



InverX 30, 45, 65, 80

POPS Group (The POPS Group Pty Ltd as Trustee for The Pool Shops Trust)

Chemwatch Hazard Alert Code: 3

Chemwatch: 5650-16

Version No: 3.1

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

Issue Date: 26/02/2024

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L.GHS.AUS.EN.E

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

Product Identifier

Product name	InverX 30, 45, 65, 80
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	LITHIUM ION BATTERIES (including lithium ion polymer batteries)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Pool cleaner battery Use according to manufacturer's directions.
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Details of the manufacturer or supplier of the safety data sheet

Registered company name	POPS Group (The POPS Group Pty Ltd as Trustee for The Pool Shops Trust)
Address	10-12 Cairns Street Loganholme QLD 4129 Australia
Telephone	+61 7 3209 7884
Fax	+61 7 3209 8635
Website	http://www.poolpro.com.au/
Email	office@poolpro.com.au

Emergency telephone number

Association / Organisation	IXOM
Emergency telephone numbers	+61 3 9663 2130 (International) (24 hours)
Other emergency telephone numbers	+61 1800 033 111

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2A, Hazardous to the Aquatic Environment Long-Term Hazard Category 4
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
Signal word	Warning

Hazard statement(s)

H315	Causes skin irritation.
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H319	Causes serious eye irritation.
H413	May cause long lasting harmful effects to aquatic life.

Precautionary statement(s) Prevention

P273	Avoid release to the environment.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P264	Wash all exposed external body areas thoroughly after handling.

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.
P337+P313	If eye irritation persists: Get medical advice/attention.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

Not Applicable

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

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available		hermetically sealed metal case containing,
12190-79-3	15-40	<u>lithium cobaltate</u>
7782-42-5	10-30	<u>graphite</u>
21324-40-3	10-30	<u>lithium fluorophosphate</u>
7440-50-8	7-13	<u>copper</u>
7429-90-5.	5-10	<u>APSC Aluminium Foil</u>
7440-02-0	1-5	<u>nickel</u>

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

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

Eye Contact	<p>If battery is leaking and material contacts the eye. 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. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ 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. ▶ Seek medical attention without delay.
Skin Contact	<p>If battery is leaking and material contacts the skin. Remove all contaminated clothing, including footwear. Wash thoroughly all affected areas with water and soap. Seek medical attention if swelling/redness/blistering or irritation occurs.</p>
Inhalation	<p>If battery is leaking, contents may be irritating to respiratory passages. Remove patient to fresh air and seek medical attention.</p>
Ingestion	<ul style="list-style-type: none"> ▶ For advice, contact a Poisons Information Centre or a doctor.

Indication of any immediate medical attention and special treatment needed

Following acute or short term repeated exposure to hydrofluoric acid:

- ▶ Subcutaneous injections of Calcium Gluconate may be necessary around the burnt area. Continued application of Calcium Gluconate Gel or subcutaneous Calcium Gluconate should then continue for 3-4 days at a frequency of 4-6 times per day. If a "burning" sensation recurs, apply more frequently.
- ▶ Systemic effects of extensive hydrofluoric acid burns include renal damage, hypocalcaemia and consequent cardiac arrhythmias. Monitor haematological, respiratory, renal, cardiac and electrolyte status at least daily. Tests should include FBE, blood gases, chest X-ray, creatinine and electrolytes, urine output, Ca ions, Mg ions and phosphate ions. Continuous ECG monitoring may be required.
- ▶ Where serum calcium is low, or clinical, or ECG signs of hypocalcaemia develop, infusions of calcium gluconate, or if less serious, oral Sandocal, should be given. Hydrocortisone 500 mg in a four to six hourly infusion may help.
- ▶ Antibiotics should not be given as a routine, but only when indicated.
- ▶ Eye contact pain may be excruciating and 2-3 drops of 0.05% pentocaine hydrochloride may be instilled, followed by further irrigation

These represent the determinants observed in specimens collected from a healthy worker exposed at the Exposure Standard (ES or TLV):

Determinant	Index	Sampling Time	Comments
1. Methaemoglobin in blood	1.5% of haemoglobin	During or end of shift	B, NS, SQ

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

NS: Non-specific determinant; Also seen after exposure to other materials

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

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

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

[Ellenhorn and Barceloux: Medical Toxicology]

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

BIOLOGICAL EXPOSURE INDEX (BEI)

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

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

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

SECTION 5 Firefighting measures

Extinguishing media

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

DO NOT USE WATER, CO₂ or FOAM.

- ▶ Use DRY sand, graphite powder, dry sodium chloride based extinguishers, G-1 or Met L-X to smother fire.
- ▶ Confining or smothering material is preferable to applying water as chemical reaction may produce flammable and explosive hydrogen gas.
- ▶ Chemical reaction with CO₂ may produce flammable and explosive methane.
- ▶ If impossible to extinguish, withdraw, protect surroundings and allow fire to burn itself out.
- ▶ Sand, dry powder extinguishers or other inerts should be used to smother dust fires.

At temperatures above 1500 C, carbon, graphite or graphene reacts with substances containing oxygen, including water and carbon dioxide. In case of intensely hot fires sand should be used to cover and isolate these materials.

- ▶ **DO NOT** use halogenated fire extinguishing agents.

Special hazards arising from the substrate or mixture

Fire Incompatibility	▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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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. <p>Slight hazard when exposed to heat, flame and oxidisers.</p>
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ DO NOT disturb burning dust. Explosion may result if dust is stirred into a cloud, by providing oxygen to a large surface of hot metal. ▶ DO NOT use water or foam as generation of explosive hydrogen may result. <p>With the exception of the metals that burn in contact with air or water (for example, sodium), masses of combustible metals do not represent unusual fire risks because they have the ability to conduct heat away from hot spots so efficiently that the heat of combustion cannot be maintained - this means that it will require a lot of heat to ignite a mass of combustible metal. Generally, metal fire risks exist when sawdust, machine shavings and other metal 'fines' are present.</p> <p>Metal powders, while generally regarded as non-combustible:</p> <ul style="list-style-type: none"> ▶ May burn when metal is finely divided and energy input is high. ▶ May react explosively with water. ▶ May be ignited by friction, heat, sparks or flame. ▶ May REIGNITE after fire is extinguished. ▶ Will burn with intense heat. <p>Note:</p> <ul style="list-style-type: none"> ▶ Metal dust fires are slow moving but intense and difficult to extinguish. ▶ Containers may explode on heating. ▶ Dusts or fumes may form explosive mixtures with air. ▶ Gases generated in fire may be poisonous, corrosive or irritating. ▶ Hot or burning metals may react violently upon contact with other materials, such as oxidising agents and extinguishing agents used on fires involving ordinary combustibles or flammable liquids. ▶ Temperatures produced by burning metals can be higher than temperatures generated by burning flammable liquids ▶ Some metals can continue to burn in carbon dioxide, nitrogen, water, or steam atmospheres in which ordinary combustibles or flammable

liquids would be incapable of burning.
 carbon dioxide (CO₂)
 hydrogen fluoride
 phosphorus oxides (PO_x)
 metal oxides
 other pyrolysis products typical of burning organic material.
 Articles and manufactured articles may constitute a fire hazard where polymers form their outer layers or where combustible packaging remains in place.
 Certain substances, found throughout their construction, may degrade or become volatile when heated to high temperatures. This may create a secondary hazard.
 A fire in bulk finely divided carbon may not be obviously visible unless the material is disturbed and sparks appear. A straw broom may be useful to produce the disturbance.
 Explosion and Ignition Behaviour of Carbon Black with Air

Lower Limit for Explosion:	50 g/m 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.)

Notes on Test Methods:

Tests 1, 2 and 3 were conducted by Bergwerkeschaftliche Versuchsstrecke, Dortmunde-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)

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. ▶ Secure load if safe to do so. ▶ Bundle/collect recoverable product. ▶ Collect remaining material in containers with covers for disposal.
Major Spills	<ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Wear protective clothing, safety glasses, dust mask, gloves. ▶ Secure load if safe to do so. Bundle/collect recoverable product. ▶ Use dry clean up procedures and avoid generating dust. ▶ Vacuum up (consider explosion-proof machines designed to be grounded during storage and use). ▶ Water may be used to prevent dusting. ▶ Collect remaining material in containers with covers for disposal. ▶ Flush spill area with water.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ Limit all unnecessary personal contact. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ When handling DO NOT eat, drink or smoke. ▶ Always wash hands with soap and water after handling. ▶ Avoid physical damage to containers. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
Other information	<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. ▶ Store away from incompatible materials. <p>Keep out of reach of children.</p>

Conditions for safe storage, including any incompatibilities

Continued...

Suitable container	<ul style="list-style-type: none"> ▸ Packaging as recommended by manufacturer.
Storage incompatibility	<ul style="list-style-type: none"> ▸ Avoid strong bases. ▸ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates. ▸ Avoid reaction with oxidising agents ▸ Keep dry

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	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.
Australia Exposure Standards	copper	Copper (fume)	0.2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	copper	Copper, dusts & mists (as Cu)	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	APSC Aluminium Foil	Aluminium, pyro powders (as Al)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	APSC Aluminium Foil	Aluminium (welding fumes) (as Al)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	APSC Aluminium Foil	Aluminium (metal dust)	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	nickel	Nickel, metal	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	nickel	Nickel, powder	1 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
graphite	6 mg/m3	330 mg/m3	2,000 mg/m3
lithium fluorophosphate	7.5 mg/m3	83 mg/m3	500 mg/m3
copper	3 mg/m3	33 mg/m3	200 mg/m3
nickel	4.5 mg/m3	50 mg/m3	99 mg/m3

Ingredient	Original IDLH	Revised IDLH
lithium cobaltate	Not Available	Not Available
graphite	1,250 mg/m3	Not Available
lithium fluorophosphate	Not Available	Not Available
copper	100 mg/m3	Not Available
APSC Aluminium Foil	Not Available	Not Available
nickel	10 mg/m3	Not Available


Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
lithium cobaltate	E	≤ 0.01 mg/m ³
lithium fluorophosphate	E	≤ 0.01 mg/m ³

Notes: Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.

MATERIAL DATA

Exposure controls

Appropriate engineering controls	General exhaust is adequate under normal operating conditions.
Individual protection measures, such as personal protective equipment	
Eye and face protection	<p>None under normal operating conditions.</p> <p>OTHERWISE:</p> <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	None under normal operating conditions. OTHERWISE: <ul style="list-style-type: none"> ▸ Wear chemical protective gloves, e.g. PVC. ▸ Wear safety footwear or safety gumboots, e.g. Rubber NOTE: <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	No special equipment needed when handling small quantities otherwise use <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.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Black coloured hermetically sealed cuboid battery with a medical ether-like odour if leaking; does not mix with water unless individual components exposed.		
Physical state	Manufactured	Relative density (Water = 1)	Not Applicable
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable
pH (as supplied)	Not Applicable	Decomposition temperature (°C)	Not Applicable
Melting point / freezing point (°C)	Not Applicable	Viscosity (cSt)	Not Applicable
Initial boiling point and boiling range (°C)	Not Applicable	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Applicable	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Applicable
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Applicable
Vapour pressure (kPa)	Not Applicable	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	Not Applicable	VOC g/L	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Inhalation of vapors or fumes released due to heat or a large number of leaking batteries may cause respiratory and eye irritation. Not normally a hazard due to physical form of product.
Ingestion	Contents of a cell if opened destructively or leaking may be harmful if swallowed. Not normally a hazard due to physical form of product. Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.
Skin Contact	Contact with battery contents will cause irritation. A shorted lithium battery can cause thermal and chemical burns upon contact with skin. Not normally a hazard due to physical form of product.
Eye	Contact with battery contents will cause irritation. Not normally a hazard due to physical form of product.
Chronic	<p>The chemicals in this product are contained in a sealed can and exposure does not occur during normal handling and use. Not normally a hazard due to physical form of product.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population.</p> <p>Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.</p> <p>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.</p> <p>Prolonged exposure may produce anorexia, weight loss and emaciation. The kidneys, behavioural/ central nervous system and peripheral nervous system may also show adverse effects.</p> <p>Various types of dermatitis (psoriasis, alopecia, cutaneous ulcers, acne, follicular papules, xerosis cutis, exfoliative) may also result from chronic skin exposure.</p> <p>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.</p> <p>Lithium ions interfere with ion transport processes (involving the "sodium pump") that relay and amplify messages carried to the cells of the brain. Mania is associated with irregular increases in protein kinase C (PKC) activity within the brain. Lithium carbonate and sodium valproate, another drug traditionally used to treat the disorder, act in the brain by inhibiting PKC's activity and help to produce other compounds that also inhibit the PKC.</p> <p>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.</p> <p>Lithium intoxication can affect the central nervous system and renal system and can be lethal</p> <p>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.</p> <p>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.</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.</p> <p>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/m³, and in mice at concentrations of .038 mg cobalt/m³. Both sexes of rats had greatly increased incidences (>90% incidence) of alveolar lesions at all exposure levels, including inflammatory changes, fibrosis, and metaplasia. Similar changes were seen in mice at all exposure levels, though the changes in mice were less severe.</p> <p>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. May possibly affect fertility*.</p>
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	TOXICITY	IRRITATION
InverX 30, 45, 65, 80	Not Available	Not Available
lithium cobaltate	dermal (rat) LD50: >2000 mg/kg ^[1]	Not Available
	Inhalation(Rat) LC50: 5.05 mg/14h ^[1]	
	Oral (Rat) LD50: >5000 mg/kg ^[1]	
graphite	Inhalation(Rat) LC50: >2 mg/L4h ^[1]	Not Available
	Oral (Rat) LD50: >200 mg/kg ^[1]	
lithium fluorophosphate	Oral (Rat) LD50: 50-300 mg/kg ^[1]	Not Available
copper	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation(Rat) LC50: 0.733 mg/14h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Mouse) LD50: 0.7 mg/kg ^[2]	
APSC Aluminium Foil	Inhalation(Rat) LC50: >2.3 mg/14h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
nickel	Oral (Rat) LD50: 5000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[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

LITHIUM COBALTATE	<p>Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens).</p> <p>Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis.</p> <p>Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.</p> <p>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:</p> <ul style="list-style-type: none"> ▶ Vitexin, a flavanoid, 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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COPPER	<p>WARNING: Inhalation of high concentrations of copper fume may cause "metal fume fever", an acute industrial disease of short duration. Symptoms are tiredness, influenza like respiratory tract irritation with fever.</p> <p>for copper and its compounds (typically copper chloride):</p> <p>Acute toxicity: There are no reliable acute oral toxicity results available. In an acute dermal toxicity study (OECD TG 402), one group of 5 male rats and 5 groups of 5 female rats received doses of 1000, 1500 and 2000 mg/kg bw via dermal application for 24 hours. The LD50 values of copper monochloride were 2,000 mg/kg bw or greater for male (no deaths observed) and 1,224 mg/kg bw for female. Four females died at both 1500 and 2000 mg/kg bw, and one at 1,000 mg/kg bw. Symptom of the hardness of skin, an exudation of hardness site, the formation of scar and reddish changes were observed on application sites in all treated animals. Skin inflammation and injury were also noted. In addition, a reddish or black urine was observed in females at 2,000, 1,500 and 1,000 mg/kg bw. Female rats appeared to be more sensitive than male based on mortality and clinical signs.</p> <p>No reliable skin/eye irritation studies were available. The acute dermal study with copper monochloride suggests that it has a potential to cause skin irritation.</p> <p>Repeat dose toxicity: In repeated dose toxicity study performed according to OECD TG 422, copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39 - 51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL value was 5 and 1.3 mg/kg bw/day for male and female rats, respectively. No deaths were observed in male rats. One treatment-related death was observed in female rats in the high dose group. Erythropoietic toxicity (anaemia) was seen in both sexes at the 80 mg/kg bw/day. The frequency of squamous cell hyperplasia of the forestomach was increased in a dose-dependent manner in male and female rats at all treatment groups, and was statistically significant in males at doses of =20 mg/kg bw/day and in females at doses of =5 mg/kg bw/day doses. The observed effects are considered to be local, non-systemic effect on the forestomach which result from oral (gavage) administration of copper monochloride.</p> <p>Genotoxicity: An in vitro genotoxicity study with copper monochloride showed negative results in a bacterial reverse mutation test with Salmonella typhimurium strains (TA 98, TA 100, TA 1535, and TA 1537) with and without S9 mix at concentrations of up to 1,000 ug/plate. An in vitro test for chromosome aberration in Chinese hamster lung (CHL) cells showed that copper monochloride induced structural and numerical aberrations at the concentration of 50, 70 and 100 ug/mL without S9 mix. In the presence of the metabolic activation system, significant increases of structural aberrations were observed at 50 and 70 ug/mL and significant increases of numerical aberrations were observed at 70 ug/mL. In an in vivo mammalian erythrocyte micronucleus assay, all animals dosed (15 - 60 mg/kg bw) with copper monochloride exhibited similar PCE/(PCE+NCE) ratios and MNPCE frequencies compared to those of the negative control animals. Therefore copper monochloride is not an in vivo mutagen.</p> <p>Carcinogenicity: there was insufficient information to evaluate the carcinogenic activity of copper monochloride.</p> <p>Reproductive and developmental toxicity: In the combined repeated dose toxicity study with the reproduction/developmental toxicity screening test (OECD TG 422), copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39-51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL of copper monochloride for fertility toxicity was 80 mg/kg bw/day for the parental animals. No treatment-related effects were observed on the reproductive organs and the fertility parameters assessed. For developmental toxicity the NOAEL was 20 mg/kg bw/day. Three of 120 pups appeared to have icterus at birth; 4 of 120 pups appeared runted at the highest dose tested (80 mg/kg bw/day).</p>		
NICKEL	<p>Oral (rat) TDLo: 500 mg/kg/5D-I Inhalation (rat) TCLo: 0.1 mg/m³/24H/17W-C</p> <p>WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.</p> <p>Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [National Toxicology Program: U.S. Dep. of Health & Human Services 2002]</p>		
LITHIUM COBALTATE & COPPER & NICKEL	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>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.</p>		
LITHIUM COBALTATE & GRAPHITE & LITHIUM FLUOROPHOSPHATE	No significant acute toxicological data identified in literature search.		
GRAPHITE & LITHIUM FLUOROPHOSPHATE	<p>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.</p>		
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

	Endpoint	Test Duration (hr)	Species	Value	Source
	InverX 30, 45, 65, 80	Not Available	Not Available	Not Available	Not Available
lithium cobaltate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	96h	Algae or other aquatic plants	23.8mg/l	2
	EC50	48h	Crustacea	0.241mg/L	2

Continued...

	EC50	72h	Algae or other aquatic plants	0.029mg/L	2
	EC10(ECx)	168h	Crustacea	0.001mg/L	2
	LC50	96h	Fish	0.8mg/l	2
graphite	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	>100mg/l	2
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	NOEC(ECx)	48h	Crustacea	>=100mg/l	2
	LC50	96h	Fish	>100mg/l	2
lithium fluorophosphate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	98mg/l	2
	EC50	96h	Algae or other aquatic plants	43mg/l	2
	EC50	72h	Algae or other aquatic plants	62mg/l	2
	LC50	96h	Fish	42mg/l	2
	NOEC(ECx)	528h	Fish	0.2mg/l	2
copper	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	0.0006-0.0017mg/l	4
	EC50	96h	Algae or other aquatic plants	0.03-0.058mg/l	4
	EC50	72h	Algae or other aquatic plants	0.011-0.017mg/L	4
	NOEC(ECx)	48h	Fish	0.00009mg/l	4
	LC50	96h	Fish	0.003mg/L	2
APSC Aluminium Foil	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	0.736mg/L	2
	EC50	96h	Algae or other aquatic plants	0.005mg/L	2
	EC50	72h	Algae or other aquatic plants	0.017mg/L	2
	NOEC(ECx)	48h	Crustacea	>100mg/l	1
	LC50	96h	Fish	0.078-0.108mg/l	2
nickel	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	>100mg/l	1
	EC50	96h	Algae or other aquatic plants	0.174-0.311mg/l	4
	EC50	72h	Algae or other aquatic plants	0.18mg/l	1
	EC50(ECx)	72h	Algae or other aquatic plants	0.18mg/l	1
	LC50	96h	Fish	0.06mg/l	4
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 extent, 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.

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 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* larvis embryo gives rise to duplication of dorsoanterior structures such as the notochord, neural tube and eyes.

for cobalt compounds:

Environmental Fate:

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

Between 45 and 100% of dissolved cobalt was found to occur in very strong complexes. The distribution coefficient of cobalt may vary considerably in the same sediment in response to conditions affecting the pH, redox conditions, ionic strength, and amount of dissolved organic matter. Uptake of 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 (*Senenastrum capricornutum*) with concentration factors (dry weight) ranging from 15,000 to 40,000 and 2,300 to 18,000, respectively. Elimination experiments with the algae indicate a two component biological half-life, 1 hour and 11 days, respectively, and suggest that the cobalt might be absorbed not only on the surface, but also intracellularly. Since these organisms are at the bottom of the food chain, they could play an important role in the trophic transfer of 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.

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 crustacea 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 open ocean water to much higher levels in some coastal waters. Freshwater environments have comparable levels of vitamin B12. The high level of cobalamins in coastal water appears to be related to the occurrence of macrophytes in these areas with their high concentrations of vitamin B12. Cobalamins are released into the water when the organisms die.

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

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
	No Data available for all ingredients	No Data available for all ingredients

Bioaccumulative potential

Ingredient	Bioaccumulation

Ingredient	Bioaccumulation
	No Data available for all ingredients


Mobility in soil

Ingredient	Mobility
	No Data available for all ingredients

SECTION 13 Disposal considerations**Waste treatment methods**

Product / Packaging disposal	
	<ul style="list-style-type: none"> ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Management Authority for disposal. ▶ Bury residue in an authorised landfill. ▶ 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	3480	
14.2. UN proper shipping name	LITHIUM ION BATTERIES (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	188 230 310 348 376 377 384 387
	Limited quantity	0

Air transport (ICAO-IATA / DGR)

14.1. UN number	3480	
14.2. UN proper shipping name	Lithium ion batteries (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	A88 A99 A154 A164 A183 A201 A213 A331 A334 A802
	Cargo Only Packing Instructions	See 965
	Cargo Only Maximum Qty / Pack	See 965
	Passenger and Cargo Packing Instructions	Forbidden
	Passenger and Cargo Maximum Qty / Pack	Forbidden
	Passenger and Cargo Limited Quantity Packing Instructions	Forbidden
	Passenger and Cargo Limited Maximum Qty / Pack	Forbidden

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	3480
14.2. UN proper shipping name	LITHIUM ION BATTERIES (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	188 230 310 348 376 377 384 387
	Limited Quantities	0

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

Not Applicable

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

Product name	Group
lithium cobaltate	Not Available
graphite	Not Available
lithium fluorophosphate	Not Available
copper	Not Available
APSC Aluminium Foil	Not Available
nickel	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
lithium cobaltate	Not Available
graphite	Not Available
lithium fluorophosphate	Not Available
copper	Not Available
APSC Aluminium Foil	Not Available
nickel	Not Available

SECTION 15 Regulatory information**Safety, health and environmental regulations / legislation specific for the substance or mixture****lithium cobaltate is found on the following regulatory lists**

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

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)

copper is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
 Australian Inventory of Industrial Chemicals (AIIC)
 International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

APSC Aluminium Foil is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
 Australian Inventory of Industrial Chemicals (AIIC)
 International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

nickel is found on the following regulatory lists

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

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (lithium fluorophosphate)
Canada - NDSL	No (lithium cobaltate; graphite; copper; APSC Aluminium Foil; nickel)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (graphite; lithium fluorophosphate; copper; APSC Aluminium Foil; nickel)
Korea - KECI	Yes
New Zealand - NZIoC	No (lithium fluorophosphate)
Philippines - PICCS	No (lithium cobaltate)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (lithium cobaltate; lithium fluorophosphate)
Vietnam - NCI	Yes
Russia - FBEPH	No (lithium cobaltate; lithium fluorophosphate)
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	26/02/2024
Initial Date	23/02/2024

SDS Version Summary

Version	Date of Update	Sections Updated
3.1	26/02/2024	Toxicological information - Chronic Health, Hazards identification - Classification, Exposure controls / personal protection - Exposure Standard, Toxicological information - Toxicity and Irritation (Other), Identification of the substance / mixture and of the company / undertaking - Use

Other information

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

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

Definitions and abbreviations

- ▶ PC - TWA: Permissible Concentration-Time Weighted Average
- ▶ PC - STEL: Permissible Concentration-Short Term Exposure Limit
- ▶ IARC: International Agency for Research on Cancer
- ▶ ACGIH: American Conference of Governmental Industrial Hygienists
- ▶ STEL: Short Term Exposure Limit
- ▶ TEEL: Temporary Emergency Exposure Limit
- ▶ IDLH: Immediately Dangerous to Life or Health Concentrations
- ▶ ES: Exposure Standard
- ▶ OSF: Odour Safety Factor
- ▶ NOAEL: No Observed Adverse Effect Level
- ▶ LOAEL: Lowest Observed Adverse Effect Level
- ▶ TLV: Threshold Limit Value
- ▶ LOD: Limit Of Detection
- ▶ OTV: Odour Threshold Value
- ▶ BCF: BioConcentration Factors
- ▶ BEI: Biological Exposure Index
- ▶ DNEL: Derived No-Effect Level
- ▶ PNEC: Predicted no-effect concentration

- ▶ AIIC: Australian Inventory of Industrial Chemicals
- ▶ DSL: Domestic Substances List
- ▶ NDSL: Non-Domestic Substances List
- ▶ IECSC: Inventory of Existing Chemical Substance in China
- ▶ EINECS: European Inventory of Existing Commercial chemical Substances
- ▶ ELINCS: European List of Notified Chemical Substances
- ▶ NLP: No-Longer Polymers
- ▶ ENCS: Existing and New Chemical Substances Inventory
- ▶ KECI: Korea Existing Chemicals Inventory
- ▶ NZIoC: New Zealand Inventory of Chemicals
- ▶ PICCS: Philippine Inventory of Chemicals and Chemical Substances
- ▶ TSCA: Toxic Substances Control Act
- ▶ TCSI: Taiwan Chemical Substance Inventory

- ▶ **INSQ:** Inventario Nacional de Sustancias Químicas
- ▶ **NCI:** National Chemical Inventory
- ▶ **FBEPH:** Russian Register of Potentially Hazardous Chemical and Biological Substances

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