



2000LM RECHARGEABLE SHOP LIGHT

Apex Tool Group

Part Number: **GWSL2000AU**

Version No: **2.3**

Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Initial Date: **25/09/2025**

Revision Date: **25/09/2025**

Print Date: **25/09/2025**

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

Product Identifier

Product name	2000LM RECHARGEABLE SHOP LIGHT
Synonyms	Not Available
Proper shipping name	LITHIUM ION BATTERIES PACKED WITH EQUIPMENT (including lithium ion polymer batteries); LITHIUM ION BATTERIES CONTAINED IN EQUIPMENT (including lithium ion polymer batteries)
Other means of identification	GWSL2000AU

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

Relevant identified uses	Not Available
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Details of the manufacturer or importer of the safety data sheet

Registered company name	Apex Tool Group
Address	Suite 201, 184 Bourke Road Alexandria New South Wales 2015 Australia
Telephone	02 6021 6666
Fax	Not Available
Website	www.gearwrench.com.au
Email	salesaus@apextoolgroup.com

Emergency telephone number

Association / Organisation	Poisons Information Hotline
Emergency telephone number(s)	131126
Other emergency telephone number(s)	000

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]	Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Carcinogenicity Category 1A, Specific Target Organ Toxicity - Repeated Exposure 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)	
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Signal word	Danger
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Hazard statement(s)

H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H318	Causes serious eye damage.
H335	May cause respiratory irritation.
H350	May cause cancer.
H373	May cause damage to organs through prolonged or repeated exposure.
AUH019	May form explosive peroxides.

Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P202	Do not handle until all safety precautions have been read and understood.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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No further product hazard information.

SECTION 3 Composition / information on ingredients**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
346417-97-8	36-40	<u>lithium nickel manganese cobalt oxide</u>
7782-42-5	20-26	<u>graphite</u>
24937-79-9	2	<u>vinylidene fluoride homopolymer</u>
96-49-1	15-20	<u>ethylene carbonate</u>
21324-40-3	1-4	<u>lithium fluorophosphate</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

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay.
Ingestion	<ul style="list-style-type: none"> ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

Clinical effects of lithium intoxication appear to relate to duration of exposure as well as to level.

- ▶ Lithium produces a generalised slowing of the electroencephalogram; the anion gap may increase in severe cases.
- ▶ Emesis (or lavage if the patient is obtunded or convulsing) is indicated for ingestions exceeding 40 mg (Li)/Kg.
- ▶ Overdose may delay absorption; decontamination measures may be more effective several hours after cathartics.
- ▶ Charcoal is not useful. No clinical data are available to guide the administration of 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 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

- Sand, dry powder extinguishers or other inerts should be used to smother dust fires.

Special hazards arising from the substrate or mixture

Fire Incompatibility	<ul style="list-style-type: none"> ► Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ► Alert Fire Brigade and tell them location and nature of hazard. ► Wear breathing apparatus plus protective gloves in the event of a fire. ► Prevent, by any means available, spillage from entering drains or water courses. ► Use fire fighting procedures suitable for surrounding area. ► DO NOT approach containers suspected to be hot. ► Cool fire exposed containers with water spray from a protected location. ► If safe to do so, remove containers from path of fire. ► Equipment should be thoroughly decontaminated after use. 												
Fire/Explosion Hazard	<p>carbon dioxide (CO₂) metal oxides other pyrolysis products typical of burning organic material. WARNING: Long standing in contact with air and light may result in the formation of potentially explosive peroxides. 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</p> <table border="1"> <tr> <td>Lower Limit for Explosion:</td> <td>50 g/m³ (carbon black in air)</td> </tr> <tr> <td>Maximum Explosion Pressure:</td> <td>10 bar</td> </tr> <tr> <td>Maximum Rate of Pressure Rise:</td> <td>30-100 bar/sec</td> </tr> <tr> <td>Minimum Ignition Temperature:</td> <td>315 deg. C.</td> </tr> <tr> <td>Ignition Energy:</td> <td>>1 kJ</td> </tr> <tr> <td>Glow Temperature:</td> <td>500 deg. C. (approx.)</td> </tr> </table> <p>Notes on Test Methods: Tests 1, 2 and 3 were conducted by Bergwerkschaffliche Versuchstrecke, Dortmund-Derne, using a 1 m³ 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 m³ 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)</p>	Lower Limit for Explosion:	50 g/m ³ (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.)
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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

Minor Spills	<ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid breathing vapours/ aerosols/ or dusts and avoid contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb spill with sand, earth, inert material or vermiculite. ▶ Place in a suitable, labelled container for waste disposal.
Major Spills	<ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Neutralise/decontaminate residue (see Section 13 for specific agent). ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage**Precautions for safe handling**

Safe handling	<p>NOTE:</p> <ul style="list-style-type: none"> ▶ 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. <p>The substance accumulates peroxides which may become hazardous only if it evaporates or is distilled or otherwise treated to concentrate the peroxides. The substance may concentrate around the container opening for example.</p> <p>Purchases of peroxidisable chemicals should be restricted to ensure that the chemical is used completely before it can become peroxidised.</p> <ul style="list-style-type: none"> ▶ A responsible person should maintain an inventory of peroxidisable chemicals or annotate the general chemical inventory to indicate which chemicals are subject to peroxidation. An expiration date should be determined. The chemical should either be treated to remove peroxides or disposed of before this date. ▶ The person or laboratory receiving the chemical should record a receipt date on the bottle. The individual opening the container should add an opening date. ▶ Unopened containers received from the supplier should be safe to store for 18 months. ▶ Opened containers should not be stored for more than 12 months. ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ DO NOT allow material to contact humans, exposed food or food utensils. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	<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"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

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Suitable container	<ul style="list-style-type: none"> Polyethylene or polypropylene container. Packing as recommended by manufacturer. Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<ul style="list-style-type: none"> WARNING: 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. The pi-complexes formed between chromium(0), vanadium(0) and other transition metals (haloarene-metal complexes) and mono- or poly-fluorobenzene show extreme sensitivity to heat and are explosive. Avoid reaction with borohydrides or cyanoborohydrides Avoid reaction with oxidising agents, bases and strong reducing agents. The unhindered oxygen atom found on cyclic ethers such as the epoxides, oxetanes, furans, dioxanes and pyrans, carries two unshared pairs of electrons - a structure which favors the formation of coordination complexes and the solvation of cations. Cyclic ethers are used as important solvents, as chemical intermediate and as monomers for ring-opening polymerization. They are unstable at room temperature due to possibility of peroxide formation; stabiliser is sometimes needed for storage and transportation. <p>NOTE: Ethers lacking non-methyl hydrogen atoms adjacent to the ether link are thought to be relatively safe</p> <p>For carbon powders:</p> <ul style="list-style-type: none"> 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 m²/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.



+ — 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	lithium nickel manganese cobalt oxide	Manganese, dust & compounds (as Mn)	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	graphite	Graphite (all forms except fibres) (respirable dust) (natural & synthetic)	3 mg/m3	Not Available	Not Available	(e) Containing no asbestos and < 1% crystalline silica.

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Ingredient	Original IDLH	Revised IDLH
lithium nickel manganese cobalt oxide	500 mg/m3 / 10 mg/m3	Not Available
graphite	1,250 mg/m3	Not Available
vinylidene fluoride homopolymer	Not Available	Not Available
ethylene carbonate	Not Available	Not Available
lithium fluorophosphate	Not Available	Not Available

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 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.

Exposure controls

Appropriate engineering controls	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.</p> <p>An approved self contained breathing apparatus (SCBA) may be required in some situations.</p> <p>Provide adequate ventilation in warehouse or closed storage area. 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>
Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyor transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyor loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)

Within each range the appropriate value depends on:

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

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2

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	<p>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>
Individual protection measures, such as personal protective equipment	    
Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].
Skin protection	See Hand protection below
Hands/feet protection	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber <p>NOTE:</p> <ul style="list-style-type: none"> ▶ 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. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit.

Respiratory protection

- 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**

Appearance	Not Available		
Physical state	Not Available	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available

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pH (as supplied)	Not Available	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Available
Flash point (°C)	Not Available	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Not Available	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Heat of Combustion (kJ/g)	Not Available	Ignition Distance (cm)	Not Available
Flame Height (cm)	Not Available	Flame Duration (s)	Not Available
Enclosed Space Ignition Time Equivalent (s/m³)	Not Available	Enclosed Space Ignition Deflagration Density (g/m³)	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

a) Acute Toxicity	Based on available data, the classification criteria are not met.
b) Skin Irritation/Corrosion	There is sufficient evidence to classify this material as skin corrosive or irritating.
c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	There is sufficient evidence to classify this material as sensitising to skin or the respiratory system
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	There is sufficient evidence to classify this material as carcinogenic
g) Reproductivity	Based on available data, the classification criteria are not met.
h) STOT - Single Exposure	There is sufficient evidence to classify this material as toxic to specific organs through single exposure
i) STOT - Repeated Exposure	There is sufficient evidence to classify this material as toxic to specific organs through repeated exposure
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	<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>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/m³ as cobalt hydrocarbonyl. Exposure to 9 mg cobalt/m³ 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/m³ (but not 1.9 mg cobalt/m³) as cobalt sulfate over 16 days, but exposure to 11.4 mg cobalt/m³ over 13 weeks was lethal only to mice and not to rats. Exposure to 1.14 mg cobalt/m³ as cobalt sulfate for 104 weeks resulted in no increase in mortality in rats and mice of either sex.</p>
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	<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/m³ as cobalt hydrocarbon) 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/m³) 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/m³ 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/m³ as cobalt dust for 3 months. Lifetime exposure of hamsters to 7.9 mg cobalt/m³ 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/m³ and mice exposed to 1.9 mg cobalt/m³ 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>
Ingestion	<p>Accidental ingestion of the material may be damaging to the health of the individual. Large doses of lithium ion have caused dizziness and prostration and can cause kidney damage if sodium intake is limited. Dehydration, weight-loss, dermatological effects and thyroid disturbances have been reported. Central nervous system effects that include slurred speech, blurred vision, sensory loss, impaired concentration, irritability, lethargy, confusion, disorientation, drowsiness, anxiety, spasticity, delirium, stupor, ataxia (loss of muscle coordination), sedation, fine and gross tremor, giddiness, twitching and convulsions may occur. Diarrhoea, vomiting and neuromuscular effects such as tremor, clonus (rapid contraction and relaxation of muscles) and hyperactive reflexes may occur as a result of repeated exposure to lithium. Acute severe overexposure may affect the kidneys, resulting in renal dysfunction, albuminuria, oliguria and degenerative changes. Cardiovascular effects may also result in cardiac arrhythmias and hypotension.</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. 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>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>
Skin Contact	<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. 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>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.</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>
Eye	<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</p>

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Chronic	<p>weeks. Some particles remained permanently producing a punctate purplish-black discolouration.</p> <p>On the basis of epidemiological data, it has been concluded that prolonged inhalation of the material, in an occupational setting, may produce cancer in humans.</p> <p>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.</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 than 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>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.</p> <p>The effects of chronic occupational exposure to cobalt and cobalt compounds on the respiratory system in humans are well-documented. These effects include respiratory irritation, diminished pulmonary function, wheezing, asthma, pneumonia, and fibrosis and occurred at exposure levels ranging from 0.007 to 0.893 mg cobalt/m³ (exposure from 2 to 17 years). These effects have been observed in workers employed in cobalt refineries, as well as hard metal workers, diamond polishers, and ceramic dish painters (painting with cobalt blue dye).</p> <p>Occupational asthma attributed to the inhalation of cobalt powder has been confirmed following bronchial challenge tests. Chest tightness and chronic bronchitis have been recorded in hard-metal workers exposed to cobalt. Cobalt is known to function as a hapten, resulting in the generation of antibodies against cobalt-protein complexes. Although the minimum exposure level associated with cobalt sensitisation has not been determined, sensitisation has been demonstrated in hard metal workers with work-related asthma who have experienced prolonged occupational exposure (>3 years) to levels ranging from 0.007 to 0.893 mg cobalt/m³. The sensitisation phenomenon includes the production of IgE and IgA antibodies to cobalt. Exposure to inhaled cobalt chloride aerosols can precipitate an asthmatic attack in sensitised individuals believed to be the result of an allergic reaction within the lungs.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>Animals, exposed to cobalt compounds also exhibit an increase in respiration, as well as tremor and convulsion. Exposure of rats and mice to aerosols of cobalt (as cobalt sulfate) at concentrations from 0.11 to 1.14 mg cobalt/m³ for 2 years 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 .011 mg cobalt/m³, with severity of the lesion increasing with increased cobalt concentration. Hyperplastic lesions of the nasal epithelium occurred in rats at concentrations of .011 mg cobalt/3, and in mice at concentrations of .038 mg cobalt/m³. Both sexes of rats had greatly increased incidences</p>
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(>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.

Cyclic ethers, including tetrahydrofuran, furan and 1,4-dioxane, produce neoplasms and carcinomas in experimental animals, typically of the liver; other target organs include the adrenal gland, nasal cavity and gall-bladder. 1,4-Dioxane was a promoter in a two-stage skin carcinogenic study in mice. Results of studies with cyclic ethers indicate that carcinogenicity is often species and sex dependent. Furan has been used to induce apoptosis (programmed cell death). Oxetanes are under investigation.

Neuromuscular effects result from chronic over-exposure to lithium compounds. These may include tremor, ataxia, clonus and hyperactive reflexes. Some animal studies have shown that exposure during pregnancy may produce birth defects. Other studies with rats, rabbits and monkeys have not shown teratogenic effects. Human data are ambiguous; it is well established that lithium can cross the human placenta. Of 225 registered pregnancies in which the mothers had received lithium (as a tranquiliser) there were 25 instances of congenital malformation. Although pharmacological doses of lithium cannot be unequivocally designated as a human teratogen, lithium therapy is contraindicated in women of childbearing potential.

Prolonged exposure may produce anorexia, weight loss and emaciation. The kidneys, behavioural/ central nervous system and peripheral nervous system may also show adverse effects.

Various types of dermatitis (psoriasis, alopecia, cutaneous ulcers, acne, follicular papules, xerosis cutis, exfoliative) may also result from chronic skin exposure.

Lithium ion can be an effective treatment for manic depression. It is thought to bind the enzyme IMPase (inositol monophosphatase) and thereby mediates its influence in producing a response to calcium-induced production of neurotransmitters and hormones thought to be responsible for the clinical picture.

Lithium ions interfere with ion transport processes (involving the "sodium pump") that relay and amplify messages carried to the cells of the brain. Maria 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 nickel at concentrations in excess of 1 mg Ni/m³ and exposure to the less soluble forms at concentrations greater than 10 mg Ni/m³. 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 polycyclic aromatic 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 polycyclic aromatic 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.

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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.

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/m³ for channel black and 56.5 mg/m³ for furnace black, no malignancies were observed in any of the animals. Channel black had little if any absorbed polycyclic aromatic 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/m³ carbon black and ~10 mg/m³ 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 electrocardiographic 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.

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.

On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body 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.

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TOXICITY

Not Available

IRRITATION

Not Available

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lithium nickel manganese cobalt oxide	TOXICITY Not Available	IRRITATION Not Available
graphite	TOXICITY Inhalation (Rat) LC50: >2 mg/L4h ^[1] Oral (Rat) LD50; >200 mg/kg ^[1]	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: no adverse effect observed (not irritating) ^[1]
vinylidene fluoride homopolymer	TOXICITY Not Available	IRRITATION Not Available
ethylene carbonate	TOXICITY dermal (rat) LD50: >2000 mg/kg ^[1] Oral (Rat) LD50; >2000 mg/kg ^[1]	IRRITATION Eye: adverse effect observed (irritating) ^[1] Skin (Rodent - rabbit): 660mg - Mild Skin: no adverse effect observed (not irritating) ^[1]
lithium fluorophosphate	TOXICITY Oral (Rat) LD50; 50-300 mg/kg ^[1]	IRRITATION Eye: adverse effect observed (irritating) ^[1] Skin: adverse effect observed (corrosive) ^[1]

Legend:

1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. Value obtained from manufacturer's SDS.
Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances

2000LM RECHARGEABLE SHOP LIGHT	<p>The material may trigger oculogyric crisis. The term "oculogyric" 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. 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. In addition to the acute presentation, oculogyric crisis can develop as a recurrent syndrome, triggered by stress and by exposure to the drugs. 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>
ETHYLENE CARBONATE	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin. for ethylene carbonate</p> <p>Mammalian toxicity: Reliable acute toxicity tests are available on ethylene carbonate. Ethylene carbonate is practically nontoxic following acute oral exposure in a test that meets OECD and EPA test guidelines; the LD50 is >5000 mg/kg. The dermal LD50 is >2000 mg/kg, in a test that meets OECD and EPA test guidelines.</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 Cr: 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, gasteroschisis, 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> <p>Ethylene glycol is quickly and extensively absorbed throughout the gastrointestinal tract. Limited information suggests that it is also absorbed through the airways; absorption through skin is apparently slow. Following absorption, it is distributed throughout the body. In humans, it is initially metabolized by alcohol dehydrogenase to form glycoaldehyde, which is rapidly converted to glycolic acid and glyoxal. These breakdown products are oxidized to glyoxylate, which may be further metabolized to formic acid,</p>

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oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate carbon dioxide, which is one of the major elimination products of ethylene glycol. In addition to exhaled carbon dioxide, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination is rapid and occurs within a few hours.

Respiratory effects: Respiratory system involvement occurs 12-24 hours after swallowing sufficient amounts of ethylene glycol. Symptoms include hyperventilation, shallow rapid breathing, and generalized swelling of the lungs with calcium oxalate deposits occasionally appearing in the lungs. Respiratory system involvement appears to be dose-dependent and occurs at the same time as cardiovascular changes. Later, there may be other changes compatible with adult respiratory distress syndrome (ARDS). Swelling of the lung can be a result of heart failure, ARDS, or aspiration of stomach contents. Symptoms related to acidosis such as fast or excessive breathing are frequently observed; however, major symptoms such as swelling of the lung and inflammation of the bronchi and lungs are relatively rare, and are usually seen only in extreme poisoning.

Cardiovascular effects: Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of ethylene glycol poisoning by swallowing, which is 12-24 hours after acute exposure. The symptoms of poisoning involving the heart include increased heart rate, heart enlargement and ventricular gallop. There may also be high or low blood pressure, which may progress to cardiogenic shock. In lethal cases, inflammation of the heart muscle has been observed at autopsy. Cardiovascular involvement appears to be rare and usually seen after swallowing higher doses of ethylene glycol. In summary, 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: Common early acute effects of swallowing ethylene glycol include nausea, vomiting with or without blood, heartburn and abdominal cramping and pain. One patient showed intermittent diarrhea and pain, and after surgery, deposition of oxalate crystals was shown to have occurred.

Musculoskeletal effects: Reported musculoskeletal effects in cases of acute ethylene glycol poisoning include diffuse muscle tenderness and pain, associated with high levels of creatinine in the blood, and jerks and contractions associated with low calcium.

Liver effects: Autopsies carried out on people who died following acute ethylene glycol poisoning showed deposition of calcium oxalate in the liver as well as hydropic and fatty degeneration and cell death (necrosis) of the liver.

Kidney effects: Adverse kidney effects are seen during the third stage of ethylene glycol poisoning, 2-3 days after acute exposure. Calcium oxalate crystals are deposited in the tubules and are seen in the urine. There may also be degeneration and death of tubule cells, and inflammation of the tubule interstitium. If untreated, the degree of kidney damage progresses and leads to blood and protein in the urine, decreased kidney function, reduction in urine output and ultimately, kidney failure. With adequate supportive therapy, kidney function can return to normal or near normal.

Metabolic effects: Metabolic changes can occur within 12 hours of exposure to ethylene glycol. There may be metabolic acidosis, caused by accumulation of glycolic acid in the blood and therefore a reduction in blood pH. The anion gap is increased, due to increased unmeasured anions (mainly glycolate).

Effects on the nervous system: Adverse reactions involving the nervous system are among the first symptoms to appear in humans after ethylene glycol is swallowed. These early effects are also the only symptoms caused by unmetabolised ethylene glycol. Together with metabolic effects (see above), they occur from 0.5-12 hours after exposure and are considered to be part of the first stage in ethylene glycol poisoning. Inco-ordination, slurred speech, confusion and sleepiness are common in the early stages, as are irritation, restlessness and disorientation. Later, there may be effects on cranial nerves (which may be reversible over many months). Swelling of the brain (cerebrum) and crystal deposits of calcium oxalate in the walls of the small blood vessels of the brain were found at autopsy in people who died after acute ethylene glycol poisoning.

Reproductive effects: Animal testing showed that ethylene glycol may affect fertility, survival of fetuses and the male reproductive organs.

Effects on development: Animal studies indicate that birth defects may occur after exposure in pregnancy; there may also be reduction in foetal weight.

Cancer: No studies are known regarding cancer effects in humans or animal, after skin exposure to ethylene glycol.

Genetic toxicity: No human studies available, but animal testing results are consistently negative.

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SHOP LIGHT & GRAPHITE
& ETHYLENE CARBONATE
& LITHIUM
FLUOROPHOSPHATE**

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.

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SHOP LIGHT & LITHIUM
NICKEL MANGANESE
COBALT OXIDE**

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.

Goitrogenic:

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

Goitrogens include:

- ▶ Vitexin, a flavonoid, which inhibits thyroid peroxidase thus contributing to goiter.
- ▶ 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.
- ▶ Lithium which inhibits thyroid hormone release.
- ▶ Certain foods, such as soy and millet (containing vitexins) and vegetables in the genus Brassica (e.g. broccoli, brussels sprouts, cabbage, horseradish).
- ▶ Caffeine (in coffee, tea, cola, chocolate) which acts on thyroid function as a suppressant.

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LITHIUM NICKEL MANGANESE COBALT OXIDE & GRAPHITE & VINYLIDENE FLUORIDE HOMOPOLYMER & LITHIUM FLUOROPHOSPHATE	No significant acute toxicological data identified in literature search.	
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Acute Toxicity	✗	Carcinogenicity	✓
Skin Irritation/Corrosion	✓	Reproductivity	✗
Serious Eye Damage/Irritation	✓	STOT - Single Exposure	✓
Respiratory or Skin sensitisation	✓	STOT - Repeated Exposure	✓
Mutagenicity	✗	Aspiration Hazard	✗

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

SECTION 12 Ecological information

Toxicity

2000LM RECHARGEABLE SHOP LIGHT	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
lithium nickel manganese cobalt oxide	Endpoint	Test Duration (hr)	Species	Value	Source
graphite	Not Available	Not Available	Not Available	Not Available	Not Available
vinylidene fluoride homopolymer	Endpoint	Test Duration (hr)	Species	Value	Source
ethylene carbonate	Not Available	Not Available	Not Available	Not Available	Not Available
lithium fluorophosphate	Endpoint	Test Duration (hr)	Species	Value	Source
Legend: Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data					

Although small amounts of fluorides are conceded to have beneficial effects, two forms of chronic toxic effect, dental fluorosis and skeletal fluorosis may be caused by excessive intake over long periods. Fluorides are absorbed by humans following inhalation of workplace and ambient air that has been contaminated, ingestion of drinking water and foods and dermal contact.

Fluoride accumulates, food-dependently in skeletal tissues of both aquatic and terrestrial vertebrates and invertebrates. Bioaccumulation occurs in marine organisms and, to a lesser extend, fresh water organisms. Reported BCF-values for marine organisms range up to approximately 150 and 60 for fish and crustacea, respectively. The most important exposure route for plants is uptake from the atmosphere. Concentrations in plants in the vicinity of a HF production plant range up to approximately 200 mg/kg, with mean levels between 20 and 50 mg/kg dry weight. Generally, lowest fluoride levels are found in herbivores and (somewhat) higher levels in predators.

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Fluorides have been shown to accumulate in animals that consume fluoride-containing foliage. However, accumulation is primarily in skeletal tissue and therefore, it is unlikely that fluoride will biomagnify up the food chain.

Both hydrogen fluoride and particulate fluorides will be transported in the atmosphere and deposited on land or water by wet and dry deposition. Non-volatile inorganic fluoride particulates are removed from the atmosphere via condensation or nucleation processes. Fluorides adsorbed on particulate matter in the atmosphere are generally stable and are not readily hydrolysed, although they may be degraded by radiation if they persist in the atmosphere. Fluorine and the silicon fluorides (fluosilicates, silicofluorides) are hydrolysed in the atmosphere to form hydrogen fluoride. Hydrogen fluoride may combine with water vapour to produce an aerosol or fog of aqueous hydrofluoric acid. Based upon available data, inorganic fluoride compounds, with the exception of sulfur hexafluoride, are not expected to remain in the troposphere for long periods or to migrate to the stratosphere. Estimates of the residence time of sulfur hexafluoride in the atmosphere range from 500 to several thousand years. Fluoride in aerosols can be transported over large distances by wind or as a result of atmospheric turbulence. The distance travelled is determined by the deposition velocity of both the gaseous hydrogen fluoride and the fluorides in particulate form. Atmospheric fluorides may be transported to soils and surface waters through both wet and dry deposition processes.

Fluorides undergo transformations in soil and water, forming complexes and binding strongly to soil and sediment.

In water, the transport and transformation of inorganic fluorides are influenced by pH, water hardness and the presence of ion-exchange materials such as clays.

In natural water, fluoride forms strong complexes with aluminum in water, and fluorine chemistry in water is largely regulated by aluminum concentration and pH. Below pH 5, fluoride is almost entirely complexed with aluminum and consequently, the concentration of free F⁻ is low. As the pH increases, Al-OH complexes dominate over Al-F complexes and the free F⁻ levels increase. Fluoride forms stable complexes with calcium and magnesium, which are present in sea water. Calcium carbonate precipitation dominates the removal of dissolved fluoride from sea water. The residence time for fluoride in ocean sediment is calculated to be 2-3 million years. Fluorosilicic acid and hydrofluoric acid in high aquatic concentrations such as may be found in industrial waste ponds may volatilise, releasing silicon tetrafluoride and hydrogen fluoride into the atmosphere.

Solubilisation of inorganic fluorides from minerals may also be enhanced by the presence of ion-exchange materials (e.g., bentonite clays and humic acid). Once dissolved, inorganic fluorides remain in solution under conditions of low pH and hardness and in the presence of ion-exchange material. Soluble inorganic fluorides may also form aerosols at the air-water interface or vaporise into the atmosphere whereas undissolved species generally undergo sedimentation.

Factors that influence the mobility of inorganic fluorides in soil are pH and the formation of aluminium and calcium complexes. In more acidic soils, concentrations of inorganic fluoride were considerably higher in the deeper horizons. The low affinity of fluorides for organic material results in leaching from the more acidic surface horizon and increased retention by clay minerals and silts in the more alkaline, deeper horizons. The maximum adsorption of fluoride to soil was reported to occur at pH 5.5. In acidic soils with pH below 6, most of the fluoride is in complexes with either aluminium or iron. Fluoride in alkaline soils at pH 6.5 and above is almost completely fixed in soils as calcium fluoride, if sufficient calcium carbonate is available. Fluoride is extremely immobile in soil, as determined by lysimeter experiments.

Populations living in areas with high fluoride levels in groundwater may be exposed to higher levels of fluorides in their drinking water or in beverages prepared with the water. Among these populations, outdoor laborers, people living in hot climates, and people with polydipsia will generally have the greatest daily intake of fluorides because they consume greater amounts of water.

Foods characteristically high in fluoride content are certain types of fish and seafood (1.9-28.5 mg/kg), especially those types in which the bones are consumed, bone products such as bone meal and gelatin, and tea, which contains approximately 0.52 mg fluoride/cup

Fluoride is mainly absorbed by the body in the form of hydrogen fluoride, which has a pKa of 3.45. That is, when ionic fluoride enters the acidic environment of the stomach lumen, it is largely converted into hydrogen fluoride. Most of the fluoride that is not absorbed from the stomach will be rapidly absorbed from the small intestine.

For manganese and its compounds:

Environmental fate:

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

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

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

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

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

Ecotoxicity:

Manganese ion is toxic to aqueous organisms

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

Daphnia magna LC50: 50 mg/l

Pseudomonas putida LC50: 10.6 mg/l

Photobacterium phosphoreum LC50: 14.7 mg/l

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

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)

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 O₂, pH 7.2) was stimulated at lithium ion concentration 10-20 mg/l, slightly inhibited at lithium ion concentration 350 mg/l and seriously inhibited at lithium ion concentration > 500 mg/l.

Microinjection of lithium chloride into prospective ventral blastomeres of a 32-cell *Xenopus* larva 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 60Co 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. 60Co is more mobile in anaerobic marine aquatic environments than in freshwater aerobic ones. In seawater sediment systems under anaerobic conditions 60Co was 250 times more mobile than 60Co in freshwater sediment systems under aerobic conditions. Under anaerobic conditions, 30% of the 60Co added to a sediment-freshwater system was "exchangeable" and therefore potentially mobile, while under aerobic conditions, 98% of the 60Co was permanently fixed. Most of the mobile 60Co 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 60Co 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⁺. 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 Kd of cobalt in a variety of soils ranged from 0.2 to 3,800. The soil properties showing the highest correlation with Kd 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 60Co was not accumulated by plants and mushrooms. Studies investigating the uptake of 60Co by tomato plants watered with 60Co contaminated water showed that tomato plants absorbed <2% of the activity available from the soil.

60Co is taken up by phytoplankton and unicellular algae (*Senecastrum 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 60Co 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.

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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 60Co 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 60Co 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 60Co was very low in whitefish, with concentrations being highest in kidney and undetectable in muscle. Similarly, while accumulation of 60Co 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 crustaceae 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 60Co 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 60Co. 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. for ethylene carbonate:

Environmental fate:

Biodegradation (21 d): 90-100% (OECD 301 A -aerobic, activated sludge, domestic, DOC reduction) - readily biodegradable.

Estimates of photodegradation based on reaction rates with hydroxyl radicals are available. Data on biodegradation were developed using current guidelines.

Using OECD Method 301B, ethylene carbonate is readily biodegradable; more than 72% was degraded to CO₂ within 9 days and more than 90% after 28 days.

Degradates to ethylene glycol in the environment.

Ecotoxicity:

Daphnia magna EC50 (48 h): >100 mg/l (OECD 202, Part 1 static - nominal concentration)

Activated sludge (domestic) EC50 (30 m): >1000 mg/l (DIN EN 8192; OECD 209-88/302/EE - nominal concentration)

Bacterium EC50 (17 h): >10000 mg/l (DIN 38412, Part 8 - nominal)

Data on acute fish and toxicity to plants from ethylene carbonate were not found, but data on ethylene glycol (the degradate) is available for ethylene glycol:

log K_{ow} : -1.93- -1.36

Half-life (hr) air : 24

Henry's atm m³/mol: 6.00E-08

BOD 5 : 0.15-0.81,12%

COD : 1.21-1.29

ThOD : 1.26

BCF : 10-190

In the atmosphere ethylene glycol exists mainly in the vapour phase. It is degraded in the atmosphere by reaction with photochemically produced hydroxy radicals (estimated half-life 24-50 hours).

Ethylene glycol does not concentrate in the food chain.

Environmental fate:

Ethylene glycol has a low vapour pressure (7.9 Pa at 20 C); it is expected to exist almost entirely in the vapour phase if released to the atmosphere. The Henry's law constant for ethylene glycol is 1.41×10^{-3} or 6.08×10^{-3} Pa.m³/mol, depending on method of calculation, indicating a low capacity for volatilisation from water bodies or soil surfaces.

Ethylene glycol adsorbed onto silica gel and irradiated with light (wavelength >290 nm) degraded by 12.1% over 17 h. Photodegradation is not expected, as the molecule should not absorb at these wavelengths; the mechanism of this breakdown is, therefore, unknown. Estimated half-life in the atmosphere for reaction with hydroxyl radicals from various reports is 2.1 days, 8-84 h or 1 day.

Ethylene glycol released to the atmosphere will be degraded by reaction with hydroxyl radicals; the half-life for the compound in this reaction has been estimated at between 0.3 and 3.5 days. No hydrolysis of ethylene glycol is expected in surface waters.

The compound has little or no capacity to bind to particulates and will be mobile in soil. Soil partition coefficients (log K_{oc}) of 0-0.62 were determined. Migration rates in five soil types were measured at between 4 and 27 cm per 12 h

The low octanol/water partition coefficient (log K_{ow} -1.93 to -1.36) and measured bioconcentration factors in a few organisms indicate low capacity for bioaccumulation. Bioconcentration factors of 190 for the green algae (*Chlorella fusca*), up to 0.27 in specific tissues of the crayfish (*Procambarus sp.*), and 10 for the golden orfe (*Leuciscus idus melanotus*) confirm low bioaccumulation.

Ethylene glycol is readily biodegradable in standard tests using sewage sludge. Many studies show biodegradation under both aerobic and anaerobic conditions. Some studies suggest a lag phase before degradation, but many do not. Degradation occurs in both adapted and unadapted sludges. Rapid degradation has been reported in surface waters (less in salt water than in fresh water), groundwater, and soil inocula. Several strains of microorganisms capable of utilising ethylene glycol as a carbon source have been identified.

Ethylene glycol has been identified as a metabolite of the growth regulator ethylene in a number of higher plants and as naturally occurring in the edible fungus *Tricholoma matsutake*

Ecotoxicity:

Fish LC50 (96 h): 118-550 mg/L

Ethylene glycol has generally low toxicity to aquatic organisms. Toxic thresholds for microorganisms are above 1000 mg/litre. EC50s for growth in microalgae are 6500 mg/litre or higher. Acute toxicity tests with aquatic invertebrates where a value could be determined show LC50s above 20 000 mg/litre, and those with fish show LC50s above 17 800 mg/litre. An amphibian test showed an LC50 for tadpoles at 17 000 mg/litre. A no-observed-effect concentration (NOEC) for chronic tests on daphnids of 8590 mg/litre (for reproductive end-points) has been reported. A NOEC following short-term exposure of fish has been reported at 15 380 mg/litre for growth. Tests using deicer containing ethylene glycol showed greater toxicity to aquatic organisms than observed with the pure compound, indicating other toxic components of the formulations. Laboratory tests exposing aquatic organisms to stream water receiving runoff from airports have demonstrated toxic effects and death. Field studies in the vicinity of an airport have reported toxic signs consistent with ethylene glycol poisoning, fish kills, and reduced biodiversity. These effects cannot definitively be ascribed to ethylene glycol. Terrestrial organisms are much less likely to be exposed to ethylene glycol and generally show low sensitivity to the compound. Concentrations above 100 000 mg/litre were needed to produce toxic effects on yeasts and fungi from soil. Very high concentrations and soaking of seeds produced inhibition of germination in some experiments; these are not considered of environmental significance. A no-observed-effect level (NOEL) for orally dosed ducks at 1221 mg/kg body weight and reported lethal doses for poultry at around 8000 mg/kg body weight indicate low toxicity to birds.

DO NOT discharge into sewer or waterways.

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Ingredient	Persistence: Water/Soil	Persistence: Air
vinylidene fluoride homopolymer	LOW	LOW
ethylene carbonate	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
vinylidene fluoride homopolymer	LOW (LogKOW = 1.24)
ethylene carbonate	LOW (LogKOW = -0.3388)

Mobility in soil

Ingredient	Mobility
vinylidene fluoride homopolymer	LOW (Log KOC = 35.04)
ethylene carbonate	LOW (Log KOC = 9.168)

SECTION 13 Disposal considerations**Waste treatment methods**

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product. ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority. ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Authority for disposal. ▶ Bury or incinerate residue at an approved site. ▶ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information**Labels Required**

	
Marine Pollutant	NO
HAZCHEM	2Y

Land transport (ADG)

14.1. UN number or ID number	3481	
14.2. UN proper shipping name	LITHIUM ION BATTERIES PACKED WITH EQUIPMENT (including lithium ion polymer batteries); LITHIUM ION BATTERIES CONTAINED IN EQUIPMENT (including lithium ion polymer batteries)	
14.3. Transport hazard class(es)	Class	9
	Subsidiary Hazard	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Special provisions	188 230 310 348 360 376 377 384 387 390
	Limited quantity	0

Air transport (ICAO-IATA / DGR)

14.1. UN number	3481	
14.2. UN proper shipping name	Lithium ion batteries packed with equipment (including lithium ion polymer batteries); Lithium ion batteries contained in equipment (including lithium ion polymer batteries)	
14.3. Transport hazard class(es)	ICAO/IATA Class	9
	ICAO / IATA Subsidiary Hazard	Not Applicable
	ERG Code	12FZ
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Special provisions	A48 A88 A99 A154 A164 A181 A185 A213 A220; A88 A99 A154 A164 A181 A185 A213 A802
	Cargo Only Packing Instructions	967; 966
	Cargo Only Maximum Qty / Pack	35 kg
	Passenger and Cargo Packing Instructions	967; 966
	Passenger and Cargo Maximum Qty / Pack	5 kg
	Passenger and Cargo Limited Quantity Packing Instructions	Forbidden
	Passenger and Cargo Limited Maximum Qty / Pack	Forbidden

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	3481	
14.2. UN proper shipping name	LITHIUM ION BATTERIES CONTAINED IN EQUIPMENT (including lithium ion polymer batteries); LITHIUM ION BATTERIES PACKED WITH EQUIPMENT (including lithium ion polymer batteries)	
14.3. Transport hazard class(es)	IMDG Class	9
	IMDG Subsidiary Hazard	Not Applicable
14.4. Packing group	Not Applicable	
14.5 Environmental hazard	Not Applicable	
14.6. Special precautions for user	EMS Number	F-A, S-I
	Special provisions	188 230 310 348 360 376 377 384 387 390
	Limited Quantities	0

14.7. Maritime transport in bulk according to IMO instruments**14.7.1. Transport in bulk according to Annex II of MARPOL and the IBC code**

Not Applicable

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

Product name	Group
lithium nickel manganese cobalt oxide	Not Available
graphite	Not Available
vinylidene fluoride homopolymer	Not Available
ethylene carbonate	Not Available
lithium fluorophosphate	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
lithium nickel manganese cobalt oxide	Not Available
graphite	Not Available
vinylidene fluoride homopolymer	Not Available

Product name	Ship Type
ethylene carbonate	Not Available
lithium fluorophosphate	Not Available

SECTION 15 Regulatory information

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

lithium nickel manganese cobalt oxide 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)

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)

vinylidene fluoride homopolymer 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)

ethylene carbonate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

lithium fluorophosphate 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)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIC / Australia Non-Industrial Use	No (lithium nickel manganese cobalt oxide)
Canada - DSL	No (lithium nickel manganese cobalt oxide; lithium fluorophosphate)
Canada - NDSL	No (lithium nickel manganese cobalt oxide; graphite; vinylidene fluoride homopolymer; ethylene carbonate)
China - IECSC	No (lithium nickel manganese cobalt oxide)
Europe - EINEC / ELINCS / NLP	No (lithium nickel manganese cobalt oxide; vinylidene fluoride homopolymer)
Japan - ENCS	No (lithium nickel manganese cobalt oxide; graphite)
Korea - KECI	No (lithium nickel manganese cobalt oxide)
New Zealand - NZIoC	No (lithium nickel manganese cobalt oxide; lithium fluorophosphate)
Philippines - PICCS	No (lithium nickel manganese cobalt oxide)
USA - TSCA	TSCA Inventory 'Active' substance(s) (graphite; vinylidene fluoride homopolymer; ethylene carbonate; lithium fluorophosphate); No (lithium nickel manganese cobalt oxide)
Taiwan - TCSI	Yes
Mexico - INSQ	No (lithium nickel manganese cobalt oxide; vinylidene fluoride homopolymer; ethylene carbonate; lithium fluorophosphate)
Vietnam - NCI	Yes
Russia - FBEPH	No (lithium nickel manganese cobalt oxide; lithium fluorophosphate)
UAE - Control List (Banned/Restricted Substances)	No (lithium nickel manganese cobalt oxide; vinylidene fluoride homopolymer; ethylene carbonate; lithium fluorophosphate)
Legend:	<i>Yes = All CAS declared ingredients are on the inventory</i> <i>No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.</i>

SECTION 16 Other information

Initial Date	25/09/2025
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Other information

Ingredients with multiple cas numbers

Name	CAS No
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

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

Definitions and abbreviations

- ▶ PC - TWA: Permissible Concentration-Time Weighted Average
- ▶ PC - STEL: Permissible Concentration-Short Term Exposure Limit
- ▶ IARC: International Agency for Research on Cancer
- ▶ ACGIH: American Conference of Governmental Industrial Hygienists
- ▶ STEL: Short Term Exposure Limit
- ▶ TEEL: Temporary Emergency Exposure Limit
- ▶ IDLH: Immediately Dangerous to Life or Health Concentrations
- ▶ ES: Exposure Standard
- ▶ OSF: Odour Safety Factor
- ▶ NOAEL: No Observed Adverse Effect Level
- ▶ LOAEL: Lowest Observed Adverse Effect Level
- ▶ TLV: Threshold Limit Value
- ▶ LOD: Limit Of Detection
- ▶ OTV: Odour Threshold Value
- ▶ BCF: BioConcentration Factors
- ▶ BEI: Biological Exposure Index
- ▶ DNEL: Derived No-Effect Level
- ▶ PNEC: Predicted no-effect concentration
- ▶ MARPOL: International Convention for the Prevention of Pollution from Ships
- ▶ IMSBC: International Maritime Solid Bulk Cargoes Code
- ▶ IGC: International Gas Carrier Code
- ▶ IBC: International Bulk Chemical Code
- ▶ AIIC: Australian Inventory of Industrial Chemicals
- ▶ DSL: Domestic Substances List
- ▶ NDSL: Non-Domestic Substances List
- ▶ IECSC: Inventory of Existing Chemical Substance in China
- ▶ EINECS: European INventory of Existing Commercial chemical Substances
- ▶ ELINCS: European List of Notified Chemical Substances
- ▶ NLP: No-Longer Polymers
- ▶ ENCS: Existing and New Chemical Substances Inventory
- ▶ KECI: Korea Existing Chemicals Inventory
- ▶ NZIoC: New Zealand Inventory of Chemicals
- ▶ PICCS: Philippine Inventory of Chemicals and Chemical Substances
- ▶ TSCA: Toxic Substances Control Act
- ▶ TCSI: Taiwan Chemical Substance Inventory
- ▶ INSQ: Inventario Nacional de Sustancias Químicas
- ▶ NCI: National Chemical Inventory
- ▶ FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances