



NARVA

PROJECTA

WILDCAT

KICKER

JB

Aerpro

rain-x

KT

12V 1400A PROFESSIONAL JUMP STARTER

Brown & Watson International Pty Ltd

Chemwatch Hazard Alert Code: 4

Chemwatch: 5443-59

Version No: 9.1

Safety Data Sheet (Conforms to Annex II of REACH (1907/2006) - Regulation 2020/878)

Issue Date: 20/10/2023

Print Date: 18/06/2024

L.REACH.NLD.EN.E

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

1.1. Product Identifier

Product name	12V 1400A PROFESSIONAL JUMP STARTER
Chemical Name	Not Applicable
Synonyms	Part Number: IS1400; 12V 900A EMERGENCY JUMP STARTER (Part Number: IS920 & PJ-IS920-2); 12V 1200A EMERGENCY JUMP STARTER (Part Number: IS1220 & PJ-IS1220-2); 12V 1400A PROFESSIONAL JUMP STARTER (Part Number: IS1400 & PJ-IS1400-2); 12/24V 2000A PROFESSIONAL JUMP STARTER (Part Number: IS2000 & PJ-IS2000-2); 12/24V 3000A PROFESSIONAL JUMP STARTER (Part Number: IS3000 & PJ-IS3000-2); 12/24V 5000A PROFESSIONAL JUMP STARTER (Part Number: IS5000 & PJ-IS5000-2); JS900; JS1200
Proper shipping name	LITHIUM ION BATTERIES (including lithium ion polymer batteries) (contains lithium fluorophosphate)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Rechargeable battery. NOTE: Hazard statement relates to battery contents. Potential for exposure should not exist unless the battery leaks, is exposed to high temperatures or is mechanically, physically or electrically abused. Risk of exposure exists only in case of mechanical, electrical or thermal abuse. Thus the batteries should not short circuit, recharge, puncture, incinerate, crush, immerse in water, force discharge, or expose to temperatures above the temperature range of the cell or battery. Use according to manufacturer's directions.
Uses advised against	No specific uses advised against are identified.

1.3. Details of the manufacturer or supplier of the safety data sheet

Registered company name	Brown & Watson International Pty Ltd
Address	1500 Ferntree Gully Road Knoxfield VIC 3180 Australia
Telephone	+61 3 9730 6000
Fax	+61 3 9730 6050
Website	www.narva.com.au & www.projecta.com.au
Email	info@narva.com.au

1.4. Emergency telephone number


Association / Organisation	Australia Poisons Information Centre
Emergency telephone numbers	13 11 26 (All Hours)
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

2.1. Classification of the substance or mixture

Classification according to regulation (EC) No 1272/2008 [CLP] and amendments [1]	H314 - Skin Corrosion/Irritation Category 1B, H411 - Hazardous to the Aquatic Environment Long-Term Hazard Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

2.2. Label elements

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

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

Hazard statement(s)

H314	Causes severe skin burns and eye damage.
H411	Toxic to aquatic life with long lasting effects.

Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P260	Do not breathe dust/fume.
P264	Wash all exposed external body areas thoroughly after handling.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P273	Avoid release to the environment.

Precautionary statement(s) Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P363	Wash contaminated clothing before reuse.
P391	Collect spillage.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.
------	------------------

Precautionary statement(s) Disposal

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

Material contains graphite, lithium fluorophosphate, ethylene carbonate.

2.3. Other hazards

Inhalation may produce severe health damage*.

Cumulative effects may result following exposure*.

Limited evidence of a carcinogenic effect*.

May possibly be harmful to breastfed babies.*

dimethyl carbonate	Listed in the Europe Regulation (EC) No 1907/2006 - Annex XVII (Restrictions may apply)
aluminium	Listed in the Europe Regulation (EC) No 1907/2006 - Annex XVII (Restrictions may apply)

SECTION 3 Composition / information on ingredients

3.1. Substances

See 'Composition on ingredients' in Section 3.2

3.2. Mixtures

1. CAS No 2. EC No 3. Index No 4. REACH No	% [weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP] and amendments	SCL / M-Factor	Nanoform Particle Characteristics
Not Available		Sealed metal containers with electrochemical contents, typically	Not Applicable	Not Applicable	Not Available
1. 15365-14-7 2. 476-700-9 3. Not Available 4. Not Available	28	<u>lithium iron phosphate</u>	Hazardous to the Aquatic Environment Long-Term Hazard Category 4; H413 ^[1]	Not Available Acute M factor: Not Available	Not Available

Continued...

1. CAS No 2. EC No 3. Index No 4. REACH No	% [weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP] and amendments	SCL / M- Factor	Nanoform Particle Characteristics
				Chronic M factor: Not Available	
1. 7440-50-8 2. 231-159-6 3. 029-024-00-X 4. None	13	<u>copper</u>	Hazardous to the Aquatic Environment Long-Term Hazard Category 2; H411 [2]	Not Available Acute M factor: Not Available Chronic M factor: Not Available	Not Available
1. 7782-42-5 2. 231-955-3 3. Not Available 4. Not Available	12	<u>graphite</u>	Serious Eye Damage/Eye Irritation Category 2, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Specific Target Organ Toxicity - Repeated Exposure Category 2; H319, H335, H373 [1]	Not Available Acute M factor: Not Available Chronic M factor: Not Available	Not Available
1. 21324-40-3 2. 244-334-7 3. Not Available 4. Not Available	9	<u>lithium fluorophosphate</u>	Acute Toxicity (Oral) Category 4, Acute Toxicity (Dermal) Category 3, Skin Corrosion/Irritation Category 1B, Serious Eye Damage/Eye Irritation Category 1; H302, H311, H314, H318 [1]	Not Available Acute M factor: Not Available Chronic M factor: Not Available	Not Available
1. 96-49-1 2. 202-510-0 3. Not Available 4. Not Available	9	<u>ethylene carbonate</u>	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3; H315, H318, H335, EUH019 [1]	Not Available Acute M factor: Not Available Chronic M factor: Not Available	Not Available
1. 616-38-6 2. 210-478-4 3. 607-013-00-6 4. Not Available	9	<u>dimethyl carbonate</u>	Flammable Liquids Category 2; H225 [2]	Not Available Acute M factor: Not Available Chronic M factor: Not Available	Not Available
1. 7429-90-5 2. 231-072-3 3. 013-001-00-6[013-002-00-1 4. Not Available	7	<u>aluminium</u>	Pyrophoric Solids Category 1, Substances and Mixtures which in Contact with Water Emit Flammable Gases Category 2; H250, H261 [2]	Not Available Acute M factor: Not Available Chronic M factor: Not Available	Not Available
1. 9003-07-0 2. Not Available 3. Not Available 4. Not Available	5	<u>polypropylene</u>	Not Classified [1]	Not Available Acute M factor: Not Available Chronic M factor: Not Available	Not Available
1. 9002-88-4 2. Not Available 3. Not Available 4. Not Available	5	<u>polyethylene</u>	Not Classified [1]	Not Available Acute M factor: Not Available Chronic M factor: Not Available	Not Available
1. 24937-79-9 2. Not Available 3. Not Available 4. Not Available	2	<u>vinylidene fluoride</u> <u>homopolymer</u>	Not Classified [1]	Not Available Acute M factor: Not Available	Not Available

1. CAS No 2. EC No 3. Index No 4. REACH No	% [weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP] and amendments	SCL / M-Factor	Nanoform Particle Characteristics
				Chronic M factor: Not Available	
1. 9004-32-4 2. Not Available 3. Not Available 4. Not Available	0.5	<u>sodium carboxymethylcellulose</u>	Not Classified ^[1]	Not Available Acute M factor: Not Available Chronic M factor: Not Available	Not Available
Not Available		case material as	Not Applicable	Not Applicable	Not Available
1. 9003-56-9 2. Not Available 3. Not Available 4. Not Available	100	<u>styrene/ butadiene/ acrylonitrile copolymer</u>	EUH032 ^[1]	Not Available Acute M factor: Not Available Chronic M factor: Not Available	Not Available

Legend: 1. Classified by Chemwatch; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 3. Classification drawn from C&L; * EU IOELVs available; [e] Substance identified as having endocrine disrupting properties

SECTION 4 First aid measures

4.1. 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. ▶ Generally not applicable.
Skin Contact	<p>For thermal burns:</p> <ul style="list-style-type: none"> ▶ Decontaminate area around burn. ▶ Consider the use of cold packs and topical antibiotics. <p>For first-degree burns (affecting top layer of skin)</p> <ul style="list-style-type: none"> ▶ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides. ▶ Use compresses if running water is not available. ▶ Cover with sterile non-adhesive bandage or clean cloth. ▶ Do NOT apply butter or ointments; this may cause infection. ▶ Give over-the-counter pain relievers if pain increases or swelling, redness, fever occur. <p>For second-degree burns (affecting top two layers of skin)</p> <ul style="list-style-type: none"> ▶ Cool the burn by immerse in cold running water for 10-15 minutes. ▶ Use compresses if running water is not available. ▶ Do NOT apply ice as this may lower body temperature and cause further damage. ▶ Do NOT break blisters or apply butter or ointments; this may cause infection. ▶ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape. <p>To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort):</p> <ul style="list-style-type: none"> ▶ Lay the person flat. ▶ Elevate feet about 12 inches. ▶ Elevate burn area above heart level, if possible. ▶ Cover the person with coat or blanket. ▶ Seek medical assistance. <p>For third-degree burns Seek immediate medical or emergency assistance.</p> <p>In the mean time:</p> <ul style="list-style-type: none"> ▶ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound. ▶ Separate burned toes and fingers with dry, sterile dressings. ▶ Do not soak burn in water or apply ointments or butter; this may cause infection. ▶ To prevent shock see above. ▶ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. ▶ Have a person with a facial burn sit up. ▶ Check pulse and breathing to monitor for shock until emergency help arrives. ▶ Generally not applicable.
Inhalation	▶ Generally not applicable.
Ingestion	▶ Generally not applicable.

4.2 Most important symptoms and effects, both acute and delayed

See Section 11

4.3. Indication of any immediate medical attention and special treatment needed

Continued...

for phosphate salts intoxication:

- ▶ All treatments should be based on observed signs and symptoms of distress in the patient. Consideration should be given to the possibility that overexposure to materials other than this product may have occurred.
- ▶ Ingestion of large quantities of phosphate salts (over 1.0 grams for an adult) may cause an osmotic catharsis resulting in diarrhoea and probable abdominal cramps. Larger doses such as 4-8 grams will almost certainly cause these effects in everyone. In healthy individuals most of the ingested salt will be excreted in the faeces with the diarrhoea and, thus, not cause any systemic toxicity. Doses greater than 10 grams hypothetically may cause systemic toxicity.
- ▶ Treatment should take into consideration both anionic and cation portion of the molecule.
- ▶ All phosphate salts, except calcium salts, have a hypothetical risk of hypocalcaemia, so calcium levels should be monitored.

Treat symptomatically.

for copper intoxication:

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

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

- ▶ A role for activated charcoals for emesis is, as yet, unproven.
- ▶ In severe poisoning CaNa₂EDTA has been proposed.

[ELLENHORN & BARCELOUX: Medical Toxicology]

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

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

[Ellenhorn and Barceloux: Medical Toxicology]

For acute or short term repeated exposures to ethylene glycol:

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

[Ellenhorn and Barceloux: Medical Toxicology]

It has been suggested that there is a need for establishing a new biological exposure limit before a workshift that is clearly below 100 mmol ethoxy-acetic acids per mole creatinine in morning urine of people occupationally exposed to ethylene glycol ethers. This arises from the finding that an increase in urinary stones may be associated with such exposures.

Laitinen J., et al: *Occupational & Environmental Medicine* 1996; 53, 595-600

SECTION 5 Firefighting measures

5.1. 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.

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.

5.2. Special hazards arising from the substrate or mixture

Fire Incompatibility	<ul style="list-style-type: none"> ▶ Reacts with acids producing flammable / explosive hydrogen (H₂) gas ▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result ▶ Keep dry ▶ NOTE: May develop pressure in containers; open carefully. Vent periodically.
-----------------------------	---

5.3. 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	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.

Decomposes on heating and produces:

- carbon dioxide (CO₂)
- hydrogen fluoride
- nitrogen oxides (NO_x)
- phosphorus oxides (PO_x)
- metal oxides
- other pyrolysis products typical of burning organic material.

SECTION 6 Accidental release measures

6.1. Personal precautions, protective equipment and emergency procedures

See section 8

6.2. Environmental precautions

See section 12

6.3. 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"> ▶ 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. · Do not use compressed air to remove metal dusts from floors, beams or equipment · Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation. · Use non-sparking handling equipment, tools and natural bristle brushes. · Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations · Cover and reseal partially empty containers. · Do not allow chips, fines or dusts to contact water, particularly in enclosed areas. If molten: <ul style="list-style-type: none"> ▶ Contain the flow using dry sand or salt flux as a dam. ▶ All tooling (e.g., shovels or hand tools) and containers which come in contact with molten metal must be preheated or specially coated, rust free and approved for such use. ▶ Allow the spill to cool before remelting scrap. ▶ Clean up all spills immediately. ▶ Wear protective clothing, safety glasses, dust mask, gloves. ▶ Secure load if safe to do so. Bundle/collect recoverable product. ▶ Use dry clean up procedures and avoid generating dust. ▶ Vacuum up (consider explosion-proof machines designed to be grounded during storage and use). ▶ Water may be used to prevent dusting. ▶ Collect remaining material in containers with covers for disposal. ▶ Flush spill area with water. <p>Minor hazard.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Control personal contact with the substance, by using protective equipment as required. ▶ Prevent spillage from entering drains or water ways. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Absorb remaining product with sand, earth or vermiculite and place in appropriate containers for disposal. ▶ Wash area and prevent runoff into drains or waterways. ▶ If contamination of drains or waterways occurs, advise emergency services.

6.4. Reference to other sections

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

SECTION 7 Handling and storage

7.1. Precautions for safe handling

Safe handling	<p>Polyalpaolefin (PAO) dimers require bonding and grounding to prevent static hazards which could cause a fire</p> <p>Graphite:</p> <ul style="list-style-type: none"> · is a good conductor of electricity; avoid contact with electrical circuitry. · is a highly lubricious material and may present a slip hazard if spilled on pedestrian surfaces. <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.
----------------------	--

12V 1400A PROFESSIONAL JUMP STARTER

- ▶ Before entry to such areas, sampling and test procedures for low oxygen levels should be undertaken; control conditions should be established to ensure the availability of adequate oxygen supply.

For molten metals:

- Molten metal and water can be an explosive combination. The risk is greatest when there is sufficient molten metal to entrap or seal off water. Water and other forms of contamination on or contained in scrap or remelt ingot are known to have caused explosions in melting operations. While the products may have minimal surface roughness and internal voids, there remains the possibility of moisture contamination or entrapment. If confined, even a few drops can lead to violent explosions.
 - All tooling, containers, molds and ladles, which come in contact with molten metal must be preheated or specially coated, rust free and approved for such use.
 - Any surfaces that may contact molten metal (e.g. concrete) should be specially coated
 - Drops of molten metal in water (e.g. from plasma arc cutting), while not normally an explosion hazard, can generate enough flammable hydrogen gas to present an explosion hazard. Vigorous circulation of the water and removal of the particles minimise the hazard.
- During melting operations, the following minimum guidelines should be observed:
- Inspect all materials prior to furnace charging and completely remove surface contamination such as water, ice, snow, deposits of grease and oil or other surface contamination resulting from weather exposure, shipment, or storage.
 - Store materials in dry, heated areas with any cracks or cavities pointed downwards.
 - Preheat and dry large objects adequately before charging in to a furnace containing molten metal. This is typically done by the use of a drying oven or homogenising furnace. The dry cycle should bring the metal temperature of the coldest item of the batch to 200 degree C (400 deg F) and then hold at that temperature for 6 hours.
 - Electrostatic discharge may be generated during pumping - this may result in fire.
 - Ensure electrical continuity by bonding and grounding (earthing) all equipment.
 - Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (≤ 1 m/sec until fill pipe submerged to twice its diameter, then ≤ 7 m/sec).
 - Avoid splash filling.
 - Do NOT use compressed air for filling discharging or handling operations.
 - Wait 2 minutes after tank filling (for tanks such as those on road tanker vehicles) before opening hatches or manholes.
 - Wait 30 minutes after tank filling (for large storage tanks) before opening hatches or manholes. Even with proper grounding and bonding, this material can still accumulate an electrostatic charge. If sufficient charge is allowed to accumulate, electrostatic discharge and ignition of flammable air-vapour mixtures can occur. Be aware of handling operations that may give rise to additional hazards that result from the accumulation of static charges. These include but are not limited to pumping (especially turbulent flow), mixing, filtering, splash filling, cleaning and filling of tanks and containers, sampling, switch loading, gauging, vacuum truck operations, and mechanical movements. These activities may lead to static discharge e.g. spark formation. Restrict line velocity during pumping in order to avoid generation of electrostatic discharge ($= 1$ m/s until fill pipe submerged to twice its diameter, then $= 7$ m/s). Avoid splash filling.
 - Do NOT use compressed air for filling, discharging, or handling operations
 - ▶ 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.

Fire and explosion protection	See section 5
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.

7.2. Conditions for safe storage, including any incompatibilities

Suitable container	Generally packaging as originally supplied with the article or manufactured item is sufficient to protect against physical hazards. If repackaging is required ensure the article is intact and does not show signs of wear. As far as is practicably possible, reuse the original packaging or something providing a similar level of protection to both the article and the handler.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Keep dry ▶ NOTE: May develop pressure in containers; open carefully. Vent periodically.
Hazard categories in accordance with Regulation (EC) No 2012/18/EU (Seveso III)	E2: Hazardous to the Aquatic Environment in Category Chronic 2
Qualifying quantity (tonnes) of dangerous substances as referred to in Article 3(10) for the application of	E2 Lower- / Upper-tier requirements: 200 / 500

7.3. Specific end use(s)

See section 1.2

SECTION 8 Exposure controls / personal protection

8.1. Control parameters

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
lithium iron phosphate	Dermal 1 mg/kg bw/day (Systemic, Chronic) Inhalation 4.2 mg/m ³ (Systemic, Chronic)	10 mg/L (STP) 0.33 mg/kg food (Oral)
copper	Dermal 137 mg/kg bw/day (Systemic, Chronic) Dermal 273 mg/kg bw/day (Systemic, Acute) Dermal 137 mg/kg bw/day (Systemic, Chronic) * Dermal 273 mg/kg bw/day (Systemic, Acute) *	6.3 µg/L (Water (Fresh)) 5.2 µg/L (Water (Marine)) 87 mg/kg sediment dw (Sediment (Fresh Water)) 676 mg/kg sediment dw (Sediment (Marine)) 65 mg/kg soil dw (Soil) 230 µg/L (STP)
lithium fluorophosphate	Dermal 133 µg/kg bw/day (Systemic, Chronic) Inhalation 0.931 mg/m ³ (Systemic, Chronic)	0.31 mg/L (Water (Fresh)) 0.68 mg/L (Water - Intermittent release) 0.031 mg/L (Water (Marine)) 7.73 mg/kg sediment dw (Sediment (Fresh Water)) 1.55 mg/kg sediment dw (Sediment (Marine)) 13.5 mg/kg soil dw (Soil) 48 mg/L (STP)
ethylene carbonate	Dermal 4.3 mg/kg bw/day (Systemic, Chronic) Inhalation 15 mg/m ³ (Systemic, Chronic) Dermal 2.1 mg/kg bw/day (Systemic, Chronic) * Inhalation 3.7 mg/m ³ (Systemic, Chronic) * Oral 2.1 mg/kg bw/day (Systemic, Chronic) *	5.9 mg/L (Water (Fresh)) 59 mg/L (Water - Intermittent release) 0.59 mg/L (Water (Marine)) 28.3 mg/kg sediment dw (Sediment (Fresh Water)) 2.83 mg/kg sediment dw (Sediment (Marine)) 2.2 mg/kg soil dw (Soil)
dimethyl carbonate	Dermal 5 mg/kg bw/day (Systemic, Chronic) Inhalation 34.9 mg/m ³ (Systemic, Chronic) Dermal 2.5 mg/kg bw/day (Systemic, Chronic) * Inhalation 8.7 mg/m ³ (Systemic, Chronic) * Oral 2.5 mg/kg bw/day (Systemic, Chronic) *	0.5 mg/L (Water (Fresh)) 1 mg/L (Water - Intermittent release) 0.05 mg/L (Water (Marine)) 188 mg/L (STP)
aluminium	Inhalation 3.72 mg/m ³ (Systemic, Chronic) Inhalation 3.72 mg/m ³ (Local, Chronic) Oral 3.95 mg/kg bw/day (Systemic, Chronic) *	74.9 µg/L (Water (Fresh)) 20 mg/L (STP)

* Values for General Population

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Netherlands Occupational Exposure Limits	copper	Koper en anorganische koperverbindingen (inhaleerbaar)	0.1 mg/m ³	Not Available	Not Available	A
Netherlands Occupational Exposure Limits	lithium fluorophosphate	Fluoriden, anorganisch en oplosbaar (als F)	Not Available	2 mg/m ³	Not Available	A

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
copper	3 mg/m ³	33 mg/m ³	200 mg/m ³
graphite	6 mg/m ³	330 mg/m ³	2,000 mg/m ³
lithium fluorophosphate	7.5 mg/m ³	83 mg/m ³	500 mg/m ³
ethylene carbonate	30 mg/m ³	330 mg/m ³	2,000 mg/m ³
dimethyl carbonate	11 ppm	120 ppm	700 ppm
polypropylene	5.2 mg/m ³	58 mg/m ³	350 mg/m ³
polyethylene	16 mg/m ³	170 mg/m ³	1,000 mg/m ³


Ingredient	Original IDLH	Revised IDLH
lithium iron phosphate	Not Available	Not Available
copper	100 mg/m ³	Not Available
graphite	1,250 mg/m ³	Not Available
lithium fluorophosphate	Not Available	Not Available
ethylene carbonate	Not Available	Not Available
dimethyl carbonate	Not Available	Not Available
aluminium	Not Available	Not Available
polypropylene	Not Available	Not Available
polyethylene	Not Available	Not Available
vinylidene fluoride homopolymer	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
sodium carboxymethylcellulose	Not Available	Not Available
styrene/ butadiene/ acrylonitrile copolymer	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
graphite	E	≤ 0.01 mg/m ³
ethylene carbonate	E	≤ 0.01 mg/m ³
Notes:	<i>Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.</i>	

MATERIAL DATA**8.2. Exposure controls**

8.2.1. Appropriate engineering controls	Articles or manufactured items, in their original condition, generally don't require engineering controls during handling or in normal use. Exceptions may arise following extensive use and subsequent wear, during recycling or disposal operations where substances, found in the article, may be released to the environment.													
	<p>Metal dusts must be collected at the source of generation as they are potentially explosive.</p> <ul style="list-style-type: none"> ▶ Avoid ignition sources. ▶ Good housekeeping practices must be maintained. ▶ Dust accumulation on the floor, ledges and beams can present a risk of ignition, flame propagation and secondary explosions. ▶ Do not use compressed air to remove settled materials from floors, beams or equipment ▶ Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation. ▶ Use non-sparking handling equipment, tools and natural bristle brushes. Cover and reseal partially empty containers. Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations. ▶ Do not allow chips, fines or dusts to contact water, particularly in enclosed areas. ▶ Metal spraying and blasting should, where possible, be conducted in separate rooms. This minimises the risk of supplying oxygen, in the form of metal oxides, to potentially reactive finely divided metals such as aluminium, zinc, magnesium or titanium. ▶ Work-shops designed for metal spraying should possess smooth walls and a minimum of obstructions, such as ledges, on which dust accumulation is possible. ▶ Wet scrubbers are preferable to dry dust collectors. ▶ Bag or filter-type collectors should be sited outside the workrooms and be fitted with explosion relief doors. ▶ Cyclones should be protected against entry of moisture as reactive metal dusts are capable of spontaneous combustion in humid or partially wetted states. ▶ Local exhaust systems must be designed to provide a minimum capture velocity at the fume source, away from the worker, of 0.5 metre/sec. ▶ Local ventilation and vacuum systems must be designed to handle explosive dusts. Dry vacuum and electrostatic precipitators must not be used, unless specifically approved for use with flammable/ explosive dusts. <p>Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p> <table border="1"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>welding, brazing fumes (released at relatively low velocity into moderately still air)</td> <td>0.5-1.0 m/s (100-200 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p> <p>Exhaust ventilation should be designed to prevent accumulation and recirculation in the workplace and safely remove carbon black from the air.</p> <p>Note: Wet, activated carbon removes oxygen from the air and thus presents a severe hazard to workers inside carbon vessels and enclosed or confined spaces. Before entering such areas sampling and test procedures for low oxygen levels should be undertaken and control conditions set up to ensure ample oxygen availability.[Linde]</p>	Type of Contaminant:	Air Speed:	welding, brazing fumes (released at relatively low velocity into moderately still air)	0.5-1.0 m/s (100-200 f/min.)	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use	4: Large hood or large air mass in motion
Type of Contaminant:	Air Speed:													
welding, brazing fumes (released at relatively low velocity into moderately still air)	0.5-1.0 m/s (100-200 f/min.)													
Lower end of the range	Upper end of the range													
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents													
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity													
3: Intermittent, low production.	3: High production, heavy use													
4: Large hood or large air mass in motion	4: Small hood-local control only													
8.2.2. Individual protection measures, such as personal protective equipment														
Eye and face protection	No special equipment required due to the physical form of the product.													
Skin protection	See Hand protection below													
Hands/feet protection	No special equipment required due to the physical form of the product.													
Body protection	See Other protection below													

Other protection

- During repair or maintenance activities the potential exists for exposures to toxic metal particulate in excess of the occupational standards. Under these circumstances, protecting workers can require the use of specific work practices or procedures involving the combined use of ventilation, wet and vacuum cleaning methods, respiratory protection, decontamination, special protective clothing, and when necessary, restricted work zones.
- Protective over-garments or work clothing must be worn by persons who may become contaminated with particulate during activities such as machining, furnace rebuilding, air cleaning equipment filter changes, maintenance, furnace tending, etc. Contaminated work clothing and over-garments must be managed in a controlled manner to prevent secondary exposure to workers of third parties, to prevent the spread of particulate to other areas, and to prevent particulate from being taken home by workers.
- Personnel who handle and work with molten metal should utilise primary protective clothing like polycarbonate face shields, fire resistant tapper's jackets, neck shades (snoods), leggings, spats and similar equipment to prevent burn injuries. In addition to primary protection, secondary or day-to-day work clothing that is fire resistant and sheds metal splash is recommended for use with molten metal. Synthetic materials should never be worn even as secondary clothing (undergarments).

Respiratory protection

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

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

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

^ - Full-face

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

Respiratory protection not normally required due to the physical form of the product.

8.2.3. Environmental exposure controls

See section 12

SECTION 9 Physical and chemical properties**9.1. Information on basic physical and chemical properties**

Appearance	Rectangular shaped battery; insoluble in water. IS920: Nominal Voltage: 12V. Rated Capacity: 1600mAh. Watt Hour: 20.48Wh IS1220: Nominal Voltage: 12V. Rated Capacity: 3000mAh. Watt Hour: 38.4Wh IS1400: Nominal Voltage: 12V. Rated Capacity: 4000mAh. Watt Hour: 51.2Wh IS2000: Nominal Voltage: 12V. Rated Capacity: 6000mAh. Watt Hour: 76.8Wh IS3000: Nominal Voltage: 12V. Rated Capacity: 6000mAh. Watt Hour: 76.8Wh IS5000: Nominal Voltage: 12V. Rated Capacity: 12000mAh. Watt Hour: 153.6Wh
------------	---

Physical state	Manufactured	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable
pH (as supplied)	Not Applicable	Decomposition temperature (°C)	Not Applicable
Melting point / freezing point (°C)	Not Available	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 Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Applicable
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Applicable	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Nanoform Solubility	Not Available	Nanoform Particle Characteristics	Not Available
Particle Size	Not Available		

9.2. Other information

Not Available

SECTION 10 Stability and reactivity

10.1.Reactivity	See section 7.2
10.2. Chemical stability	<ul style="list-style-type: none"> ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable.

Continued...

	▶ Hazardous polymerisation will not occur.
10.3. Possibility of hazardous reactions	See section 7.2
10.4. Conditions to avoid	See section 7.2
10.5. Incompatible materials	See section 7.2
10.6. Hazardous decomposition products	See section 5.3

SECTION 11 Toxicological information

11.1. Information on hazard classes as defined in Regulation (EC) No 1272/2008

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>Acidic corrosives produce respiratory tract irritation with coughing, choking and mucous membrane damage. Symptoms of exposure may include dizziness, headache, nausea and weakness. In more severe exposures, pulmonary oedema may be evident either immediately or after a latent period of 5-72 hours. Symptoms of pulmonary oedema include a tightness in the chest, dyspnoea, frothy sputum and cyanosis. Examination may reveal hypotension, a weak and rapid pulse and moist rates. Death, due to anoxia, may occur several hours after onset of the pulmonary oedema.</p> <p>Exposure to toxic levels of butadiene has also produced chromosome damage. Human volunteers exposed at 2000-8000 ppm 1,3-butadiene for 6-8 hours showed slight smarting of the eyes, difficulty in focusing on instrument scales and a transient objection to butadiene odour. Characteristics of exposure include dry nose/mouth/throat, fatigue, headache, vertigo, nausea, narcosis, respiratory paralysis, and central nervous system depression. Very high concentrations may cause loss of consciousness or death. Repeated and prolonged exposure to 1,3-butadiene vapour may cause kidney and liver damage. Deep anaesthesia was induced in rabbits in 8 to 10 minutes at 200000 to 250000 ppm. Recovery from brief periods of anaesthesia occurred within two minutes of terminating the exposure.</p> <p>Although carbon itself has no toxic action, associated impurities may be toxic. Iodine is often found as an impurity and air-borne carbon dusts, as a result, may produce irritation of the mucous membranes, the eyes, and skin. Symptoms of exposure may include coughing, irritation of the nose and throat and burning of the eyes.</p> <p>Copper poisoning following exposure to copper dusts and fume may result in headache, cold sweat and weak pulse. Capillary, kidney, liver and brain damage are the longer term manifestations of such poisoning. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure.</p> <p>Considered an unlikely route of entry in commercial/industrial environments Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p>
Ingestion	<p>Considered an unlikely route of entry in commercial/industrial environments Accidental ingestion of the material may be damaging to the health of the individual. The material can produce severe chemical burns within the oral cavity and gastrointestinal tract following ingestion.</p>
Skin Contact	<p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. Irritation and skin reactions are possible with sensitive skin Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. Considered an unlikely route of entry in commercial/industrial environments The material can produce severe chemical burns following direct contact with the skin.</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. Considered an unlikely route of entry in commercial/industrial environments The material can produce severe chemical burns to the eye following direct contact. Vapours or mists may be extremely irritating.</p>
Chronic	<p>Repeated or prolonged exposure to acids may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis.</p> <p>The impact of inhaled acidic agents on the respiratory tract depends upon a number of interrelated factors. These include physicochemical characteristics, e.g., gas versus aerosol; particle size (small particles can penetrate deeper into the lung); water solubility (more soluble agents are more likely to be removed in the nose and mouth). Given the general lack of information on the particle size of aerosols involved in occupational exposures to acids, it is difficult to identify their principal deposition site within the respiratory tract. Acid mists containing particles with a diameter of up to a few micrometers will be deposited in both the upper and lower airways. They are irritating to mucous epithelia, they cause dental erosion, and they produce acute effects in the lungs (symptoms and changes in pulmonary function). Asthmatics appear to be at particular risk for pulmonary effects.</p> <p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>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.</p> <p>Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have</p>

been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic. Because aluminium competes with calcium for absorption, increased amounts of dietary aluminium may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies, aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseous tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory. These salts' oestrogen-like effects have led to their classification as a metalloestrogen. Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.

After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus.

Aluminium may persist for a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans.

At high levels of exposure, some aluminium compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminium compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminium potassium sulphate at high levels in the diet.

Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer's disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produce lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, testes, embryotoxicity, and the developing nervous system of 52, 75, 100, and 50 mg aluminium/kg bw/day, respectively. Similarly, the lowest no-observed-adverse-effect levels (NOAELs) for effects on these endpoints were reported at 30, 27, 100, and for effects on the developing nervous system, between 10 and 42 mg aluminium/kg bw per day, respectively.

Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 mg/l aluminium compared with communities where the aluminium level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminium exposure to brain disease. Aluminium concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramid-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations.

Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of "tau" a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminium hyperphosphorylates tau in vitro. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade. Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA).

Aluminium enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminium include baking powder, antacids and aluminium products used for general food preparation and storage (over 12 months, aluminium levels in soft drink packed in aluminium cans rose from 0.05 to 0.9 mg/l). [Walton, J and Bryson-Taylor, D. - *Chemistry in Australia, August 1995*]

the main target organs of aluminum are the central nervous system and bone. Aluminum binds with dietary phosphorus and impairs gastrointestinal absorption of phosphorus. The decreased phosphate body burden results in osteomalacia (softening of the bones due to defective bone mineralization) and rickets. Aluminum's neurotoxicity is believed to involve several mechanisms. Changes in cytoskeletal protein functions as a results of altered phosphorylation, proteolysis, transport, and synthesis are believed to be one cause. Aluminum may induce neurobehavioral effects by affecting permeability of the blood-brain barrier, cholinergic activity, signal transduction pathways, lipid peroxidation, and impair neuronal glutamate nitric oxide-cyclic GMP pathway, as well as interfere with metabolism of essential trace elements because of similar coordination chemistries and consequent competitive interactions. It has been suggested that aluminum's interaction with estrogen receptors, but studies have not been able to establish a clear link between aluminum and increased risk of breast cancer). Certain aluminum salts induce immune responses by activating inflammasomes.

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

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

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

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

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

Lithium ions interfere with ion transport processes (involving the "sodium pump") that relay and amplify messages carried to the cells of the brain. Mania is associated with irregular increases in protein kinase C (PKC) activity within the brain. Lithium carbonate and sodium valproate, another drug traditionally used to treat the disorder, act in the brain by inhibiting PKC's activity and help to produce other compounds that also inhibit the PKC.

Taking lithium salts has risks and side effects. Extended use of lithium to treat various mental disorders has been known to lead to acquired nephrogenic diabetes insipidus. Nephrogenic diabetes insipidus (NDI), also known as renal diabetes insipidus, is a form of diabetes insipidus

primarily due to pathology of the kidney. This is in contrast to central or neurogenic diabetes insipidus, which is caused by insufficient levels of antidiuretic hormone (ADH, also called vasopressin). Nephrogenic diabetes insipidus is caused by an improper response of the kidney to ADH, leading to a decrease in the ability of the kidney to concentrate the urine by removing free water.

Lithium intoxication can affect the central nervous system and renal system and can be lethal

In subchronic studies, rats were exposed to 3 milliequivalents Li/kg/day (equivalent to 1450 mg for a 70 kg person) but did not accumulate Li whilst on a high sodium diet. However when sodium was restricted, fatal kidney toxicity developed. Dogs survived daily dose of 50 mg LiCl/kg for 150 days to the termination of the experiment on a normal sodium intake, whereas the same dose was lethal in 12 to 18 days on a low sodium diet: 20 mg LiCl/kg/day resulted in death in 18 to 30 days.

Several reports have demonstrated that lithium may impair basal ganglia activity. Lithium intoxication has been associated, severe and persistent oculogyric crises. Oculogyric crisis (OGC) is the name of a dystonic reaction to certain drugs or medical conditions characterized by a prolonged involuntary upward deviation of the eyes. The term "oculogyric" refers to the bilateral elevation of the visual gaze but several other responses are associated with the crisis.

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

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

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

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

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

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

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 polyaromatic hydrocarbons (PAHs) (as benzene extractables) whilst furnace black had 0.28%.

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

Carbon black appears to act like PSLT particles, which can elicit lung tumours in rats following prolonged exposure to sufficiently high concentrations of particles. Particle surface area dose was found to be most predictive of pulmonary inflammation and tumour response in rats when comparing the dose-response relationships for various types and sizes of PSLT including carbon black. Compared to fine PSLT, much lower concentrations of ultrafine PSLT (e.g. 2.5, 6.5 or 11.5 mg/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 electrocardiac changes indicative of right atrial and right ventricular strain. These changes increased progressively until after 10,000 hours of exposure, when the changes were marked. The authors of this study concluded that there was no significant effect due to prolonged exposure other than those expected from the accumulation of non-toxic dusts in the pulmonary system. Exposure to furnace black produced a similar picture although electrocardiographic change was first observed in monkeys after 2500 hours' exposure and marked atrial and right ventricular strain after 10,000 hours' exposure. The authors concluded that there was no significant effect due to prolonged exposure other than those expected from the accumulation of nontoxic dusts in the pulmonary system. Exposure to furnace black produced a similar picture although electrocardiographic change was first observed in monkeys after 2500 hours exposure and marked atrial and right ventricular strain after 10,000 hours exposure.

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

For copper and its compounds (typically copper chloride):

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

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

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

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

Amongst humans occupationally exposed to 1,3-butadiene several cancer sites with high statistically significant mortality ratios were identified.

These included cancer of the testes, cancers of the digestive system (oesophagus, stomach, large intestine), larynx and Hodgkin's disease. Exposure by rats to 1,3-butadiene gas at 1000 ppm/6hrs/day, 5 days/week (105 weeks for females and 111 weeks for males) caused significant increases in the incidence of tumours at various sites; mammary gland adenomas and sarcomas; uterine sarcomas; Zymbal gland carcinomas; thyroid adenomas and pancreatic adenomas. A high incidence of malignant lymphoma was found amongst a group of exposed rats in a second study

Dogs given daily doses of sodium phosphate dibasic for 9-22 weeks showed calcium deposits in the kidneys (nephrocalcinosis) with disseminated atrophy of the proximal tubule. Animals fed on sodium phosphate dibasic and potassium dihydrogen phosphate, in both short- and long-term studies, showed increased bone porosity; hyperparathyroidism and soft tissue calcification were also evident.

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

Chronic excessive iron exposure has been associated with haemosiderosis and consequent possible damage to the liver and pancreas. Haemosiderin is a golden-brown insoluble protein produced by phagocytic digestion of haematin (an iron-based pigment). Haemosiderin is found in most tissues, especially in the liver, in the form of granules. Other sites of haemosiderin deposition include the pancreas and skin. A related condition, haemochromatosis, which involves a disorder of metabolism of these deposits, may produce cirrhosis of the liver, diabetes, and bronze pigmentation of the skin - heart failure may eventually occur.

Such exposure may also produce conjunctivitis, choroiditis, retinitis (both inflammatory conditions involving the eye) and siderosis of tissues if iron remains in these tissues. Siderosis is a form of pneumoconiosis produced by iron dusts. Siderosis also includes discoloration of organs, excess circulating iron and degeneration of the retina, lens and uvea as a result of the deposition of intraocular iron. Siderosis might also involve the lungs - involvement rarely develops before ten years of regular exposure. Often there is an accompanying inflammatory reaction of the bronchi. Permanent scarring of the lungs does not normally occur.

High levels of iron may raise the risk of cancer. This concern stems from the theory that iron causes oxidative damage to tissues and organs by generating highly reactive chemicals, called free radicals, which subsequently react with DNA. Cells may be disrupted and may become cancerous. People whose genetic disposition prevents them from keeping tight control over iron (e.g. those with the inherited disorder, haemochromatosis) may be at increased risk.

Iron overload in men may lead to diabetes, arthritis, liver cancer, heart irregularities and problems with other organs as iron builds up. [K. Schmidt, New Scientist, No. 1919 pp.11-12, 2nd April, 1994]

12V 1400A PROFESSIONAL JUMP STARTER	TOXICITY	IRRITATION
	Not Available	Not Available
lithium iron phosphate	TOXICITY	IRRITATION
	dermal (rat) LD50: 2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (Rat) LC50: >3.2 mg/l4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >2000 mg/kg ^[1]	
copper	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (Rat) LC50: 0.733 mg/l4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Mouse) LD50: 0.7 mg/kg ^[2]	
graphite	TOXICITY	IRRITATION
	Inhalation (Rat) LC50: >2 mg/L4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >200 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
lithium fluorophosphate	TOXICITY	IRRITATION
	Oral (Rat) LD50: 50-300 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
		Skin: adverse effect observed (corrosive) ^[1]
ethylene carbonate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 20 mg - mild [CCInfo]*
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit): 660 mg - moderate
		Skin: no adverse effect observed (not irritating) ^[1]
dimethyl carbonate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (Rat) LC50: >5.36 mg/l4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >5000 mg/kg ^[1]	
aluminium	TOXICITY	IRRITATION
	Inhalation (Rat) LC50: >2.3 mg/l4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
polypropylene	TOXICITY	IRRITATION
	Oral (Mouse) LD50: 3200 mg/kg ^[2]	Not Available
polyethylene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Not Available
	Oral (Rat) LD50: >2000 mg/kg ^[1]	
vinylidene fluoride homopolymer	TOXICITY	IRRITATION
	Not Available	Not Available

12V 1400A PROFESSIONAL JUMP STARTER

sodium carboxymethylcellulose	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Not Available
	Inhalation (Rat) LC50: >5.8 mg/L4h ^[2]	
	Oral (Guinea) LD50; 16000 mg/kg ^[2]	
styrene/ butadiene/ acrylonitrile copolymer	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 5010 mg/kg ^[2]	Not Available
	Oral (Rat) LD50: 5010 mg/kg ^[2]	

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 IRON PHOSPHATE	<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.
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. 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. for copper and its compounds (typically copper chloride): 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. 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. 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. 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. Carcinogenicity: there was insufficient information to evaluate the carcinogenic activity of copper monochloride. 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>
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 a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. for ethylene carbonate 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. 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>

Ethylene carbonate was mixed in the diet of 26 male and 26 female Crl: CD(SD) rats for 18 months at concentrations of 25,000 ppm for males and females and 50,000 ppm for females; males were also fed 50,000 ppm for 42 weeks, and 40,000 ppm for 16 weeks. Survivors were observed to 24 months. Compound intake (mg/kg/day) was not reported, but is estimated to be approximately 250 and 500 mg/kg/day. No toxic effects were found in females, but increased mortality was seen in males at both dose levels. No high-dose males survived week 60 and only 10 low-dose males survived to week 78. Males had severe nephrotoxicity, characteristic of ethylene glycol toxicity.

The following *in vitro* 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 *in vivo* genotoxicity studies on ethylene carbonate were found; however, ethylene glycol has been tested and was negative in a rat dominant lethal assay.

Gavage administration of ethylene carbonate to pregnant rats days 6-15 of gestation resulted in systemic toxicity at doses of 3000 mg/kg/day, including post-dose salivation. The NOAEL for maternal toxicity was 1500 mg/kg/day. Similar to ethylene glycol, there were increased soft tissue (hydrocephalus, umbilical herniation, gastroschisis, cleft palate, misshapen and compressed stomach) and skeletal malformations at 3000 mg/kg/day, but not at 1500 mg/kg/day.

For ethylene glycol:

Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO₂, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO₂, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

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

Cardiovascular Effects. Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12-24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol.

Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases. Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.

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

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

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

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

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

Neurological Effects: Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion.

Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months.

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

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

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

Genotoxic Effects: Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available *in vivo* and *in vitro* laboratory studies provide consistently negative genotoxicity results for ethylene glycol.

POLYETHYLENE	polyethylene pyrolyzate Inclusion of polyethylene in the diet of rats at 8 g/kg/day did not result in treatment-related effects. Polyethylene implanted into rats and mice has reportedly caused local tumorigenic activity at doses of 33 to 2120 mg/kg, but the relevance to human exposure is not certain.
SODIUM CARBOXYMETHYLCELLULOSE	Neoplastic by RTECS criteria While thought to be uncommon, case reports of severe reactions to carboxymethylcellulose exist. In one such instance, a woman was known to experience anaphylaxis following exposure. Skin testing is believed to be a useful diagnostic tool for this purpose. Effects on inflammation, microbiota-related metabolic syndrome, and colitis are a subject of research Carboxymethyl cellulose has been found to cause inflammation of the gut, altering microbiota, and was found to be a triggering factor of inflammatory bowel diseases such as ulcerative colitis and Crohn's disease
STYRENE/ BUTADIENE/ ACRYLONITRILE COPOLYMER	Ultrafine particles (UFPs) may be produced at lower temperatures during the 3D printing process Concerns have been raised regarding airborne UFP concentrations generated while printing with ABS, as UFPs have been linked with adverse health effects
LITHIUM IRON PHOSPHATE & GRAPHITE & LITHIUM FLUOROPHOSPHATE & ALUMINIUM & VINYLIDENE FLUORIDE HOMOPOLYMER	No significant acute toxicological data identified in literature search.
GRAPHITE & LITHIUM FLUOROPHOSPHATE & ETHYLENE CARBONATE	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.
POLYPROPYLENE & POLYETHYLENE	<p>For poly-alpha-olefins (PAOs):</p> <p>PAOs are highly branched isoparaffinic chemicals produced by oligomerisation of 1-octene, 1-decene, and/or 1-dodecene. The crude polyalphaolefin mixture is then distilled into appropriate product fractions to meet specific viscosity specifications and hydrogenated. Read across data exist for health effects endpoints from the following similar hydrogenated long chain branched alkanes derived from a C8, C10, and/or C12 alpha olefins:</p> <ul style="list-style-type: none"> ▸ Decene homopolymer ▸ Decene/dodecene copolymer ▸ Octene/decene/dodecene copolymer ▸ Dodecene trimer <p>The data for these structural analogs demonstrated no evidence of health effects. In addition, there is evidence in the literature that alkanes with 30 or more carbon atoms are unlikely to be absorbed when administered orally. The physicochemical data suggest that it is unlikely that significant absorption will occur. If a substance of the size and structure of a typical PAO is absorbed, then the principal mechanisms of absorption after oral administration are likely to be passive diffusion and absorption by way of the lymphatic system. The former requires both good lipid solubility and good water solubility as the substance has to partition from an aqueous environment through a lipophilic membrane into another aqueous environment during absorption. Absorption by way of the lymphatics occurs by mechanisms analogous to those that absorb fatty acids and is limited by the size of the molecule. Lipophilicity generally enhances the ability of chemicals to cross biological membranes. Biotransformation by mixed function oxidases often increases the water solubility of a substance; however, existing data suggest that these substances will not undergo oxidation to more hydrophilic metabolites. Finally, a chemical must have an active functional group that can interact chemically or physically with the target cell or receptor upon reaching it; there are no moieties in PAOs that represent a functional group that may have biological activity. The water solubilities of a C10 dimer PAO and a C12 trimer PAO were determined to be <1 ppb and < 1 ppt respectively. The partition coefficient for a C12 trimer PAO was determined to be log Kow of >7 . Given the very low water solubility it is extremely unlikely that PAOs will be absorbed by passive diffusion following oral administration, and the size of the molecules suggest that the extent of lymphatic absorption is likely to be very low. Although PAOs are relatively large lipophilic compounds, and molecular size may be a critical limiting determinant for absorption, there is some evidence that these substances are absorbed. However, the lack of observed toxicity in the studies with PAOs suggests that these products are absorbed poorly, if at all. Furthermore, a review of the literature regarding the absorption and metabolism of long chain alkanes indicates that alkanes with 30+ carbon atoms are unlikely to be absorbed. For example the absorption of squalane, an analogous C30 product, administered orally to male CD rats was examined - essentially all of the squalane was recovered unchanged in the faeces. At the same time, the hydrophobic properties of PAOs suggest that, should they be absorbed, they would undergo limited distribution in the aqueous systemic circulation and reach potential target organs in limited concentrations.</p> <p>In addition to the general considerations discussed above, the low volatility of PAOs indicates that, under normal conditions of use or transportation, exposure by the inhalation route is unlikely. In particular, the high viscosity of these substances suggests that it would be difficult to generate a high concentration of respirable particles in the air.</p> <p>Acute toxicity: PAOs (decene/dodecene copolymer, octene/decene/dodecene homo-polymer, and dodecene trimer) have been adequately tested for acute oral toxicity. There were no deaths when the test materials were administered at doses of 5,000 mg/kg (decene/dodecene copolymer and dodecene trimer) and at 2,000 mg/kg (octene/decene/dodecene copolymer) in rats. Overall, the acute oral LD50 for these substances was greater than the 2000 mg/kg limit dose, indicating a relatively low order of toxicity.</p> <p>PAOs (decene/dodecene copolymer, octene/decene/dodecene copolymer, and dodecene trimer) have been tested for acute dermal toxicity. No mortality was observed for any substance when administered at the limit dose of 2000 or 5000 mg/kg. Overall, the acute dermal LD50 for these substances was greater than the 2000 mg/kg limit dose, indicating a relatively low order of toxicity.</p> <p>1-Decene, homopolymer, is absorbed (unexpectedly for a high molecular weight polymer) to a moderate degree in rat skin and is eliminated slowly</p> <p>PAOs (decene homopolymer, decene/dodecene copolymer, and decene trimer) have been tested for acute inhalation toxicity. Rats were exposed to aerosols of the substances at nominal atmospheric concentrations of 2.5, 5.0, and 5.06 mg/L, respectively, for four hours. These levels were the maximum attainable concentrations under the conditions of the tests, due to the low volatility and high viscosity of the test material. No mortality was noted, and all animals fully recovered following depuration. The lack of mortality at concentrations at or above the limit dose of 2.0 mg/L indicates a relatively low order of toxicity for these substances.</p> <p>Repeat dose toxicity: Eight repeated-dose toxicity studies using two different animal species, rats and mice, and oral and dermal routes of administration have been conducted with three structural analogs. These data suggest that the structural analogs exhibit a low order of toxicity following repeated applications, due to their similarity in chemical structures and physicochemical properties.</p> <p>One 28-day oral toxicity study in rats, one 90-day dermal and two 90-day dietary studies in rats, and a dermal carcinogenicity study in mice exist for decene homopolymer. A rat oral combined reproductive toxicity and 91-day systemic toxicity study was also conducted with decene homopolymer. In addition, 28-day rat oral toxicity studies exist for two structurally analogous substances (dodecene trimer and octene/decene/dodecene copolymer); and a 90-day rat dermal toxicity study exists for octene/decene/dodecene copolymer. Results from these studies show a low order of repeated dose toxicity. The dermal NOAEL for systemic toxicity studies was equal to or greater than 2000 mg/kg/day.</p> <p>The oral NOAEL for 1-decene homopolymer is between 5,000 and 20,000 mg/kg/day in Sprague-Dawley rats.</p> <p>Rats exposed repeatedly by dermal exposure at doses of 2000 mg/kg decene/dodecene copolymer showed increased incidences of hyperplasia of the sebaceous glands, hyperplasia/hyperkeratosis of the epidermis and dermal inflammation. These symptoms generally subsided within 2 weeks. Males showed decreased body weight gain and altered serum chemistry.</p>

In a 90-day feeding study rats receiving 20000 ppm of 1-decene, homopolymer, hydrogenated did not exhibit any clinical signs of systemic toxicity. Marginal effects on clinical chemistry (glucose and ALT in males; sodium, phosphorus and calcium in females) were seen.

Reproductive toxicity: Data are available for decene homopolymer. Results from these studies show a low order of reproductive/developmental toxicity. The NOAEL for reproductive toxicity was 1000 mg/kg/day, the highest concentration tested. The lack of effects on fertility in this study or effects on reproductive organs in this or other subchronic studies with closely related chemicals indicates that PAOs are unlikely to exert effects on reproduction.

Developmental toxicity: Decene homopolymer (with 10 ppm of an antioxidant) was administered once daily on gestation days 0-19 via dermal application to presumed-pregnant rats at doses of 0, 800, and 2000 mg/kg/day. Dermal administration of the test material did not adversely affect parameters of reproductive performance during gestation, nor did it adversely affect in utero survival and development of the offspring. The NOAEL in this study for developmental parameters was 2000 mg/kg/day.

Genotoxicity: Information for the following PAOs (decene homopolymer, octene/decene/dodecene copolymer, dodecene trimer; and decene/dodecene copolymer [prepared from 10% C12 and 90% C10 alpha olefins; approx. 33% trimer and 51% tetramer, 16% pentamer and higher]) is available. Either bacterial or mammalian gene mutation assays, in vitro chromosomal aberration assays, or in vivo chromosomal aberration assays have been conducted for these substances. Neither mutagenicity nor clastogenicity were exhibited by any of these substances in the referenced in vivo or in vitro tests, with or without metabolic activation.

Carcinogenicity: While alpha-olefin polymers have similar properties to mineral oils, they do not contain polycyclic aromatic hydrocarbons, or other known possible carcinogens.

Decene homopolymer produced no treatment-related tumors in C3H mice treated with a 50 ul/application twice weekly for 104 weeks. In addition, survival (56%) was greater than in any other group, including the untreated control.

POLYPROPYLENE & POLYETHYLENE & STYRENE/ BUTADIENE/ ACRYLONITRILE COPOLYMER

The substance is classified by IARC as Group 3:
NOT classifiable as to its carcinogenicity to humans.
 Evidence of carcinogenicity may be inadequate or limited in animal testing.

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

11.2 Information on other hazards

11.2.1. Endocrine disrupting properties

No evidence of endocrine disrupting properties were found in the current literature.

11.2.2. Other information

See Section 11.1

SECTION 12 Ecological information

12.1. Toxicity

12V 1400A PROFESSIONAL JUMP STARTER	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
lithium iron phosphate	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	>28mg/l	2
	EC50	72h	Algae or other aquatic plants	>24mg/l	2
	EC50	48h	Crustacea	>28mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	>=24mg/l	2
copper	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	48h	Fish	<0.001mg/L	4
	EC50	72h	Algae or other aquatic plants	0.011-0.017mg/L	4
	EC50	96h	Algae or other aquatic plants	0.03-0.058mg/l	4
	EC50	48h	Crustacea	<0.001mg/L	4
	LC50	96h	Fish	0.003mg/L	2
graphite	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	96h	Fish	>=100mg/l	2
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	LC50	96h	Fish	>100mg/l	2

	Endpoint	Test Duration (hr)	Species	Value	Source
lithium fluorophosphate	NOEC(ECx)	528h	Fish	0.2mg/l	2
	EC50	72h	Algae or other aquatic plants	62mg/l	2
	LC50	96h	Fish	42mg/l	2
	EC50	96h	Algae or other aquatic plants	43mg/l	2
	EC50	48h	Crustacea	98mg/l	2
ethylene carbonate	NOEC(ECx)	72h	Algae or other aquatic plants	100mg/l	2
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	LC50	96h	Fish	>100mg/l	2
dimethyl carbonate	LC50	96h	Fish	>=100mg/l	2
	NOEC(ECx)	504h	Crustacea	25mg/l	2
	EC50	72h	Algae or other aquatic plants	>57.29mg/l	2
	EC50	96h	Algae or other aquatic plants	166.6-211mg/l	2
	EC50	48h	Crustacea	>74.16mg/l	2
aluminium	NOEC(ECx)	72h	Algae or other aquatic plants	>100mg/l	1
	EC50	72h	Algae or other aquatic plants	0.017mg/L	2
	EC50	96h	Algae or other aquatic plants	0.005mg/L	2
	EC50	48h	Crustacea	0.736mg/L	2
	LC50	96h	Fish	0.078-0.108mg/l	2
polypropylene	Not Available	Not Available	Not Available	Not Available	Not Available
polyethylene	Not Available	Not Available	Not Available	Not Available	Not Available
vinylidene fluoride homopolymer	Not Available	Not Available	Not Available	Not Available	Not Available
sodium carboxymethylcellulose	EC50(ECx)	48h	Crustacea	46.04-165.37mg/l	4
	EC50	48h	Crustacea	46.04-165.37mg/l	4
	LC50	96h	Fish	>20000mg/L	4
styrene/ butadiene/ acrylonitrile copolymer	Not Available	Not Available	Not Available	Not Available	Not Available
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				

May cause long-term adverse effects in the aquatic environment.

DO NOT discharge into sewer or waterways.

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ethylene carbonate	HIGH	HIGH
dimethyl carbonate	HIGH	HIGH
polypropylene	LOW	LOW
polyethylene	LOW	LOW
vinylidene fluoride	LOW	LOW

Continued...

Ingredient	Persistence: Water/Soil	Persistence: Air
homopolymer		

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
ethylene carbonate	LOW (LogKOW = -0.3388)
dimethyl carbonate	LOW (LogKOW = 0.2336)
polypropylene	LOW (LogKOW = 1.6783)
polyethylene	LOW (LogKOW = 1.2658)
vinylidene fluoride homopolymer	LOW (LogKOW = 1.24)

12.4. Mobility in soil

Ingredient	Mobility
ethylene carbonate	LOW (Log KOC = 9.168)
dimethyl carbonate	LOW (Log KOC = 8.254)
polypropylene	LOW (Log KOC = 23.74)
polyethylene	LOW (Log KOC = 14.3)
vinylidene fluoride homopolymer	LOW (Log KOC = 35.04)

12.5. Results of PBT and vPvB assessment

	P	B	T
Relevant available data	Not Available	Not Available	Not Available
PBT	✗	✗	✗
vPvB	✗	✗	✗
PBT Criteria fulfilled?	No		
vPvB	No		

12.6. Endocrine disrupting properties

No evidence of endocrine disrupting properties were found in the current literature.



12.7. Other adverse effects

No evidence of ozone depleting properties were found in the current literature.

SECTION 13 Disposal considerations**13.1. 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. ▶ 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.
Waste treatment options	Not Available
Sewage disposal options	Not Available

SECTION 14 Transport information**Labels Required**

	
Marine Pollutant	

Land transport (ADR-RID)

Continued...

14.1. UN number or ID number	3480	
14.2. UN proper shipping name	LITHIUM ION BATTERIES (including lithium ion polymer batteries) (contains lithium fluorophosphate)	
14.3. Transport hazard class(es)	Class	9
	Subsidiary Hazard	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Hazard identification (Kemler)	Not Applicable
	Classification code	M4
	Hazard Label	9A
	Special provisions	188 230 310 348 376 377 387 636
	Limited quantity	0
	Tunnel Restriction Code	E

Air transport (ICAO-IATA / DGR)

14.1. UN number	3480	
14.2. UN proper shipping name	Lithium ion batteries (including lithium ion polymer batteries) (contains lithium fluorophosphate)	
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	Environmentally hazardous	
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) (contains lithium fluorophosphate)	
14.3. Transport hazard class(es)	IMDG Class	9
	IMDG Subsidiary Hazard	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Marine Pollutant	
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

Inland waterways transport (ADN)

14.1. UN number	3480	
14.2. UN proper shipping name	LITHIUM ION BATTERIES (including lithium ion polymer batteries) (contains lithium fluorophosphate)	
14.3. Transport hazard class(es)	9	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Classification code	M4
	Special provisions	188; 230; 310; 348; 376; 377; 387; 636
	Limited quantity	0

Equipment required	PP
Fire cones number	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 iron phosphate	Not Available
copper	Not Available
graphite	Not Available
lithium fluorophosphate	Not Available
ethylene carbonate	Not Available
dimethyl carbonate	Not Available
aluminium	Not Available
polypropylene	Not Available
polyethylene	Not Available
vinylidene fluoride homopolymer	Not Available
sodium carboxymethylcellulose	Not Available
styrene/ butadiene/ acrylonitrile copolymer	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
lithium iron phosphate	Not Available
copper	Not Available
graphite	Not Available
lithium fluorophosphate	Not Available
ethylene carbonate	Not Available
dimethyl carbonate	Not Available
aluminium	Not Available
polypropylene	Not Available
polyethylene	Not Available
vinylidene fluoride homopolymer	Not Available
sodium carboxymethylcellulose	Not Available
styrene/ butadiene/ acrylonitrile copolymer	Not Available

SECTION 15 Regulatory information

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

lithium iron phosphate is found on the following regulatory lists

Europe EC Inventory

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

copper is found on the following regulatory lists

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

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

Netherlands Occupational Exposure Limits

graphite is found on the following regulatory lists

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

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

lithium fluorophosphate is found on the following regulatory lists

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

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

Netherlands Occupational Exposure Limits

ethylene carbonate is found on the following regulatory lists

EU European Chemicals Agency (ECHA) Community Rolling Action Plan (CoRAP) List of Substances

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

dimethyl carbonate is found on the following regulatory lists

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

aluminium is found on the following regulatory lists

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

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

polypropylene 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 - Not Classified as Carcinogenic

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

polyethylene is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

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

vinylidene fluoride homopolymer is found on the following regulatory lists

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

sodium carboxymethylcellulose is found on the following regulatory lists

Not Applicable

styrene/ butadiene/ acrylonitrile copolymer is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

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

Additional Regulatory Information

Not Applicable

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : Directives 98/24/EC, - 92/85/EEC, - 94/33/EC, - 2008/98/EC, - 2010/75/EU; Commission Regulation (EU) 2020/878; Regulation (EC) No 1272/2008 as updated through ATPs.

Information according to 2012/18/EU (Seveso III):

Seveso Category	E2

15.2. Chemical safety assessment

No Chemical Safety Assessment has been carried out for this substance/mixture by the supplier.

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (lithium iron phosphate)
Canada - DSL	No (lithium fluorophosphate)
Canada - NDSL	No (lithium iron phosphate; copper; graphite; ethylene carbonate; dimethyl carbonate; aluminium; polypropylene; polyethylene; vinylidene fluoride homopolymer; sodium carboxymethylcellulose; styrene/ butadiene/ acrylonitrile copolymer)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (polypropylene; polyethylene; vinylidene fluoride homopolymer; sodium carboxymethylcellulose; styrene/ butadiene/ acrylonitrile copolymer)
Japan - ENCS	No (copper; graphite; lithium fluorophosphate; aluminium)
Korea - KECI	Yes
New Zealand - NZIoC	No (lithium iron phosphate; lithium fluorophosphate)
Philippines - PICCS	No (lithium iron phosphate)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (lithium iron phosphate; lithium fluorophosphate; ethylene carbonate; vinylidene fluoride homopolymer)
Vietnam - NCI	Yes
Russia - FBEPH	No (lithium iron phosphate; lithium fluorophosphate)
Legend:	Yes = All CAS declared ingredients are on the inventory

Continued...

National Inventory	Status
	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	20/10/2023
Initial Date	22/12/2020

Full text Risk and Hazard codes

H225	Highly flammable liquid and vapour.
H250	Catches fire spontaneously if exposed to air.
H261	In contact with water releases flammable gases.
H302	Harmful if swallowed.
H311	Toxic in contact with skin.
H315	Causes skin irritation.
H318	Causes serious eye damage.
H319	Causes serious eye irritation.
H335	May cause respiratory irritation.
H373	May cause damage to organs through prolonged or repeated exposure.
H413	May cause long lasting harmful effects to aquatic life.

SDS Version Summary

Version	Date of Update	Sections Updated
8.1	25/08/2023	Identification of the substance / mixture and of the company / undertaking - Synonyms
9.1	20/10/2023	Hazards identification - Classification

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.

For detailed advice on Personal Protective Equipment, refer to the following EU CEN Standards:

EN 166 Personal eye-protection

EN 340 Protective clothing

EN 374 Protective gloves against chemicals and micro-organisms

EN 13832 Footwear protecting against chemicals

EN 133 Respiratory protective devices

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

Classification and procedure used to derive the classification for mixtures according to Regulation (EC) 1272/2008 [CLP]

Classification according to regulation (EC) No 1272/2008 [CLP] and amendments	Classification Procedure
Skin Corrosion/Irritation Category 1B, H314	Calculation method
Hazardous to the Aquatic Environment Long-Term Hazard Category 2, H411	Expert judgement

This document is copyright.

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

TEL (+61 3) 9572 4700.

