the non-removable head type.</li> <li>▸ Where a can is to be used as an inner package, the can must have a screwed enclosure.</li> </ul> <p>For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.):</p> <ul style="list-style-type: none"> <li>▸ Removable head packaging;</li> <li>▸ Cans with friction closures and</li> <li>▸ low pressure tubes and cartridges</li> </ul> <p>may be used.</p> <p>-</p> <p>Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages *.</p> <p>-</p> <p>In addition, where inner packagings are glass and contain liquids of packing group I and II there must be sufficient inert absorbent to absorb any spillage *.</p> <p>-</p> <p>* unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.</p>  |
| <b>Storage incompatibility</b> | <p>Inorganic derivative of Group 11 metal.</p> <p>For aluminas (aluminium oxide):</p> <p>Incompatible with hot chlorinated rubber.</p> <p>In the presence of chlorine trifluoride may react violently and ignite.</p> <ul style="list-style-type: none"> <li>-May initiate explosive polymerisation of olefin oxides including ethylene oxide.</li> <li>-Produces exothermic reaction above 200°C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals.</li> <li>-Produces exothermic reaction with oxygen difluoride.</li> <li>-May form explosive mixture with oxygen difluoride.</li> <li>-Forms explosive mixtures with sodium nitrate.</li> <li>-Reacts vigorously with vinyl acetate.</li> </ul> <p>Aluminium oxide is an amphoteric substance, meaning it can react with both acids and bases, such as hydrofluoric acid and sodium hydroxide, acting as an acid with a base and a base with an acid, neutralising the other and producing a salt.</p> <ul style="list-style-type: none"> <li>▸ <b>WARNING:</b> Avoid or control reaction with peroxides. All <i>transition metal</i> peroxides should be considered as potentially explosive. For example transition metal complexes of alkyl hydroperoxides may decompose explosively.</li> <li>▸ 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.</li> <li>▸ Avoid reaction with borohydrides or cyanoborohydrides</li> <li>▸ Contact with water liberates highly flammable gases</li> <li>▸ Many metals may incandesce, react violently, ignite or react explosively upon addition of concentrated nitric acid.</li> </ul> <p>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:</p> <ul style="list-style-type: none"> <li>▸ can react exothermically with oxidising acids to form noxious gases.</li> <li>▸ catalyse polymerisation and other reactions, particularly when finely divided</li> <li>▸ react with halogenated hydrocarbons (for example, copper dissolves when heated in carbon tetrachloride), sometimes forming explosive compounds.</li> </ul> <ul style="list-style-type: none"> <li>▸ 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.</li> <li>▸ Safe handling is possible in relatively low concentrations of oxygen in an inert gas.</li> <li>▸ 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.</li> <li>▸ The reaction residues from various metal syntheses (involving vacuum evaporation and co-deposition with a ligand) are often pyrophoric.</li> </ul> <p>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.</p> <ul style="list-style-type: none"> <li>▸ 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.</li> <li>▸ Elemental metals may react with azo/diazo compounds to form explosive products.</li> <li>▸ Some elemental metals form explosive products with halogenated hydrocarbons.</li> </ul> |



## 24V battery pack

For carbon powders:

- ▶ Avoid oxidising agents, reducing agents.
- ▶ Reaction with finely divided metals, bromates, chlorates, chloramine monoxide, dichlorine oxide, iodates, metal nitrates, oxygen difluoride, peroxyformic acid, peroxyfuroic acid and trioxygen difluoride may result in an exotherm with ignition or explosion. Less active forms of carbon will ignite or explode on suitably intimate contact with oxygen, oxides, peroxides, oxosalts, halogens, interhalogens and other oxidising species.
- ▶ Explosive reaction with ammonium nitrate, ammonium perchlorate, calcium hypochlorite and iodine pentoxide may occur following heating. Carbon may react violently with nitric acid and may be explosively reactive with nitrogen trifluoride at reduced temperatures. In the presence of nitrogen oxide, incandescence and ignition may occur. Finely divided or highly porous forms of carbon, exhibiting a high surface area to mass (up to 2000 m<sup>2</sup>/g) may function as unusually active fuels possessing both adsorptive and catalytic properties which accelerate the release of energy in the presence of oxidising substances. Dry metal-impregnated charcoal catalysts may generate sufficient static, during handling, to cause ignition.
- ▶ Graphite in contact with liquid potassium, rubidium or caesium at 300 deg. C. produces intercalation compounds (C8M) which ignite in air and may react explosively with water. The fusion of powdered diamond and potassium hydroxide may produce explosive decomposition.
- ▶ Activated carbon, when exposed to air, represents a potential fire hazard due to a high surface area and adsorptive capacity. Freshly prepared material may ignite spontaneously in the presence of air especially at high humidity. Spontaneous combustion in air may occur at 90-100 deg. C. The presence of moisture in air facilitates the ignition. Drying oils and oxidising oils promote spontaneous heating and ignition; contamination with these must be avoided. Unsaturated drying oils (linseed oil etc.) may ignite following adsorption owing to an enormous increase in the surface area of oil exposed to air; the rate of oxidation may also be catalysed by metallic impurities in the carbon. A similar, but slower effect occurs on fibrous materials such as cotton waste. Spontaneous heating of activated carbon is related to the composition and method of preparation of the activated carbon. Free radicals, present in charcoal, are responsible for autoignition. Self-heating and autoignition may also result from adsorption of various vapours and gases (especially oxygen). For example, activated carbon auto-ignites in flowing air at 452-518 deg. C.; when the base, triethylenediamine, is adsorbed on the carbon (5%) the autoignition temperature is reduced to 230-260 deg. C.. An exotherm is produced at 230-260 deg. C., at high flow rates of air, although ignition did not occur until 500 deg. C.. Mixtures of sodium borohydride with activated carbons, in air, promote the oxidation of sodium borohydride, producing a self-heating reaction that may result in the ignition of charcoal and in the production of hydrogen through thermal decomposition of the borohydride.



X — Must not be stored together

0 — May be stored together with specific preventions

+ — May be stored together

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

## SECTION 8 Exposure controls / personal protection

### Control parameters

#### Occupational Exposure Limits (OEL)

#### INGREDIENT DATA

| Source                       | Ingredient                                    | Material name  | TWA                   | STEL          | Peak          | Notes   |
|------------------------------|---|--|-----------------------|---------------|---------------|---|
| Australia Exposure Standards | cobalt lithium manganese nickelate            | Manganese, dust & compounds (as Mn)  | 1 mg/m <sup>3</sup>   | Not Available | Not Available | Not Available   |
| Australia Exposure Standards | copper  | Copper, dusts & mists (as Cu)  | 1 mg/m <sup>3</sup>   | Not Available | Not Available | Not Available   |
| Australia Exposure Standards | copper  | Copper (fume)  | 0.2 mg/m <sup>3</sup> | Not Available | Not Available | Not Available   |
| Australia Exposure Standards | graphite                                      | Graphite (all forms except fibres) (respirable dust) (natural & synthetic) | 3 mg/m <sup>3</sup>   | Not Available | Not Available | (e) Containing no asbestos and < 1% crystalline silica. |
| Australia Exposure Standards | aluminium                                     | Aluminium, pyro powders (as Al)  | 5 mg/m <sup>3</sup>   | Not Available | Not Available | Not Available   |
| Australia Exposure Standards | aluminium                                     | Aluminium (metal dust)   | 10 mg/m <sup>3</sup>  | Not Available | Not Available | Not Available   |
| Australia Exposure Standards | aluminium                                     | Aluminium (welding fumes) (as Al)  | 5 mg/m <sup>3</sup>   | Not Available | Not Available | Not Available   |
| Australia Exposure Standards | nickel catalyst, finely divided, >= 40% water | Nickel, powder   | 1 mg/m <sup>3</sup>   | Not Available | Not Available | Not Available   |

## 24V battery pack

| Source                       | Ingredient                                    | Material name | TWA     | STEL          | Peak          | Notes         |
|------------------------------|---|---------------|---------|---------------|---------------|---------------|
| Australia Exposure Standards | nickel catalyst, finely divided, >= 40% water | Nickel, metal | 1 mg/m3 | Not Available | Not Available | Not Available |

## Emergency Limits

| Ingredient                                    | TEEL-1    | TEEL-2    | TEEL-3      |
|---|-----------|-----------|-------------|
| carbon, non-activated                         | 6 mg/m3   | 330 mg/m3 | 2,000 mg/m3 |
| copper  | 3 mg/m3   | 33 mg/m3  | 200 mg/m3   |
| graphite                                      | 6 mg/m3   | 330 mg/m3 | 2,000 mg/m3 |
| ethylene carbonate                            | 30 mg/m3  | 330 mg/m3 | 2,000 mg/m3 |
| nickel catalyst, finely divided, >= 40% water | 4.5 mg/m3 | 50 mg/m3  | 99 mg/m3    |
| lithium                                       | 3.3 mg/m3 | 36 mg/m3  | 220 mg/m3   |

| Ingredient                                    | Original IDLH        | Revised IDLH  |
|---|----------------------|---------------|
| cobalt lithium manganese nickelate            | 500 mg/m3 / 10 mg/m3 | Not Available |
| carbon, non-activated                         | Not Available        | Not Available |
| copper  | 100 mg/m3            | Not Available |
| graphite                                      | 1,250 mg/m3          | Not Available |
| aluminium                                     | Not Available        | Not Available |
| ethylene carbonate                            | Not Available        | Not Available |
| nickel catalyst, finely divided, >= 40% water | 10 mg/m3             | Not Available |
| lithium                                       | Not Available        | Not Available |

## Occupational Exposure Banding

| Ingredient         | Occupational Exposure Band Rating   | Occupational Exposure Band Limit                                  |
|--------------------|---|---|
| ethylene carbonate | E   | ≤ 0.01 mg/m <sup>3</sup>  |
| lithium            | C   | > 0.1 to ≤ milligrams per cubic meter of air (mg/m <sup>3</sup> ) |
| <b>Notes:</b>      | <i>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.</i> |   |

## MATERIAL DATA

For graphite:

Graphite pneumoconiosis resembles coal workers' pneumoconiosis. Data indicate that the higher the crystalline silica content of graphite the more likely the disease will increase in severity. The presence of anthracite coal in the production of some synthetic grades of graphite appears to make arbitrary the use of the term, "synthetic", "artificial" or "natural".

The TLV-TWA for carbon black is recommended to minimise complaints of excessive dirtiness and applies only to commercially produced carbon blacks or to soots derived from combustion sources containing absorbed polycyclic aromatic hydrocarbons (PAHs). When PAHs are present in carbon black (measured as the cyclohexane-extractable fraction) NIOSH has established a REL-TWA of 0.1 mg/m3 and considers the material to be an occupational carcinogen.

The NIOSH REL-TWA was "selected on the basis of professional judgement rather than on data delineating safe from unsafe concentrations of PAHs".

This limit was justified on the basis of feasibility of measurement and not on a demonstration of its safety.

For aluminium oxide and pyrophoric grades of aluminium:

Twenty seven year experience with aluminium oxide dust (particle size 96% 1,2 um) without adverse effects either systemically or on the lung, and at a calculated concentration equivalent to 2 mg/m3 over an 8-hour shift has lead to the current recommendation of the TLV-TWA.

The limit should also apply to aluminium pyro powders whose toxicity is reportedly greater than aluminium dusts and should be protective against lung changes.

For aluminium oxide:

The experimental and clinical data indicate that aluminium oxide acts as an "inert" material when inhaled and seems to have little effect on the lungs nor does it produce significant organic disease or toxic effects when exposures are kept under reasonable control.

[Documentation of the Threshold Limit Values], ACGIH, Sixth Edition


for cobalt:

In view of the serious effects seen in experimental animals after a relatively short exposure period at 0.1 mg/m3 the recommended TLV-TWA is thought to reduce the significant risk of material impairment of health posed by respiratory disease and pulmonary sensitisation which have been shown to occur at higher levels of exposure. The value does not apply generally to cobalt compounds.

A significant increase in the risk of lung cancer was reported among workers involved in cobalt production (with concomitant exposure to nickel and arsenic) and hard-metal workers with documented exposure to cobalt-containing dusts. A significant increase in lung cancer risk has been observed in workers whose exposure began more than 20 years previously. A number of single cases of malignant tumours, mostly sarcomas, have been reported at the site, following implant of cobalt-containing orthopedic implants.

The concentration of dust, for application of respirable dust limits, is to be determined from the fraction that penetrates a separator whose size collection efficiency is described by a cumulative log-normal function with a median aerodynamic diameter of 4.0 µm (+-) 0.3 µm and with a geometric standard deviation of 1.5 µm (+-) 0.1 µm, i.e..generally less than 5 µm.

**Exposure controls**

| <p><b>Appropriate engineering controls</b></p>   | <p>Metal dusts must be collected at the source of generation as they are potentially explosive.</p> <ul style="list-style-type: none"> <li>▶ Avoid ignition sources.</li> <li>▶ Good housekeeping practices must be maintained.</li> <li>▶ Dust accumulation on the floor, ledges and beams can present a risk of ignition, flame propagation and secondary explosions.</li> <li>▶ Do not use compressed air to remove settled materials from floors, beams or equipment</li> <li>▶ Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation.</li> <li>▶ Use non-sparking handling equipment, tools and natural bristle brushes. Cover and reseal partially empty containers. Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations.</li> <li>▶ Do not allow chips, fines or dusts to contact water, particularly in enclosed areas.</li> <li>▶ Metal spraying and blasting should, where possible, be conducted in separate rooms. This minimises the risk of supplying oxygen, in the form of metal oxides, to potentially reactive finely divided metals such as aluminium, zinc, magnesium or titanium.</li> <li>▶ Work-shops designed for metal spraying should possess smooth walls and a minimum of obstructions, such as ledges, on which dust accumulation is possible.</li> <li>▶ Wet scrubbers are preferable to dry dust collectors.</li> <li>▶ Bag or filter-type collectors should be sited outside the workrooms and be fitted with explosion relief doors.</li> <li>▶ Cyclones should be protected against entry of moisture as reactive metal dusts are capable of spontaneous combustion in humid or partially wetted states.</li> <li>▶ Local exhaust systems must be designed to provide a minimum capture velocity at the fume source, away from the worker, of 0.5 metre/sec.</li> <li>▶ Local ventilation and vacuum systems must be designed to handle explosive dusts. Dry vacuum and electrostatic precipitators must not be used, unless specifically approved for use with flammable/ explosive dusts.</li> </ul> <p>Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p> <table border="1" data-bbox="389 1151 1353 1227"> <tr> <td>Type of Contaminant:</td> <td>Air Speed:</td> </tr> <tr> <td>welding, brazing fumes (released at relatively low velocity into moderately still air)</td> <td>0.5-1.0 m/s (100-200 f/min.)</td> </tr> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="389 1317 1174 1505"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p> <p>Exhaust ventilation should be designed to prevent accumulation and recirculation in the workplace and safely remove carbon black from the air.</p> <p>Note: Wet, activated carbon removes oxygen from the air and thus presents a severe hazard to workers inside carbon vessels and enclosed or confined spaces. Before entering such areas sampling and test procedures for low oxygen levels should be undertaken and control conditions set up to ensure ample oxygen availability.[Linde]</p> | Type of Contaminant: | Air Speed: | welding, brazing fumes (released at relatively low velocity into moderately still air) | 0.5-1.0 m/s (100-200 f/min.) | Lower end of the range | Upper end of the range | 1: Room air currents minimal or favourable to capture | 1: Disturbing room air currents | 2: Contaminants of low toxicity or of nuisance value only. | 2: Contaminants of high toxicity | 3: Intermittent, low production. | 3: High production, heavy use | 4: Large hood or large air mass in motion | 4: Small hood-local control only |
|--|---|----------------------|------------|--|------------------------------|------------------------|------------------------|---|---------------------------------|--|----------------------------------|----------------------------------|-------------------------------|---|----------------------------------|
| Type of Contaminant:   | Air Speed:  |                      |            |  |                              |                        |                        |   |                                 |  |                                  |                                  |                               |   |                                  |
| welding, brazing fumes (released at relatively low velocity into moderately still air) | 0.5-1.0 m/s (100-200 f/min.)  |                      |            |  |                              |                        |                        |   |                                 |  |                                  |                                  |                               |   |                                  |
| Lower end of the range   | Upper end of the range  |                      |            |  |                              |                        |                        |   |                                 |  |                                  |                                  |                               |   |                                  |
| 1: Room air currents minimal or favourable to capture                                  | 1: Disturbing room air currents   |                      |            |  |                              |                        |                        |   |                                 |  |                                  |                                  |                               |   |                                  |
| 2: Contaminants of low toxicity or of nuisance value only.                             | 2: Contaminants of high toxicity  |                      |            |  |                              |                        |                        |   |                                 |  |                                  |                                  |                               |   |                                  |
| 3: Intermittent, low production.   | 3: High production, heavy use   |                      |            |  |                              |                        |                        |   |                                 |  |                                  |                                  |                               |   |                                  |
| 4: Large hood or large air mass in motion  | 4: Small hood-local control only  |                      |            |  |                              |                        |                        |   |                                 |  |                                  |                                  |                               |   |                                  |
| <p><b>Personal protection</b></p>  |   |                      |            |  |                              |                        |                        |   |                                 |  |                                  |                                  |                               |   |                                  |
| <p><b>Eye and face protection</b></p>  | <ul style="list-style-type: none"> <li>▶ Safety glasses with side shields.</li> <li>▶ Chemical goggles.</li> <li>▶ 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.</li> </ul>   |                      |            |  |                              |                        |                        |   |                                 |  |                                  |                                  |                               |   |                                  |

## 24V battery pack

|                              |  |
|------------------------------|--|
|                              | <p>Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]</p>   |
| <b>Skin protection</b>       | See Hand protection below  |
| <b>Hands/feet protection</b> | <ul style="list-style-type: none"> <li>▸ Wear chemical protective gloves, e.g. PVC.</li> <li>▸ Wear safety footwear or safety gumboots, e.g. Rubber</li> </ul> <p><b>NOTE:</b></p> <ul style="list-style-type: none"> <li>▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.</li> <li>▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.</li> </ul> <p>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.</p> <p>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.</p> <p>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.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> <li>· frequency and duration of contact,</li> <li>· chemical resistance of glove material,</li> <li>· glove thickness and</li> <li>· dexterity</li> </ul> <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> <li>· 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.</li> <li>· 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.</li> <li>· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.</li> <li>· Contaminated gloves should be replaced.</li> </ul> <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> <li>· Excellent when breakthrough time &gt; 480 min</li> <li>· Good when breakthrough time &gt; 20 min</li> <li>· Fair when breakthrough time &lt; 20 min</li> <li>· Poor when glove material degrades</li> </ul> <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>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.</p> <p>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.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> <li>· 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.</li> <li>· 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</li> </ul> <p>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.</p> <ul style="list-style-type: none"> <li>▸ Protective gloves eg. Leather gloves or gloves with Leather facing</li> </ul> |
| <b>Body protection</b>       | See Other protection below   |
| <b>Other protection</b>      | <ul style="list-style-type: none"> <li>▸ Protective overalls, closely fitted at neck and wrist.</li> <li>▸ Eye-wash unit.</li> </ul> <p><b>IN CONFINED SPACES:</b></p> <ul style="list-style-type: none"> <li>▸ Non-sparking protective boots</li> <li>▸ Static-free clothing.</li> <li>▸ Ensure availability of lifeline.</li> </ul> <p>Staff should be trained in all aspects of rescue work.</p> <p>Rescue gear: Two sets of SCBA breathing apparatus Rescue Harness, lines etc.</p>  |

**Respiratory protection**

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

| Required Minimum Protection Factor | Half-Face Respirator | Full-Face Respirator | Powered Air Respirator |
|------------------------------------|----------------------|----------------------|------------------------|
| up to 10 x ES                      | A P1<br>Air-line*    | -<br>-               | A PAPR-P1<br>-         |
| up to 50 x ES                      | Air-line**           | A P2                 | A PAPR-P2              |

## 24V battery pack

|                |   |            |           |
|----------------|---|------------|-----------|
| up to 100 x ES | - | A P3       | -         |
|                |   | Air-line*  | -         |
| 100+ x ES      | - | Air-line** | A PAPR-P3 |

\* - Negative pressure demand \*\* - Continuous flow

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(SO<sub>2</sub>), G = Agricultural chemicals, K = Ammonia(NH<sub>3</sub>), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

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

Where significant concentrations of the material are likely to enter the breathing zone, a Class P3 respirator may be required.

Class P3 particulate filters are used for protection against highly toxic or highly irritant particulates.

Filtration rate: Filters at least 99.95% of airborne particles

Suitable for:

- Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.
- Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.
- Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS
- Highly toxic particles e.g. Organophosphate Insecticides, Radionuclides, Asbestos

Note: P3 Rating can only be achieved when used with a Full Face Respirator or Powered Air-Purifying Respirator (PAPR). If used with any other respirator, it will only provide filtration protection up to a P2 rating.

## SECTION 9 Physical and chemical properties

### Information on basic physical and chemical properties

|   |                |  |                |
|---|----------------|--|----------------|
| <b>Appearance</b>                                   | Dark           |  |                |
| <b>Physical state</b>                               | Solid          | <b>Relative density (Water = 1)</b>            | Not Available  |
| <b>Odour</b>  | Not Available  | <b>Partition coefficient n-octanol / water</b> | Not Available  |
| <b>Odour threshold</b>                              | Not Available  | <b>Auto-ignition temperature (°C)</b>          | Not Available  |
| <b>pH (as supplied)</b>                             | Not Available  | <b>Decomposition temperature (°C)</b>          | Not Available  |
| <b>Melting point / freezing point (°C)</b>          | Not Available  | <b>Viscosity (cSt)</b>                         | Not Available  |
| <b>Initial boiling point and boiling range (°C)</b> | Not Available  | <b>Molecular weight (g/mol)</b>                | Not Available  |
| <b>Flash point (°C)</b>                             | Not Available  | <b>Taste</b>                                   | Not Available  |
| <b>Evaporation rate</b>                             | Not Available  | <b>Explosive properties</b>                    | Not Available  |
| <b>Flammability</b>                                 | Not Available  | <b>Oxidising properties</b>                    | Not Available  |
| <b>Upper Explosive Limit (%)</b>                    | Not Available  | <b>Surface Tension (dyn/cm or mN/m)</b>        | Not Applicable |
| <b>Lower Explosive Limit (%)</b>                    | Not Available  | <b>Volatile Component (%vol)</b>               | Not Available  |
| <b>Vapour pressure (kPa)</b>                        | Not Available  | <b>Gas group</b>                               | Not Available  |
| <b>Solubility in water</b>                          | Not Applicable | <b>pH as a solution (Not Available%)</b>       | Not Available  |
| <b>Vapour density (Air = 1)</b>                     | Not Available  | <b>VOC g/L</b>                                 | Not Available  |

## 24V battery pack

**SECTION 10 Stability and reactivity**

|   |  |
|---|--|
| <b>Reactivity</b>                         | See section 7  |
| <b>Chemical stability</b>                 | <ul style="list-style-type: none"> <li>▸ Unstable in the presence of incompatible materials.</li> <li>▸ Product is considered stable.</li> <li>▸ Hazardous polymerisation will not occur.</li> </ul> |
| <b>Possibility of hazardous reactions</b> | See section 7  |
| <b>Conditions to avoid</b>                | See section 7  |
| <b>Incompatible materials</b>             | See section 7  |
| <b>Hazardous decomposition products</b>   | See section 5  |

**SECTION 11 Toxicological information****Information on toxicological effects**

|                |   |
|----------------|---|
| <b>Inhaled</b> | <p>Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial 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.</p> <p>Inhalation of dusts, generated by the material during the course of normal handling, may produce severe damage to the health of the individual. Relatively small amounts absorbed from the lungs may prove fatal.</p> <p>Side effects of the inhalation of cobalt and its compounds may include flushing of the face and ringing in the ears (tinnitus). Cobalt inhalation can be lethal in animals if exposure is sufficiently high or prolonged. The acute LC50 for a 30-minute inhalation exposure in rats was 165 mg cobalt/m<sup>3</sup> as cobalt hydrocarbonyl. Exposure to 9 mg cobalt/m<sup>3</sup> as cobalt hydrocarbonyl for 6 hours/day, 5 days/week for 3 months resulted in 16 deaths out of 75 rats. Death was reported in rats and mice exposed to 19 mg cobalt/m<sup>3</sup> (but not 1.9 mg cobalt/m<sup>3</sup>) as cobalt sulfate over 16 days, but exposure to 11.4 mg cobalt/m<sup>3</sup> over 13 weeks was lethal only to mice and not to rats. Exposure to 1.14 mg cobalt/m<sup>3</sup> as cobalt sulfate for 104 weeks resulted in no increase in mortality in rats and mice of either sex.</p> <p>Inhalation of stable cobalt by humans and/or animals resulted in respiratory, cardiovascular, hematological, hepatic, renal, endocrine, ocular, and body weight effects. As with exposures in humans, exposures of animals to cobalt-containing aerosols have resulted in pronounced respiratory effects. Animals exposed to aerosols of cobalt oxides and cobalt sulfate developed respiratory effects that varied in severity with exposure level and duration. A single 30-minute exposure of rats to relatively high levels (26-236 mg cobalt/m<sup>3</sup> as cobalt hydrocarbonyl) resulted in congestion, edema, and hemorrhage of the lung. Prolonged exposure (3-4 months) of rats and rabbits to mixed cobalt oxides (0.4-9 mg cobalt/m<sup>3</sup>) resulted in lesions in the alveolar region of the respiratory tract characterised histologically by nodular accumulation of Type II epithelial cells, accumulations of enlarged highly vacuolated macrophages, interstitial inflammation, and fibrosis. In at least one instance, the lesions appeared to regress when exposure was terminated. Guinea pigs sensitized to cobalt by repeated dermal application and then exposed to 2.4 mg cobalt/m<sup>3</sup> as cobalt chloride showed pulmonary inflammatory changes (altered BAL fluid recovery, increased neutrophils and eosinophils in the recovered BAL fluid) that were different than those in exposed animals not sensitised to cobalt. Decreased lung compliance was found in pigs exposed to 0.1 mg cobalt/m<sup>3</sup> as cobalt dust for 3 months. Lifetime exposure of hamsters to 7.9 mg cobalt/m<sup>3</sup> as cobalt oxide resulted in emphysema. Necrosis and inflammation of the respiratory tract epithelium (nasal turbinates, larynx, trachea, bronchioles) were reported in rats exposed to 19 mg cobalt/m<sup>3</sup> and mice exposed to 1.9 mg cobalt/m<sup>3</sup> or greater as cobalt sulfate over 16 days. Exposure of rats and mice to cobalt as cobalt sulfate for 13 weeks resulted in adverse effects on all parts of the respiratory tract, with the larynx being the most sensitive part.</p> <p>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. Although carbon itself has no toxic action, associated impurities may be toxic. Iodine is often found as an impurity and air-borne carbon dusts, as a result, may produce irritation of the mucous membranes, the eyes, and skin. Symptoms of exposure may include coughing, irritation of the nose and throat and burning of the eyes.</p> <p>Copper poisoning following exposure to copper dusts and fume may result in headache, cold sweat and weak pulse. Capillary, kidney, liver and brain damage are the longer term manifestations of such poisoning. 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.</p> |
|----------------|---|

## 24V battery pack

|  |   |
|--|---|
|  | <p>Inhalation of dusts, generated by the material, during the course of normal handling, may produce severely toxic effects; these may be fatal.</p>  |
| <p style="text-align: center;"><b>Ingestion</b></p>    | <p>Severely toxic effects may result from the accidental ingestion of the material; animal experiments indicate that ingestion of less than 5 gram may be fatal or may produce serious damage to the health of the individual.</p> <p>Large doses of lithium ion have caused dizziness and prostration and can cause kidney damage if sodium intake is limited. Dehydration, weight-loss, dermatological effects and thyroid disturbances have been reported. Central nervous system effects that include slurred speech, blurred vision, sensory loss, impaired concentration, irritability, lethargy, confusion, disorientation, drowsiness, anxiety, spasticity, delirium, stupor, ataxia (loss of muscle coordination), sedation, fine and gross tremor, giddiness, twitching and convulsions may occur. Diarrhoea, vomiting and neuromuscular effects such as tremor, clonus (rapid contraction and relaxation of muscles) and hyperactive reflexes may occur as a result of repeated exposure to lithium.</p> <p>Acute severe overexposure may affect the kidneys, resulting in renal dysfunction, albuminuria, oliguria and degenerative changes. Cardiovascular effects may also result in cardiac arrhythmias and hypotension.</p> <p>The primary target organ for lithium toxicity is the central nervous system. Lithium is therefore used therapeutically on membrane transport proteins in the central nervous system when treating manic-depression. Lithium is moderately toxic with lethal dose of LiCl in rats of 526-840 mg/kg body weight. After chronic exposure to 1 meq/L decreased brain weight was observed in male offspring. Chemically, lithium resembles sodium, but is more toxic: in humans 5 g LiCl can result in fatal poisoning. In therapeutic doses, damages on the central nervous system and the kidneys have been reported.</p> <p>Ingestion of finely divided carbon may produce gagging and constipation. Aspiration does not appear to be a concern as the material is generally regarded as inert and is often used as a food additive. Ingestion may produce a black stool.</p> <p>Studies have shown that soluble cobalt compounds are generally more acutely toxic than insoluble cobalt compounds. When expressed in terms of the cobalt ion for the sake of comparison, however, the differences in lethality values from the available studies are within an order of magnitude</p> <p>Animal test indicate an increase in red blood cells (polycythaemia) following the absorption of cobalt salts. [ICI] In toxic doses soluble cobalt salts act locally on the gastro-intestinal tract to produce pain and vomiting. Systemic effects in man include a peculiar vasodilation (flushing) of the face and ears, mild hypotension, rash, tinnitus (ringing in the ears) and nerve deafness. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products]</p> <p>Numerous cases of a single oral exposure to high levels of copper have been reported. Consumption of copper-contaminated drinking water has been associated with mainly gastrointestinal symptoms including nausea, abdominal pain, vomiting and diarrhoea. A metallic taste, nausea, vomiting and epigastric burning often occur after ingestion of copper and its derivatives. The vomitus is usually green/blue and discolours contaminated skin. Acute poisonings from the ingestion of copper salts are rare due to their prompt removal by vomiting. Vomiting is due mainly to the local and astringent action of copper ion on the stomach and bowel. Emesis usually occurs within 5 to 10 minutes but may be delayed if food is present in the stomach. Should vomiting not occur, or is delayed, gradual absorption from the bowel may result in systemic poisoning with death, possibly, following within several days. Apparent recovery may be followed by lethal relapse. Systemic effects of copper resemble other heavy metal poisonings and produce wide-spread capillary damage, kidney and liver damage and central nervous system excitation followed by depression. Haemolytic anaemia (a result of red-blood cell damage) has been described in acute human poisoning. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products.]</p> <p>Other symptoms of copper poisoning include lethargy, neurotoxicity, and increased blood pressure and respiratory rates. Coma and death have followed attempted suicides using solutions of copper sulfate. Copper is an essential element and most animal tissues have measurable amounts of copper associated with them. Humans have evolved mechanisms which maintain its availability whilst limiting its toxicity (homeostasis). Copper is initially bound in the body to a blood-borne protein, serum albumin and thereafter is more firmly bound to another protein, alpha-ceruloplasmin. Such binding effectively "inactivates" the copper, thus reducing its potential to produce toxic damage. In healthy individuals, bound copper can reach relatively high levels without producing adverse health effects. Excretion in the bile represents the major pathway by which copper is removed from the body when it reaches potentially toxic levels. Copper may also be stored in the liver and bone marrow where it is bound to another protein, metallothionein. A combination of binding and excretion ensures that the body is able to tolerate relatively high loadings of copper.</p> <p>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.</p> |
| <p style="text-align: center;"><b>Skin Contact</b></p> | <p>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.</p> <p>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.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus.</p> <p>Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles.</p>   |

## 24V battery pack

|   |   |
|---|---|
|   | <p>Exposure to copper, by skin, has come from its use in pigments, ointments, ornaments, jewellery, dental amalgams and IUDs and as an antifungal agent and an algicide. Although copper algicides are used in the treatment of water in swimming pools and reservoirs, there are no reports of toxicity from these applications. Reports of allergic contact dermatitis following contact with copper and its salts have appeared in the literature, however the exposure concentrations leading to any effect have been poorly characterised. In one study, patch testing of 1190 eczema patients found that only 13 (1.1%) cross-reacted with 2% copper sulfate in petrolatum. The investigators warned, however, that the possibility of contamination with nickel (an established contact allergen) might have been the cause of the reaction. Copper salts often produce an itching eczema in contact with skin. This is, likely, of a non-allergic nature.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>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.</p>   |
| <p style="text-align: center;"><b>Eye</b></p>     | <p>When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.</p> <p>Symptoms of exposure by the eye to carbon particulates include irritation and a burning sensation. Following an industrial explosion, fine particles become embedded in the cornea and conjunctiva resulting in an inflammation which persisted for 2-3 weeks. Some particles remained permanently producing a punctate purplish-black discolouration.</p> <p>Copper salts, in contact with the eye, may produce conjunctivitis or even ulceration and turbidity of the cornea.</p>  |
| <p style="text-align: center;"><b>Chronic</b></p> | <p>On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.</p> <p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.</p> <p>Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers</p> <p>Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.</p> <p>Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.</p> <p>There is sufficient evidence to provide a strong presumption that human exposure to the material may produce heritable genetic damage.</p> <p>There is sufficient evidence to provide a strong presumption that human exposure to the material may result in the development of heritable genetic damage, generally on the basis of</p> <ul style="list-style-type: none"> <li>- appropriate animal studies,</li> <li>- other relevant information</li> </ul> <p>Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.</p> <p>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.</p> <p>There is sufficient evidence to establish a causal relationship between human exposure to the material and impaired fertility</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminum oxide during abrasives production.</p> <p>Very fine Al<sub>2</sub>O<sub>3</sub> powder was not fibrogenic in rats, guinea pigs, or hamsters when inhaled for 6 to 12 months and sacrificed at periods up to 12 months following the last exposure.</p> <p>When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C.</p> <p>The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis (aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity.</p> |



## 24V battery pack

Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction.

There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveoli (sub 5  $\mu\text{m}$ ) are able to produce pathogenic effects in the lungs.

In general, available cohort studies in humans have not reported a significant increase in total mortality as a result of cobalt exposure. Several studies have noted increased mortality rates resulting from lung cancer following occupational exposure to cobalt, either as a mixture of cobalt compounds or as hard metal, a metal alloy with a tungsten carbide and cobalt matrix. Fatal cases of hard metal disease and cardiomyopathy believed to have resulted from occupational cobalt exposure have also been reported. However, in the majority of these and other reported occupational studies, co-exposure to other substances was common, and was unable to be corrected for in the analysis.

The effects of chronic occupational exposure to cobalt and cobalt compounds on the respiratory system in humans are well-documented. These effects include respiratory irritation, diminished pulmonary function, wheezing, asthma, pneumonia, and fibrosis and occurred at exposure levels ranging from 0.007 to 0.893 mg cobalt/ $\text{m}^3$  (exposure from 2 to 17 years). These effects have been observed in workers employed in cobalt refineries, as well as hard metal workers, diamond polishers, and ceramic dish painters (painting with cobalt blue dye).

Occupational asthma attributed to the inhalation of cobalt powder has been confirmed following bronchial challenge tests. Chest tightness and chronic bronchitis have been recorded in hard-metal workers exposed to cobalt. Cobalt is known to function as a hapten, resulting in the generation of antibodies against cobalt-protein complexes. Although the minimum exposure level associated with cobalt sensitisation has not been determined, sensitisation has been demonstrated in hard metal workers with work-related asthma who have experienced prolonged occupational exposure (>3 years) to levels ranging from 0.007 to 0.893 mg cobalt/ $\text{m}^3$ . The sensitisation phenomenon includes the production of IgE and IgA antibodies to cobalt. Exposure to inhaled cobalt chloride aerosols can precipitate an asthmatic attack in sensitised individuals believed to be the result of an allergic reaction within the lungs.

Allergic dermatitis of an erythematous papular type may also occur following occupational exposure. Dermatitis is a common result of dermal exposure to cobalt in humans that has been verified in a large number of studies. Using patch tests and intradermal injections, it has been demonstrated that the dermatitis is probably caused by an allergic reaction to cobalt. Contact allergy was reported in 22 of 223 (9.9%) nurses who were tested with a patch test of 1.0% cobalt chloride as well as 16 of 79 (20.3%) of examined dentists. Persons with body piercings showed an increased prevalence of allergy to cobalt, with the incidence of contact allergy being proportional to number of piercings. The prevalence of sensitivity to cobalt following exposure to cobalt as a component of metal implants is low, with only 3.8% of patients developing a new sensitivity to cobalt following insertion of the implant.

Exposure levels associated with the development of dermatitis have not been identified. It appears that the allergic properties of cobalt result mainly from exposure to the metal itself, rather than a salt, as it has been demonstrated that daily repeated exposure to aqueous cobalt salts did not result in hand eczema in patients known to have cobalt allergy.

Occupational exposure to cobalt in humans has been reported to cause several effects on the nervous system, including memory loss, nerve deafness, and a decreased visual acuity. It should be noted though, that both of the studies reporting on these findings, had small numbers of subjects, and exposure characterization was not reported.

Chronic exposure to cobalt produces polycythaemia (increase in blood haemoglobin), increased production of cells of the bone marrow and thyroid gland, pericardial effusion and damage to the alpha cells of the pancreas. Chronic exposure to cobalt compounds may result in pericardial effusion, polycardial effusion, cardiac failure, vomiting, convulsions and thyroid enlargement. Chronic administration of cobaltous chloride has produced goiter, reduced thyroid activity and lowered synthesis rates and levels of cytochrome P-450, an enzymatic system responsible for chemical detoxification, in the liver. A toxic nephritis (kidney disease) may also develop.

Epidemic cardiomyopathy (heart disease) among heavy beer drinkers in the 1960's in Canada, the USA and Belgium has been attributed to the addition of up to 1.5 ppm of cobalt as a foam restorative and stabiliser. Other factors are probably implicated as therapeutic doses of cobalt, up to 50 mg/day (in the treatment of refractory anaemias) do not produce this effect. Inadequate protein or vitamin intake amongst heavy drinkers, or the effects of alcohol in rendering the heart more susceptible to disease may be important.

Single and repeated subcutaneous or intramuscular injection of cobalt powder and salts to rats may cause sarcoma at the injection site but evidence for carcinogenicity by any other route of exposure does not exist. A number of single cases of malignant tumours, mostly sarcomas, have been reported at the site of orthopedic implants containing cobalt.

Animals, exposed to cobalt compounds also exhibit an increase in respiration, as well as tremor and convulsion. Exposure of rats and mice to aerosols of cobalt (as cobalt sulfate) at concentrations from 0.11 to 1.14 mg cobalt/ $\text{m}^3$  for 2 years resulted in a spectrum of inflammatory, fibrotic, and proliferative lesions in the respiratory tract of male and female rats and mice. Squamous metaplasia of the larynx occurred in rats and mice at exposure concentrations of .0.11 mg cobalt/ $\text{m}^3$ , with severity of the lesion increasing with increased cobalt concentration. Hyperplastic lesions of the nasal epithelium occurred in rats at concentrations of .0.11 mg cobalt/ $\text{m}^3$ , and in mice at concentrations of .0.38 mg cobalt/ $\text{m}^3$ . Both sexes of rats had greatly increased incidences (>90% incidence) of alveolar lesions at all exposure levels, including inflammatory changes, fibrosis, and metaplasia. Similar changes were seen in mice at all exposure levels, though the changes in mice were less severe.

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

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

## 24V battery pack

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.

The most common toxic reaction to nickel is skin sensitisation which may produce a chronic eczema called "nickel itch". The first symptom is itching which occurs up to 7 days prior to the appearance of skin eruption. The primary skin eruption is erythematous or follicular and may be followed by superficial discrete ulcers (which discharge and become crusted), or eczema. In the chronic stages, pigmented or depigmented plaques may be formed. Recovery from the dermatitis usually occurs within 7 days but may take several weeks.

Nickel dusts and several specific compounds are carcinogenic in animals following inhalation or parenteral administration (but not by ingestion or skin contact). Increases in lung and nasal cavity cancers have been observed amongst nickel workers in smelters and refineries. Respiratory cancer risks primarily relate to chronic exposure to soluble nickels at concentrations in excess of 1 mg Ni/m<sup>3</sup> and exposure to the less soluble forms at concentrations greater than 10 mg Ni/m<sup>3</sup>. Metallic nickel does not appear to pose such a threat.

Prolonged or repeated inhalation of dust may result in pneumoconiosis (lung disease caused by inhalation dust).

Graphite workers have reported symptoms of headaches, coughing, depression, low appetite, dyspnoea (difficult breathing) and black sputum.

A number of studies indicate that graphitosis is a progressive and disabling disease and that the presence of crystalline silica and some silicates as graphite impurities have a pronounced synergistic effect.

Workers suffering from graphite pneumoconiosis have generally worked in the industry for long periods, i.e. 10 years or more, although some cases have been reported after as little as four years.

Data indicate the higher the crystalline silica content of graphite the greater is the severity of the pneumoconiosis.

Pre-employment and periodic examinations should be directed towards detecting significant respiratory disease through chest X-rays and pulmonary function tests

Chronic inhalation exposure of production workers has caused decreased pulmonary function and myocardial dystrophy. There is suggestive but inconclusive evidence that carbon black containing polyaromatic hydrocarbons (PAHs) has been responsible for induction of skin cancers in exposed workers.

Long term inhalation of carbon black can cause cough, phlegm, tiredness, chest pain and headache. Dermal, mucosal, or inhalation exposure can cause irritation.

Inhalation of carbon black by mice, rats and monkeys caused thickened alveolar walls, increased pulmonary collagen, right atrial and ventricular strain, hypertrophy of the right atrial and ventricular septum and increased heart weights. Although carbon black itself did not cause cancer in treated animals, carbon black containing polyaromatic hydrocarbons (PAHs) did cause cancer following chronic administration by all routes tested.

Epidemiological studies of workers in the carbon black producing industries of North America and Western Europe show no significant health effect due to occupational exposure to carbon black. Several other studies provide conflicting evidence. Early studies in the former USSR and Eastern Europe report respiratory diseases amongst workers exposed to carbon black, including bronchitis, pneumonia, emphysema and rhinitis. These studies are of questionable validity due to inadequate study design and methodology, lack of appropriate controls for cigarette smoking and other confounding factors such as concurrent exposure to carbon dioxide, coal oil and petroleum vapours. Moreover, review of these studies indicates that the concentrations of carbon black were greater than current occupational standards.

Carbon black may cause adverse pulmonary changes following prolonged or repeated inhalation of the dust; these include oral mucosal lesions, bronchitis and pneumoconiosis which may lead to lung tumours.

The body of evidence of carcinogenicity in animal studies comes from two chronic inhalation studies and two intratracheal instillation studies in rats, which showed significantly elevated rates of lung cancer in exposed animals. An inhalation study was tested on mice, but did not show significantly elevated rates of lung cancer in exposed animals. Epidemiologic data comes from three different cohort studies of carbon black production workers. Two studies, from the United Kingdom and Germany, with over 1,000 workers in each study group, showed elevated mortality from lung cancer in the carbon black workers. Another study of over 5,000 workers in the United States did not show elevated mortality from lung cancer in the carbon black workers. Newer findings of increased lung cancer mortality in an update from the UK study may suggest that carbon black could be a late-stage carcinogen. However, a more recent and larger study from Germany did not confirm this hypothesis that carbon black acts as a late-stage carcinogen.

## 24V battery pack

In studies employing channel and furnace black, hamsters, mice, guinea pigs, rabbits and monkeys exposed to dusts for 7 hours/day, 5 days/week, at concentrations of 87.4 mg/m<sup>3</sup> for channel black and 56.5 mg/m<sup>3</sup> for furnace black, no malignancies were observed in any of the animals. Channel black had little if any absorbed polyaromatic hydrocarbons (PAHs) (as benzene extractables) whilst furnace black had 0.28%.

Several findings have strengthened the association between inflammation and cancer and between the particle surface area dose of carbon black and other poorly soluble low toxicity (PSLT) particles and the pulmonary inflammation response in mice and the proinflammatory effects in lung cells in vitro. Other evidence suggests that in addition to a cancer mechanism involving indirect genotoxicity through inflammation and oxidative stress, nanoparticles may act as direct carcinogens.

Carbon black appears to act like PSLT particles, which can elicit lung tumours in rats following prolonged exposure to sufficiently high concentrations of particles. Particle surface area dose was found to be most predictive of pulmonary inflammation and tumour response in rats when comparing the dose-response relationships for various types and sizes of PSLT including carbon black. Compared to fine PSLT, much lower concentrations of ultrafine PSLT (e.g. 2.5, 6.5 or 11.5 mg/m<sup>3</sup> carbon black and ~10 mg/m<sup>3</sup> ultrafine titanium dioxide) were associated with impaired clearance, persistent inflammation, and malignant lung tumours in chronic inhalation studies in rats. Most evidence suggests that carbon black and other PSLT-elicited lung tumours occurs through a secondary genotoxic mechanism, involving chronic inflammation and oxidative stress. Experimental studies have shown that when the particle lung dose reaches a sufficiently high concentration (e.g., mass dose of ~0.5 mg fine-sized PSLT/g lung in rats), the alveolar macrophage-mediated clearance process begins to be impaired (complete impairment occurs at ~10 mg/g lung. Overloading of lung clearance is accompanied by pulmonary inflammation, leading to increased production of reactive oxygen and nitrogen species, depletion of antioxidants and/or impairment of other defense mechanisms, cell injury, cell proliferation, fibrosis, and as seen in rats, induction of mutations and eventually cancer. Rats appear to be more sensitive to carbon black and other PSLT than other rodent species. Although studies in humans have not shown a direct link between inhaled PSLT and lung cancer, many of the steps in the mechanism observed in rats have also been observed in humans who work in dusty jobs, including increased particle lung retention and pulmonary inflammation in workers exposed to coal dust or crystalline silica and elevated lung cancer has been observed in some studies of workers exposed to carbon black, crystalline silica, and diesel exhaust particles.

Monkeys exposed to channel black for 1000-1500 hours showed evidence of electrocardiac changes indicative of right atrial and right ventricular strain. These changes increased progressively until after 10,000 hours of exposure, when the changes were marked. The authors of this study concluded that there was no significant effect due to prolonged exposure other than those expected from the accumulation of non-toxic dusts in the pulmonary system. Exposure to furnace black produced a similar picture although electrocardiographic change was first observed in monkeys after 2500 hours' exposure and marked atrial and right ventricular strain after 10,000 hours' exposure. The authors concluded that there was no significant effect due to prolonged exposure other than those expected from the accumulation of nontoxic dusts in the pulmonary system. Exposure to furnace black produced a similar picture although electrocardiographic change was first observed in monkeys after 2500 hours exposure and marked atrial and right ventricular strain after 10,000 hours exposure.

Chromatographic fractions of oily material extracted from carbon black have been shown to be carcinogenic whilst the unfractionated extracts are not. The activity of some carcinogens appear to be inhibited by carbon black itself.

For copper and its compounds (typically copper chloride):

Acute toxicity: There are no reliable acute oral toxicity results available. Animal testing shows that skin in exposure to copper may lead to hardness of the skin, scar formation, exudation and reddish changes. Inflammation, irritation and injury of the skin were noted.

Repeat dose toxicity: Animal testing shows that very high levels of copper monochloride may cause anaemia.

Genetic toxicity: Copper monochloride does not appear to cause mutations in vivo, although chromosomal aberrations were seen at very high concentrations in vitro.

Cancer-causing potential: There was insufficient information to evaluate the cancer-causing activity of copper monochloride.

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.

## 24V battery pack

## TOXICITY

Not Available

## IRRITATION

Not Available

## 24V battery pack

|   |   |  |
|---|---|--|
| cobalt lithium manganese nickelate            | <b>TOXICITY</b>   | <b>IRRITATION</b>  |
|   | dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup><br>Oral (Rat) LD50; >2000 mg/kg <sup>[1]</sup>  | Not Available  |
| carbon, non-activated                         | <b>TOXICITY</b>   | <b>IRRITATION</b>  |
|   | Oral (Rat) LD50; >2000 mg/kg <sup>[1]</sup>   | Eye: no adverse effect observed (not irritating) <sup>[1]</sup><br>Skin: no adverse effect observed (not irritating) <sup>[1]</sup>  |
| copper  | <b>TOXICITY</b>   | <b>IRRITATION</b>  |
|   | dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup><br>Inhalation(Rat) LC50; 0.733 mg/l4h <sup>[1]</sup>  | Eye: no adverse effect observed (not irritating) <sup>[1]</sup><br>Skin: no adverse effect observed (not irritating) <sup>[1]</sup>  |
|   | Oral (Mouse) LD50; 0.7 mg/kg <sup>[2]</sup>   |  |
| graphite                                      | <b>TOXICITY</b>   | <b>IRRITATION</b>  |
|   | Inhalation(Rat) LC50; >2 mg/L4h <sup>[1]</sup><br>Oral (Rat) LD50; >2000 mg/kg <sup>[1]</sup>   | Not Available  |
| aluminium                                     | <b>TOXICITY</b>   | <b>IRRITATION</b>  |
|   | Inhalation(Rat) LC50; >2.3 mg/l4h <sup>[1]</sup><br>Oral (Rat) LD50; >2000 mg/kg <sup>[1]</sup>   | Eye: no adverse effect observed (not irritating) <sup>[1]</sup><br>Skin: no adverse effect observed (not irritating) <sup>[1]</sup>  |
| ethylene carbonate                            | <b>TOXICITY</b>   | <b>IRRITATION</b>  |
|   | dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup><br>Oral (Rat) LD50; >2000 mg/kg <sup>[1]</sup>  | Eye (rabbit): 20 mg - mild<br>Eye: adverse effect observed (irritating) <sup>[1]</sup><br>Skin (rabbit): 660 mg - moderate<br>Skin: no adverse effect observed (not irritating) <sup>[1]</sup> |
|   |   |  |
| nickel catalyst, finely divided, >= 40% water | <b>TOXICITY</b>   | <b>IRRITATION</b>  |
|   | Oral (Rat) LD50; 5000 mg/kg <sup>[2]</sup>  | Eye: no adverse effect observed (not irritating) <sup>[1]</sup><br>Skin: no adverse effect observed (not irritating) <sup>[1]</sup>  |
| lithium                                       | <b>TOXICITY</b>   | <b>IRRITATION</b>  |
|   | Not Available   | Eye: adverse effect observed (irritating) <sup>[1]</sup><br>Skin: adverse effect observed (corrosive) <sup>[1]</sup>   |
| <b>Legend:</b>                                | 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 |  |

|                         |  |
|-------------------------|--|
| <b>24V battery pack</b> | <p>Goitrogenic:</p> <p>Goitrogens are substances that suppress the function of the thyroid gland by interfering with iodine uptake, which can, as a result, cause an enlargement of the thyroid, i.e., a goitre</p> <p>Goitrogens include:</p> <ul style="list-style-type: none"> <li>▶ Vitexin, a flavanoid, which inhibits thyroid peroxidase thus contributing to goiter.</li> <li>▶ Ions such as thiocyanate and perchlorate which decrease iodide uptake by competitive inhibition; as a consequence of reduced thyroxine and triiodothyronine secretion by the gland, at low doses, this causes an increased release of thyrotropin (by reduced negative feedback), which then stimulates the gland.</li> <li>▶ Lithium which inhibits thyroid hormone release.</li> <li>▶ Certain foods, such as soy and millet (containing vitexins) and vegetables in the genus Brassica (e.g. broccoli, brussels sprouts, cabbage, horseradish).</li> <li>▶ Caffeine (in coffee, tea, cola, chocolate) which acts on thyroid function as a suppressant.</li> </ul> <p>The material may trigger oculo-gyric crisis. The term "oculo-gyric" refers to the bilateral elevation of the visual gaze. Initial symptoms include restlessness, agitation, malaise, or a fixed stare. Then comes the more characteristically described extreme and sustained upward deviation of the eyes. In addition, the eyes may converge, deviate upward and laterally, or deviate downward. The most frequently reported associated findings are backwards and lateral flexion of the neck, widely opened mouth, tongue protrusion, and ocular pain. However, the condition may also be associated with intensely painful jaw spasm which may result in the breaking of a tooth. A wave of exhaustion may follow an episode. The abrupt termination of the psychiatric symptoms at the conclusion of the crisis is most striking.</p> <p>Other features that are noted during attacks include mutism, palilalia, eye blinking, lacrimation, pupil dilation, drooling, respiratory dyskinesia, increased blood pressure and heart rate, facial flushing, headache, vertigo, anxiety, agitation, compulsive thinking, paranoia, depression, recurrent fixed ideas, depersonalization, violence, and obscene language.</p> |
|-------------------------|--|

## 24V battery pack

|   |   |
|---|---|
|   | <p>In addition to the acute presentation, oculogyric crisis can develop as a recurrent syndrome, triggered by stress and by exposure to the drugs.</p> <p>The diagnosis of oculogyric crisis is largely clinical and involves taking a focused history and physical examination to identify possible triggers for the crisis and rule out other causes of abnormal ocular movements.</p>  |
| <p><b>CARBON,<br/>NON-ACTIVATED</b></p> | <p>Substance has been investigated as a reproductive effector.</p>  |
| <p><b>COPPER</b></p>                    | <p>WARNING: Inhalation of high concentrations of copper fume may cause "metal fume fever", an acute industrial disease of short duration. Symptoms are tiredness, influenza like respiratory tract irritation with fever.</p> <p>for copper and its compounds (typically copper chloride):</p> <p><b>Acute toxicity:</b> There are no reliable acute oral toxicity results available. In an acute dermal toxicity study (OECD TG 402), one group of 5 male rats and 5 groups of 5 female rats received doses of 1000, 1500 and 2000 mg/kg bw via dermal application for 24 hours. The LD50 values of copper monochloride were 2,000 mg/kg bw or greater for male (no deaths observed) and 1,224 mg/kg bw for female. Four females died at both 1500 and 2000 mg/kg bw, and one at 1,000 mg/kg bw. Symptom of the hardness of skin, an exudation of hardness site, the formation of scar and reddish changes were observed on application sites in all treated animals. Skin inflammation and injury were also noted. In addition, a reddish or black urine was observed in females at 2,000, 1,500 and 1,000 mg/kg bw. Female rats appeared to be more sensitive than male based on mortality and clinical signs.</p> <p>No reliable skin/eye irritation studies were available. The acute dermal study with copper monochloride suggests that it has a potential to cause skin irritation.</p> <p><b>Repeat dose toxicity:</b> In repeated dose toxicity study performed according to OECD TG 422, copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39 - 51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL value was 5 and 1.3 mg/kg bw/day for male and female rats, respectively. No deaths were observed in male rats. One treatment-related death was observed in female rats in the high dose group. Erythropoietic toxicity (anaemia) was seen in both sexes at the 80 mg/kg bw/day. The frequency of squamous cell hyperplasia of the forestomach was increased in a dose-dependent manner in male and female rats at all treatment groups, and was statistically significant in males at doses of =20 mg/kg bw/day and in females at doses of =5 mg/kg bw/day doses. The observed effects are considered to be local, non-systemic effect on the forestomach which result from oral (gavage) administration of copper monochloride.</p> <p><b>Genotoxicity:</b> An in vitro genotoxicity study with copper monochloride showed negative results in a bacterial reverse mutation test with Salmonella typhimurium strains (TA 98, TA 100, TA 1535, and TA 1537) with and without S9 mix at concentrations of up to 1,000 ug/plate. An in vitro test for chromosome aberration in Chinese hamster lung (CHL) cells showed that copper monochloride induced structural and numerical aberrations at the concentration of 50, 70 and 100 ug/mL without S9 mix. In the presence of the metabolic activation system, significant increases of structural aberrations were observed at 50 and 70 ug/mL and significant increases of numerical aberrations were observed at 70 ug/mL. In an in vivo mammalian erythrocyte micronucleus assay, all animals dosed (15 - 60 mg/kg bw) with copper monochloride exhibited similar PCE/(PCE+NCE) ratios and MNPCE frequencies compared to those of the negative control animals. Therefore copper monochloride is not an in vivo mutagen.</p> <p><b>Carcinogenicity:</b> there was insufficient information to evaluate the carcinogenic activity of copper monochloride.</p> <p>Reproductive and developmental toxicity: In the combined repeated dose toxicity study with the reproduction/developmental toxicity screening test (OECD TG 422), copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39-51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL of copper monochloride for fertility toxicity was 80 mg/kg bw/day for the parental animals. No treatment-related effects were observed on the reproductive organs and the fertility parameters assessed. For developmental toxicity the NOAEL was 20 mg/kg bw/day. Three of 120 pups appeared to have icterus at birth; 4 of 120 pups appeared runted at the highest dose tested (80 mg/kg bw/day).</p> |
| <p><b>ETHYLENE CARBONATE</b></p>        | <p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>for ethylene carbonate</p> <p><b>Mammalian toxicity:</b> Reliable acute toxicity tests are available on ethylene carbonate. Ethylene carbonate is practically nontoxic following acute oral exposure in a test that meets OECD and EPA test guidelines; the LD50 is &gt;5000 mg/kg. The dermal LD50 is &gt;2000 mg/kg, in a test that meets OECD and EPA test guidelines.</p> <p>Ethylene carbonate is rapidly metabolized to ethylene glycol. Following gavage administration to rats, ethylene carbonate is rapidly converted into ethylene glycol; the half-life for disappearance of ethylene carbonate from blood was 0.25 hours. As a result, the mammalian toxicity of ethylene carbonate is nearly identical to that of ethylene glycol for endpoints where both have been tested</p> <p>Ethylene carbonate was mixed in the diet of 26 male and 26 female Crl: CD(SD) rats for 18 months at concentrations of 25,000 ppm for males and females and 50,000 ppm for females; males were also fed 50,000 ppm for 42 weeks, and 40,000 ppm for 16 weeks. Survivors were observed to 24 months. Compound intake (mg/kg/day) was not reported, but is estimated to be approximately 250 and 500 mg/kg/day. No toxic effects were found in females, but increased mortality was seen in males at both dose levels. No high-dose males survived week 60 and only 10 low-dose males survived to week 78. Males had severe nephrotoxicity, characteristic of ethylene glycol toxicity.</p> <p>The following <i>in vitro</i> genotoxicity tests were conducted on ethylene carbonate, without indications of genotoxicity: an Ames mutagenicity assay, an unscheduled DNA synthesis assay using rat hepatocytes, and a cell transformation assay using BALB/3T3 cells. No <i>in vivo</i> genotoxicity studies on ethylene carbonate were found; however, ethylene glycol has been tested and was negative in a rat dominant lethal assay.</p> <p>Gavage administration of ethylene carbonate to pregnant rats days 6-15 of gestation resulted in systemic toxicity at doses of 3000 mg/kg/day, including post-dose salivation. The NOAEL for maternal toxicity was 1500 mg/kg/day. Similar to ethylene glycol, there were increased soft tissue (hydrocephalus, umbilical herniation, gastroschisis, cleft palate, misshapen and compressed stomach) and skeletal malformations at 3000 mg/kg/day, but not at 1500 mg/kg/day.</p> <p>For ethylene glycol:</p>  |

## 24V battery pack

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

dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO<sub>2</sub>, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO<sub>2</sub>, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

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

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

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

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

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

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

**Metabolic Effects.** One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate).

**Neurological Effects:** Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion. Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months.

**Reproductive Effects:** Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration.

**Developmental Effects:** The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embryotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight.

**Cancer:** No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol.

## 24V battery pack

|  |   |
|--|---|
|  | <b>Genotoxic Effects:</b> Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available <i>in vivo</i> and <i>in vitro</i> laboratory studies provide consistently negative genotoxicity results for ethylene glycol.   |
| <b>NICKEL CATALYST, FINELY DIVIDED, &gt;= 40% WATER</b>  | <b>WARNING:</b> This substance has been classified by the IARC as Group 2A: Probably Carcinogenic to Humans.<br>Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen<br>[National Toxicology Program: U.S. Dep. of Health & Human Services 2002]  |
| <b>24V battery pack &amp; GRAPHITE &amp; ETHYLENE CARBONATE &amp; LITHIUM</b>  | 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 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. |
| <b>24V battery pack &amp; COBALT LITHIUM MANGANESE NICKELATE &amp; COPPER &amp; NICKEL CATALYST, FINELY DIVIDED, &gt;= 40% WATER</b> | The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.  |
| <b>COBALT LITHIUM MANGANESE NICKELATE &amp; GRAPHITE &amp; ALUMINIUM &amp; LITHIUM</b>   | No significant acute toxicological data identified in literature search.  |

|  |   |                                 |   |
|--|---|---------------------------------|---|
| <b>Acute Toxicity</b>                    | ✓ | <b>Carcinogenicity</b>          | ✓ |
| <b>Skin Irritation/Corrosion</b>         | ✓ | <b>Reproductivity</b>           | ✗ |
| <b>Serious Eye Damage/Irritation</b>     | ✓ | <b>STOT - Single Exposure</b>   | ✗ |
| <b>Respiratory or Skin sensitisation</b> | ✓ | <b>STOT - Repeated Exposure</b> | ✓ |
| <b>Mutagenicity</b>                      | ✓ | <b>Aspiration Hazard</b>        | ✗ |

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

## SECTION 12 Ecological information

## Toxicity

|   | Endpoint                | Test Duration (hr)            | Species                       | Value           | Source        |
|---|-------------------------|-------------------------------|-------------------------------|-----------------|---------------|
|   | <b>24V battery pack</b> | Not Available                 | Not Available                 | Not Available   | Not Available |
| <b>cobalt lithium manganese nickelate</b> | EC50                    | 72h                           | Algae or other aquatic plants | >1mg/l          | 2             |
|   | NOEC(ECx)               | 504h                          | Crustacea                     | >0.1<=1mg/l     | 2             |
| <b>carbon, non-activated</b>              | Endpoint                | Test Duration (hr)            | Species                       | Value           | Source        |
|   | NOEC(ECx)               | 72h                           | Algae or other aquatic plants | 50mg/L          | 4             |
| <b>copper</b>                             | Endpoint                | Test Duration (hr)            | Species                       | Value           | Source        |
|   | EC50(ECx)               | 24h                           | Algae or other aquatic plants | <0.001mg/L      | 4             |
|   | EC50                    | 72h                           | Algae or other aquatic plants | 0.011-0.017mg/L | 4             |
|   | EC50                    | 48h                           | Crustacea                     | <0.001mg/L      | 4             |
|   | LC50                    | 96h                           | Fish                          | 0.005-0.06mg/l  | 4             |
| EC50                                      | 96h                     | Algae or other aquatic plants | 0.03-0.058mg/l                | 4               |               |

## 24V battery pack

|   |   |                           |                               |                 |               |
|---|---|---------------------------|-------------------------------|-----------------|---------------|
| graphite                                      | <b>Endpoint</b>   | <b>Test Duration (hr)</b> | <b>Species</b>                | <b>Value</b>    | <b>Source</b> |
|   | NOEC(ECx)   | 72h                       | Algae or other aquatic plants | >=100mg/l       | 2             |
|   | EC50  | 72h                       | Algae or other aquatic plants | >100mg/l        | 2             |
|   | EC50  | 48h                       | Crustacea                     | >100mg/l        | 2             |
| aluminium                                     | LC50  | 96h                       | Fish                          | >100mg/l        | 2             |
|   | <b>Endpoint</b>   | <b>Test Duration (hr)</b> | <b>Species</b>                | <b>Value</b>    | <b>Source</b> |
|   | NOEC(ECx)   | 48h                       | Crustacea                     | >100mg/l        | 1             |
|   | EC50  | 72h                       | Algae or other aquatic plants | 0.2mg/l         | 2             |
|   | EC50  | 48h                       | Crustacea                     | 1.5mg/l         | 2             |
| ethylene carbonate                            | LC50  | 96h                       | Fish                          | 0.078-0.108mg/l | 2             |
|   | EC50  | 96h                       | Algae or other aquatic plants | 0.024mg/l       | 2             |
|   | <b>Endpoint</b>   | <b>Test Duration (hr)</b> | <b>Species</b>                | <b>Value</b>    | <b>Source</b> |
|   | NOEC(ECx)   | 72h                       | Algae or other aquatic plants | 100mg/l         | 2             |
| nickel catalyst, finely divided, >= 40% water | EC50  | 72h                       | Algae or other aquatic plants | >100mg/l        | 2             |
|   | EC50  | 48h                       | Crustacea                     | >100mg/l        | 2             |
|   | LC50  | 96h                       | Fish                          | >100mg/l        | 2             |
|   | EC50(ECx)   | 72h                       | Algae or other aquatic plants | 0.18mg/l        | 1             |
|   | EC50  | 72h                       | Algae or other aquatic plants | 0.18mg/l        | 1             |
| lithium                                       | EC50  | 48h                       | Crustacea                     | >100mg/l        | 1             |
|   | LC50  | 96h                       | Fish                          | 0.168mg/L       | 4             |
|   | EC50  | 96h                       | Algae or other aquatic plants | 0.36mg/l        | 2             |
|   | NOEC(ECx)   | 72h                       | Algae or other aquatic plants | 1.65mg/l        | 2             |
| Legend:                                       | EC50  | 72h                       | Algae or other aquatic plants | 25.6mg/l        | 2             |
|   | EC50  | 48h                       | Crustacea                     | 19.1mg/l        | 2             |
|   | LC50  | 96h                       | Fish                          | 18mg/l          | 2             |
|   | <p><b>Legend:</b> <i>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</i></p> |                           |                               |                 |               |

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

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 copper:

Atmospheric Fate - Copper is unlikely to accumulate in the atmosphere due to a short residence time for airborne copper aerosols. Airborne coppers, however, may be transported over large distances. Air Quality Standards: no data available.

Aquatic Fate: Toxicity of copper is affected by pH and hardness of water. Total copper is rarely useful as a predictor of toxicity. In natural sea water, more than 98% of copper is organically bound and in river waters a high percentage is often organically bound, but the actual percentage depends on the river water and its pH.

Ecotoxicity: Copper accumulates significantly in the food chain. The toxic effect of copper in the aquatic biota depends on the bio-availability of copper in water which, in turn, depends on its physico-chemical form (i.e. speciation). Bioavailability is decreased by complexation and adsorption of copper by natural organic matter, iron and manganese hydrated oxides, and chelating agents excreted by algae and other aquatic organisms. Copper exhibits significant toxicity in some aquatic organisms. Some algal species are very sensitive to copper. Silicate, iron, manganese and EDTA may reduce bioavailability.



## 24V battery pack

For copper: Ecotoxicity - Significant effects are expected on various species of microalgae, some species of macroalgae, and a range of invertebrates, including crustaceans, gastropods and sea urchins. Copper is moderately toxic to crab and their larvae and is highly toxic to gastropods (mollusks, including oysters, mussels and clams). In fish, the acute lethal concentrations of copper depends both on test species and exposure conditions. Waters with high concentrations of copper can have significant effects on diatoms and sensitive invertebrates, notably cladocerans (water fleas). Most taxonomic groups of macroalgae and invertebrates will be severely affected.

For Copper: Typical foliar levels of copper are: Uncontaminated soils (0.3-250 mg/kg) ; Contaminated soils (150-450 mg/kg) ; Mining/smelting soils (6.1-25 mg/kg) 80 mg/kg 300 mg/kg).

Terrestrial Fate: Plants - Generally, vegetation reflects soil copper levels in its foliage. This is dependent upon the bioavailability of copper and the physiological requirements of species concerned. Crops are often more sensitive to copper than the native flora. Soil: In soil, copper levels are raised by application of fertilizer, fungicides, from deposition of highway dusts and from urban, mining and industrial sources. Chronic and or acute effects on sensitive species occur as a result of human activities such as copper fertilizer addition and addition of sludge. When soil levels exceed 150 mg Cu/kg, native and agricultural species show chronic effects. Soils in the range 500-1000 mg Cu/kg act in a strongly selective fashion allowing the survival of only copper-tolerant species and strains. At 2000 Cu mg/kg, most species cannot survive. By 3500 mg Cu/kg, areas are largely devoid of vegetation cover. The organic content of the soil appears to be a key factor affecting the bioavailability of copper. On normal forest soils, non-rooted plants such as mosses and lichens show higher copper concentrations. The fruiting bodies and mycorrhizal sheaths of soil fungi associated with higher plants in forests often accumulate copper to much higher levels than plants at the same site. 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 adsorb. 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 (CO<sub>3</sub><sup>2-</sup>), and the concentration of manganese is limited by the relatively low solubility (65 mg/L) of MnCO<sub>3</sub>. 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

Transport and distribution of nickel particulates between different environmental compartments, is strongly influenced by particle size. Fine particulate matter has a longer residence time in the environment and is carried a long distance from its source; larger particles are deposited near the emission source. Atmospheric residence time for nickel particulates is estimated to be 5.4-7.9 days. Water solubility and bioavailability is affected by soil pH; decrease in pH generally mobilises nickel, thus acid rain can mobilise nickel from the soil and increase nickel concentrations in ground water. Nickel bioaccumulates in the food chain but is not bioconcentrated.

**Drinking Water Standards:**

Nickel 50 ug/l (UK max.)

20 ug/l (WHO guideline)

**Soil Guidelines:**

Dutch Criteria: 35 mg/kg (target)

**24V battery pack**

210 mg/kg (intervention)

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 O<sub>2</sub>, 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* larvis embryo gives rise to duplication of dorsoanterior structures such as the notochord, neural tube and eyes.

for cobalt compounds:

**Environmental Fate:**

Cobalt strongly binds to humic substances naturally present in aquatic environments. Humic acids can be modified by UV light and bacterial decomposition, which may change their binding characteristics over time. The lability of the complexes is strongly influenced by pH, the nature of the humic material, and the metal-to-humic substance ratio. The lability of cobalt-humate complexes decreases in time ("aging effect"). The "aging effect" indicates that after a period of time (~12 hours), complexes that were initially formed are transformed into stronger ones from which the metal ion is less readily dislodged.

Between 45 and 100% of dissolved cobalt was found to occur in very strong complexes. The distribution coefficient of cobalt may vary considerably in the same sediment in response to conditions affecting the pH, redox conditions, ionic strength, and amount of dissolved organic matter. Uptake of <sup>60</sup>Co from the water by sediment increased rapidly as the pH was increased from 5 to 7-7.5 and then slightly decrease. Therefore, pH would be an important factor affecting the migration of cobalt in surface water. Uptake was little affected by changes in liquid-to-solids ratio and ionic strength. <sup>60</sup>Co is more mobile in anaerobic marine aquatic environments than in freshwater aerobic ones. In seawater sediment systems under anaerobic conditions <sup>60</sup>Co was 250 times more mobile than <sup>60</sup>Co in freshwater sediment systems under aerobic conditions. Under anaerobic conditions, 30% of the <sup>60</sup>Co added to a sediment-freshwater system was "exchangeable" and therefore potentially mobile, while under aerobic conditions, 98% of the <sup>60</sup>Co was permanently fixed. Most of the mobile <sup>60</sup>Co produced under anaerobic conditions in seawater consisted of nonionic cobalt associated with low molecular weight organic substances that were stable to changes in pH; the exchangeable <sup>60</sup>Co appeared to be mostly ionic.

The mobility of cobalt in soil is inversely related to how strongly it is adsorbed by soil constituents. Cobalt may be retained by mineral oxides such as iron and manganese oxide, crystalline materials such as aluminosilicate and goethite, and natural organic substances in soil. Sorption of cobalt to soil occurs rapidly (within 1-2 hours). Soil-derived oxide materials were found to adsorb greater amounts of cobalt than other materials examined, although substantial amounts were also adsorbed by organic materials.

Clay minerals sorbed relatively smaller amounts of cobalt. In addition, little cobalt was desorbed from soil oxides while substantial amounts desorbed from humic acids and montmorillonite. In clay soil, adsorption may be due to ion exchange at the cationic sites on clay with either simple ionic cobalt or hydrolysed ionic species such as CoOH<sup>+</sup>. Adsorption of cobalt onto iron and manganese increases with pH. In addition, as pH increases, insoluble hydroxides or carbonates may form, which would also reduce cobalt mobility. Conversely, sorption onto mobile colloids would enhance its mobility. In most soils, cobalt is more mobile than lead, chromium (II), zinc, and nickel, but less mobile than cadmium. In several studies, the K<sub>d</sub> of cobalt in a variety of soils ranged from 0.2 to 3,800. The soil properties showing the highest correlation with K<sub>d</sub> were exchangeable calcium, pH, water content, and cation exchange capacity. Organic complexing agents such as ethylenediaminetetraacetic acid (EDTA), which are used for decontamination operations at nuclear facilities, greatly enhance the mobility of cobalt in soil. Other organic complexing agents, such as those obtained from plant decay, may also increase cobalt mobility in soil. However, both types of complexes decrease cobalt uptake by plants. Addition of sewage sludge to soil also increases the mobility of cobalt, perhaps due to organic complexation of cobalt.

Cobalt may be taken up from soil by plants. Surface deposition of cobalt on leaves of plants from airborne particles may also occur. Elevated levels of cobalt have been found in the roots of sugar beets and potato tubers in soils with high cobalt concentrations (e.g., fly ash-amended soil) due to absorption of cobalt from soil. However, the translocation of cobalt from roots to above-ground parts of plants is not significant in most soils, as indicated by the lack of cobalt in seeds of barley, oats, and wheat grown in high-cobalt soil. However, in highly acidic soil (pH as low as 3.3), significantly higher than normal concentrations of cobalt were found in rye grass foliage, oats, and barley. For example, cobalt concentrations in rye grass grown in unlimed soil (pH<5.0) was 19.7 mg/kg compared with 1.1 mg/kg in rye grass grown in limed soil (pH>5.0). Soil and plant samples taken in the 30-km zone around Chernobyl indicated that <sup>60</sup>Co was not accumulated by plants and mushrooms. Studies investigating the uptake of <sup>60</sup>Co by tomato plants watered with <sup>60</sup>Co contaminated water showed that tomato plants absorbed <2% of the activity available from the soil.

<sup>60</sup>Co is taken up by phytoplankton and unicellular algae (*Senenastrum capricornutum*) with concentration factors (dry weight) ranging from 15,000 to 40,000 and 2,300 to 18,000, respectively. Elimination experiments with the algae indicate a two component biological half-life, 1 hour and 11 days, respectively, and suggest that the cobalt might be absorbed not only on the surface, but also intracellularly. Since these organisms are at the bottom of the food chain, they could play an important role in the trophic transfer of <sup>60</sup>Co released into waterways by nuclear facilities. However, cobalt levels generally diminish with increasing trophic levels in a food chain. The low levels of cobalt in fish may also reflect cobalt's strong binding to particles and sediment. The bioaccumulation factors (dry weight basis) for cobalt in marine and freshwater fish are ~100-4,000 and <10-1,000, respectively; accumulation in the muscle of marine fish is 5- 500.

Cobalt largely accumulates in the viscera and on the skin, as opposed to the edible parts of the fish. In carp, accumulation from water accounted for 75% of <sup>60</sup>Co accumulated from both water and food; accumulation from water and food was additive. Depuration half-lives were 53 and 87 days for fish contaminated from food and water, respectively. In the case of an accidental release of <sup>60</sup>Co into waterways, the implication is that effects would manifest themselves rapidly since the primary route of exposure is from water rather than food. Uptake of <sup>60</sup>Co was very low in whitefish, with concentrations being highest in kidney and undetectable in muscle. Similarly, while accumulation of <sup>60</sup>Co by carp from food was dependent on food type, the transfer factor was very low, approximately 0.01, and no long-term bioaccumulation of the radionuclide occurred.

Concentration factors have also been reported for various other aquatic organisms. Freshwater mollusks have concentration factors of 100-14,000 (~1-300 in soft tissue). Much of the cobalt taken up by mollusks and crustaceans from water or sediment is adsorbed to the shell or exoskeleton; very little cobalt is generally accumulated in the edible parts. A concentration factor for <sup>60</sup>Co of 265 mL/g (wet weight) was determined for *Daphnia magna* in laboratory studies. The rapid decrease in radioactivity during the depuration phase indicated that adsorption to the surface was the major contamination process. However, the digestive glands of crustaceans, which are sometimes eaten by humans, may accumulate high levels of <sup>60</sup>Co. The shell accounted for more than half of the body burden. Among the soft tissue, the gills and viscera had the highest concentrations factors and the muscle had the lowest.

In mussels, higher absorption efficiencies and lower efflux rates were obtained for cobalamins than for inorganic cobalt, suggesting that it is a more bioavailable form of cobalt.

Vitamin B12, which contains cobalt, is synthesized by 58 species of seven genera of bacteria as well as blue-green algae and actinomycetes (mold-like bacteria). Consequently, vitamin B12 levels in marine water range from very low levels in some open ocean water to much higher levels in some coastal waters. Freshwater environments have comparable levels of vitamin B12. The high level of cobalamins in coastal water appears to be related to the occurrence of macrophytes in these areas with their high concentrations of vitamin B12. Cobalamins are released into the water when the organisms die.

Some female birds sequester metals into their eggs under certain conditions, a phenomenon that may jeopardize the developing embryos.

**DO NOT** discharge into sewer or waterways.

### Persistence and degradability

| Ingredient         | Persistence: Water/Soil | Persistence: Air |
|--------------------|-------------------------|------------------|
| ethylene carbonate | HIGH                    | HIGH             |

### Bioaccumulative potential

| Ingredient         | Bioaccumulation        |
|--------------------|------------------------|
| ethylene carbonate | LOW (LogKOW = -0.3388) |

### Mobility in soil

| Ingredient         | Mobility          |
|--------------------|-------------------|
| ethylene carbonate | LOW (KOC = 9.168) |



## SECTION 13 Disposal considerations

### Waste treatment methods

|                                     |   |
|-------------------------------------|---|
| <b>Product / Packaging disposal</b> | <ul style="list-style-type: none"> <li>▸ Containers may still present a chemical hazard/ danger when empty.</li> <li>▸ Return to supplier for reuse/ recycling if possible.</li> </ul> <p>Otherwise:</p> <ul style="list-style-type: none"> <li>▸ 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.</li> <li>▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product.</li> <li>▸ <b>DO NOT allow wash water from cleaning or process equipment to enter drains.</b></li> <li>▸ It may be necessary to collect all wash water for treatment before disposal.</li> <li>▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.</li> <li>▸ Where in doubt contact the responsible authority.</li> </ul> |
|-------------------------------------|---|

## SECTION 14 Transport information

### Labels Required

|                         |   |
|-------------------------|---|
|                         |  |
| <b>Marine Pollutant</b> |  |
| <b>HAZCHEM</b>          | 2Y  |

### Land transport (ADG)

|                                     |   |                                     |
|-------------------------------------|---|-------------------------------------|
| <b>UN number</b>                    | 3480  |                                     |
| <b>UN proper shipping name</b>      | LITHIUM ION BATTERIES (including lithium ion polymer batteries) |                                     |
| <b>Transport hazard class(es)</b>   | Class   | 9                                   |
|                                     | Subrisk   | Not Applicable                      |
| <b>Packing group</b>                | Not Applicable  |                                     |
| <b>Environmental hazard</b>         | Environmentally hazardous                                       |                                     |
| <b>Special precautions for user</b> | Special provisions  | 188 230 310 348 376 377 384 387 390 |

## 24V battery pack

|  |                  |   |
|--|------------------|---|
|  | Limited quantity | 0 |
|--|------------------|---|

## Air transport (ICAO-IATA / DGR)

|                                     |   |  |
|-------------------------------------|---|--|
| <b>UN number</b>                    | 3480  |  |
| <b>UN proper shipping name</b>      | Lithium ion batteries (including lithium ion polymer batteries) |  |
| <b>Transport hazard class(es)</b>   | ICAO/IATA Class   | 9  |
|                                     | ICAO / IATA Subrisk   | Not Applicable                                       |
|                                     | ERG Code  | 12FZ   |
| <b>Packing group</b>                | Not Applicable  |  |
| <b>Environmental hazard</b>         | Environmentally hazardous                                       |  |
| <b>Special precautions for user</b> | Special provisions  | A88 A99 A154 A164 A183 A201 A206 A213 A331 A334 A802 |
|                                     | Cargo Only Packing Instructions                                 | See 965  |
|                                     | Cargo Only Maximum Qty / Pack                                   | See 965  |
|                                     | 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)

|                                     |   |                                 |
|-------------------------------------|---|---------------------------------|
| <b>UN number</b>                    | 3480  |                                 |
| <b>UN proper shipping name</b>      | LITHIUM ION BATTERIES (including lithium ion polymer batteries) |                                 |
| <b>Transport hazard class(es)</b>   | IMDG Class  | 9                               |
|                                     | IMDG Subrisk  | Not Applicable                  |
| <b>Packing group</b>                | Not Applicable  |                                 |
| <b>Environmental hazard</b>         | Marine Pollutant  |                                 |
| <b>Special precautions for user</b> | EMS Number  | F-A, S-I                        |
|                                     | Special provisions  | 188 230 310 348 376 377 384 387 |
|                                     | Limited Quantities  | 0                               |

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

Not Applicable

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

| Product name                                  | Group         |
|---|---------------|
| cobalt lithium manganese nickelate            | Not Available |
| carbon, non-activated                         | Not Available |
| copper  | Not Available |
| graphite                                      | Not Available |
| aluminium                                     | Not Available |
| ethylene carbonate                            | Not Available |
| nickel catalyst, finely divided, >= 40% water | Not Available |
| lithium                                       | Not Available |

## Transport in bulk in accordance with the ICG Code

| Product name                       | Ship Type     |
|------------------------------------|---------------|
| cobalt lithium manganese nickelate | Not Available |

## 24V battery pack

| Product name                                  | Ship Type     |
|---|---------------|
| carbon, non-activated                         | Not Available |
| copper  | Not Available |
| graphite                                      | Not Available |
| aluminium                                     | Not Available |
| ethylene carbonate                            | Not Available |
| nickel catalyst, finely divided, >= 40% water | Not Available |
| lithium                                       | Not Available |

## SECTION 15 Regulatory information

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

**cobalt lithium manganese nickelate 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 1: Carcinogenic to humans  
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)**carbon, non-activated is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

**copper is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

Australian Inventory of Industrial Chemicals (AIIC)

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

**graphite is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

**aluminium is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

**ethylene carbonate is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

**nickel catalyst, finely divided, >= 40% water is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

**lithium is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

Australian Inventory of Industrial Chemicals (AIIC)

FEI Equine Prohibited Substances List - Banned Substances

FEI Equine Prohibited Substances List (EPLS)

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

## National Inventory Status

## 24V battery pack

| National Inventory                               | Status  |
|--|---|
| Australia - AIIIC / Australia Non-Industrial Use | No (cobalt lithium manganese nickelate)   |
| Canada - DSL                                     | No (cobalt lithium manganese nickelate)   |
| Canada - NDSL                                    | No (cobalt lithium manganese nickelate; carbon, non-activated; copper; graphite; aluminium; ethylene carbonate; nickel catalyst, finely divided, >= 40% water; lithium)                           |
| China - IECSC                                    | Yes   |
| Europe - EINEC / ELINCS / NLP                    | No (cobalt lithium manganese nickelate)   |
| Japan - ENCS                                     | No (cobalt lithium manganese nickelate; carbon, non-activated; copper; graphite; aluminium; nickel catalyst, finely divided, >= 40% water; lithium)   |
| Korea - KECI                                     | No (cobalt lithium manganese nickelate)   |
| New Zealand - NZIoC                              | No (cobalt lithium manganese nickelate)   |
| Philippines - PICCS                              | No (cobalt lithium manganese nickelate)   |
| USA - TSCA                                       | Yes   |
| Taiwan - TCSI                                    | Yes   |
| Mexico - INSQ                                    | No (cobalt lithium manganese nickelate; ethylene carbonate)   |
| Vietnam - NCI                                    | Yes   |
| Russia - FBEPH                                   | No (cobalt lithium manganese nickelate)   |
| <b>Legend:</b>                                   | Yes = All CAS declared ingredients are on the inventory<br>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 | 30/09/2022 |
| Initial Date  | 30/09/2022 |

## Other information

## Ingredients with multiple cas numbers

| Name                  | CAS No  |
|-----------------------|---|
| carbon, non-activated | 7440-44-0, 82600-58-6   |
| copper                | 7440-50-8, 133353-46-5, 133353-47-6, 195161-80-9, 65555-90-0, 72514-83-1, 1441640-38-5, 1993435-25-8, 2056901-56-3  |
| graphite              | 7782-42-5, 115344-49-5, 1215114-94-5, 12424-49-6, 12751-41-6, 1397692-45-3, 1399-57-1, 155660-93-8, 156854-02-3, 159251-18-0, 164973-65-3, 1811526-35-8, 182761-22-4, 2093098-71-4, 2179292-22-7, 2183464-49-3, 37265-44-4, 37265-48-8, 50814-81-8, 72840-52-9, 82696-74-0, 82696-75-1, 82701-02-8, 82701-03-9, 82701-04-0, 82701-05-1, 82701-06-2, 82709-42-0, 83797-07-3, 84739-05-9, 857167-12-5, 87934-03-0 |
| aluminium             | 7429-90-5, 91728-14-2   |

Classification of the preparation and its individual components has drawn on official and authoritative sources 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

**24V battery pack**

BEI: Biological Exposure Index  
AIIIC: 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