Li-ion polymer battery in mouthpiece
SDI (North America) Inc.

Version No: **3.1.1.1**
Safety Data Sheet according to OSHA HazCom Standard (2012) requirements

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L.GHS.USA.EN

SECTION 1 Identification

Product Identifier

Product name	Li-ion polymer battery in mouthpiece
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	Lithium ion batteries packed with equipment including lithium ion polymer batteries
Chemical formula	Not Applicable
Other means of identification	Not Available

Recommended use of the chemical and restrictions on use

Relevant identified uses	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. Use according to manufacturer's directions.

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

Registered company name	**SDI (North America) Inc.**	**SDI Limited**	**SDI Germany GmbH**
Address	1279 Hamilton Parkway Itasca IL 60143 United States	3-15 Brunsdon Street Bayswater VIC 3153 Australia	Hansestrasse 85 Cologne D-51149 Germany
Telephone	+1 630 361 9200	+61 3 8727 7111	+49 0 2203 9255 0
Fax	Not Available	+61 3 8727 7222	+49 0 2203 9255 200
Website	Not Available	www.sdi.com.au	www.sdi.com.au
Email	Not Available	info@sdi.com.au	germany@sdi.com.au

Registered company name	**SDI Brazil Industria E Comercio Ltda**
Address	Rua Dr. Virgilio de Carvalho Pinto, 612 São Paulo CEP 05415-020 Brazil
Telephone	+55 11 3092 7100
Fax	+55 11 3092 7101
Website	www.sdi.com.au
Email	brasil@sdi.com.au

Emergency phone number

Association / Organisation	**SDI Limited**
Emergency telephone numbers	131126 Poisons Information Centre
Other emergency telephone numbers	+61 3 8727 7111

SECTION 2 Hazard(s) identification

Classification of the substance or mixture

NFPA 704 diamond

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

Classification	Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Serious Eye Damage Category 1, Skin Sensitizer Category 1, Respiratory Sensitizer Category 1, Carcinogenicity Category 1B, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Specific target organ toxicity - repeated exposure Category 2, Chronic Aquatic Hazard Category 4

Label elements

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Hazard pictogram(s)	
Signal word	**Danger**

Hazard statement(s)

H302	Harmful if swallowed.
H315	Causes skin irritation.
H318	Causes serious eye damage.
H317	May cause an allergic skin reaction.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H350	May cause cancer.
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.

Hazard(s) not otherwise classified

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P281	Use personal protective equipment as required.
P285	In case of inadequate ventilation wear respiratory protection.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/attention.
P310	Immediately call a POISON CENTER or doctor/physician.
P321	Specific treatment (see advice on this label).
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER or doctor/physician.
P362	Take off contaminated clothing and wash before reuse.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P301+P312	IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell.
P330	Rinse mouth.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available		Sealed containers with electrochemical contents, typically
12190-79-3	53.6	lithium cobaltate
7782-42-5	17.4	graphite

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CAS No	%[weight]	Name
7440-50-8	12.5	copper
7429-90-5	12.5	aluminium
96-49-1	3.17	ethylene carbonate
21324-40-3	0.83	lithium fluorophosphate

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

SECTION 4 First-aid measures

Description of first aid measures

Eye Contact	‣ Generally not applicable.
Skin Contact	‣ Generally not applicable.
Inhalation	‣ Generally not applicable.
Ingestion	‣ Generally not applicable.

Most important symptoms and effects, both acute and delayed

See Section 11

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Fire-fighting measures

Extinguishing media

- Sand, dry powder extinguishers or other inerts should be used to smother dust fires.
- **DO NOT** use halogenated fire extinguishing agents.

Special hazards arising from the substrate or mixture

Fire Incompatibility	‣ If leaked, forbidden to contact with strong oxidisers, mineral acids, strong alkalies, halogenated hydrocarbons. ‣ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result ‣ Keep dry ‣ **NOTE:** May develop pressure in containers; open carefully. Vent periodically.

Special protective equipment and precautions for fire-fighters

Fire Fighting	‣ 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. Slight hazard when exposed to heat, flame and oxidisers.
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 toxic fumes of: carbon dioxide (CO_2) metal oxides other pyrolysis products typical of burning organic material. May form peroxides.

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	‣ 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	‣ 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).

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	▸ Water may be used to prevent dusting. ▸ Collect remaining material in containers with covers for disposal. ▸ Flush spill area with water.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	Conditions to avoid: Heat above 70 deg C or incinerate. Deform. Mutilate. Crush. Disassemble. Overcharge. Short circuit. Expose over a long period to humid conditions. Graphite: · 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. **NOTE:** ▸ Wet, activated carbon removes oxygen from the air thus producing a severe hazard to workers inside carbon vessels and in enclosed or confined spaces where activated carbons might accumulate. ▸ Before entry to such areas, sampling and test procedures for low oxygen levels should be undertaken; control conditions should be established to ensure the availability of adequate oxygen supply. For molten metals: · Molten metal and water can be an explosive combination. The risk is greatest when there is sufficient molten metal to entrap or seal off water. Water and other forms of contamination on or contained in scrap or remelt ingot are known to have caused explosions in melting operations. While the products may have minimal surface roughness and internal voids, there remains the possibility of moisture contamination or entrapment. If confined, even a few drops can lead to violent explosions. · All tooling, containers, molds and ladles, which come in contact with molten metal must be preheated or specially coated, rust free and approved for such use. · Any surfaces that may contact molten metal (e.g. concrete) should be specially coated · Drops of molten metal in water (e.g. from plasma arc cutting), while not normally an explosion hazard, can generate enough flammable hydrogen gas to present an explosion hazard. Vigorous circulation of the water and removal of the particles minimise the hazard. During melting operations, the following minimum guidelines should be observed: · Inspect all materials prior to furnace charging and completely remove surface contamination such as water, ice, snow, deposits of grease and oil or other surface contamination resulting from weather exposure, shipment, or storage. · Store materials in dry, heated areas with any cracks or cavities pointed downwards. · Preheat and dry large objects adequately before charging in to a furnace containing molten metal. This is typically done by the use of a drying oven or homogenising furnace. The dry cycle should bring the metal temperature of the coldest item of the batch to 200 degree C (400 deg F) and then hold at that temperature for 6 hours. ▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ **DO NOT** allow material to contact humans, exposed food or food utensils. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	▸ 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.

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	▸ Avoid reaction with oxidising agents strong alkalis water

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US ACGIH Threshold Limit Values (TLV)	lithium cobaltate	Cobalt and inorganic compounds, as Co (Inhalable particulate matter)	0.02 mg/m3	Not Available	Not Available	Pulm func changes; BEI

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US NIOSH Recommended Exposure Limits (RELs)	graphite	Black lead, Mineral carbon, Plumbago, Silver graphite, Stove black [Note: Also see specific listing for Graphite (synthetic).]	2.5 (resp) mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Levels (PELs) - Table Z3	graphite	Graphite	15 mppcf	Not Available	Not Available	(Name ((Natural)))
US OSHA Permissible Exposure Levels (PELs) - Table Z1	graphite	Graphite, natural, respirable dust	Not Available	Not Available	Not Available	See Table Z-3
US ACGIH Threshold Limit Values (TLV)	graphite	Graphite (all forms except graphite fibers) (Respirable particulate matter)	2 mg/m3	Not Available	Not Available	Pneumoconiosis
US NIOSH Recommended Exposure Limits (RELs)	copper	Copper metal dusts, Copper metal fumes	1 mg/m3	Not Available	Not Available	[*Note: The REL also applies to other copper compounds (as Cu) except Copper fume.]
US OSHA Permissible Exposure Levels (PELs) - Table Z1	copper	Copper: Dusts and mists (as Cu)	1 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Levels (PELs) - Table Z1	copper	Copper: Fume (as Cu)	0.1 mg/m3	Not Available	Not Available	Not Available
US ACGIH Threshold Limit Values (TLV)	copper	Copper Fume, as Cu	0.2 mg/m3	Not Available	Not Available	Irr; GI; metal fume fever
US ACGIH Threshold Limit Values (TLV)	copper	Copper Dusts and mists, as Cu	1 mg/m3	Not Available	Not Available	Irr; GI; metal fume fever
US NIOSH Recommended Exposure Limits (RELs)	aluminium	Aluminium, Aluminum metal, Aluminum powder, Elemental aluminum	10 (total), 5 (resp) mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Levels (PELs) - Table Z1	aluminium	Aluminum, metal (as Al): Total dust	15 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Levels (PELs) - Table Z1	aluminium	Aluminum, metal (as Al): Respirable fraction	5 mg/m3	Not Available	Not Available	Not Available
US ACGIH Threshold Limit Values (TLV)	aluminium	Aluminum metal and insoluble compounds (Respirable particulate matter)	1 mg/m3	Not Available	Not Available	Pneumoconiosis; LRT irr; neurotoxicity

Emergency Limits

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
graphite	Carbon; (Graphite, 7782-42-5)	6 mg/m3	330 mg/m3	2,000 mg/m3
copper	Copper	3 mg/m3	33 mg/m3	200 mg/m3
ethylene carbonate	Glycol carbonate; (Ethylene carbonate)	30 mg/m3	330 mg/m3	2,000 mg/m3
lithium fluorophosphate	Lithium hexafluorophosphate	7.5 mg/m3	83 mg/m3	500 mg/m3

Ingredient	Original IDLH	Revised IDLH
lithium cobaltate	Not Available	Not Available
graphite	1,250 mg/m3	Not Available
copper	100 mg/m3	Not Available
aluminium	Not Available	Not Available
ethylene carbonate	Not Available	Not Available
lithium fluorophosphate	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
ethylene carbonate	E	≤ 0.01 mg/m³
lithium fluorophosphate	E	≤ 0.01 mg/m³
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

Exposure controls

Appropriate engineering controls

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

- Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area.
- Work should be undertaken in an isolated system such as a "glove-box" . Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system.
- Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within.

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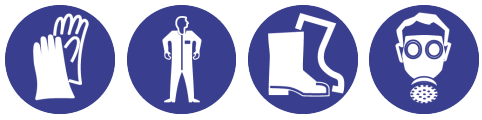
	▸ Open-vessel systems are prohibited. ▸ Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation. ▸ Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system. ▸ For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▸ Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas). ▸ Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air. ▸ Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed. 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. Exhaust ventilation should be designed to prevent accumulation and recirculation in the workplace and safely remove carbon black from the air. 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]
Personal protection	
Eye and face protection	▸ Safety glasses with side shields. ▸ Chemical goggles. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	Wear general protective gloves, eg. light weight rubber gloves. **NOTE:** ▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.
Body protection	See Other protection below
Other protection	▸ Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent] ▸ Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent] ▸ Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely. ▸ Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. ▸ Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. · 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 Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)

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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	-	A-PAPR-AUS / Class 1
up to 50 x ES	-	A-AUS / Class 1	-
up to 100 x ES	-	A-2	A-PAPR-2 ^

^ - 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.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Rectangular shaped battery; insoluble in water.

Physical state	Manufactured	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Applicable
pH (as supplied)	Not Applicable	**Decomposition temperature**	Not Available
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

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	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. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Inhalation of dusts, generated by the material during the course of normal handling, may be damaging to the health of the individual.
Ingestion	Considered an unlikely route of entry in commercial/industrial environments Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there

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	may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition 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.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. On the basis, primarily, of animal experiments, the material may be regarded as carcinogenic to humans. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in cancer on the basis of: - appropriate long-term animal studies - other relevant information 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. 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 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 osseus tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory These salts' estrogen-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*] In general, available cohort studies in humans have not reported a significant increase in total mortality as a result of cobalt exposure. Several studies have noted increased mortality rates resulting from lung cancer following occupational exposure to cobalt, either as a mixture of cobalt compounds or as hard metal, a metal alloy with a tungsten carbide and cobalt matrix. Fatal cases of hard metal disease and cardiomyopathy believed to have resulted from occupational cobalt exposure have also been reported. However, in the majority of these and other reported occupational studies, co-exposure to other substances was common, and was unable to be corrected for in the analysis. The effects of chronic occupational exposure to cobalt and cobalt compounds on the respiratory system in humans are well-documented. These effects include respiratory irritation, diminished pulmonary function, wheezing, asthma, pneumonia, and fibrosis and occurred at exposure levels ranging from 0.007 to 0.893 mg cobalt/m3 (exposure from 2 to 17 years). These effects have been observed in workers employed in cobalt refineries, as well as hard metal workers, diamond polishers, and ceramic dish painters (painting with cobalt blue dye). Occupational asthma attributed to the inhalation of cobalt powder has been confirmed following bronchial challenge tests. Chest tightness and

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chronic bronchitis have been recorded in hard-metal workers exposed to cobalt. Cobalt is known to function as a hapten, resulting in the generation of antibodies against cobalt-protein complexes. Although the minimum exposure level associated with cobalt sensitisation has not been determined, sensitisation has been demonstrated in hard metal workers with work-related asthma who have experienced prolonged occupational exposure (>3 years) to levels ranging from 0.007 to 0.893 mg cobalt/m3. The sensitisation phenomenon includes the production of IgE and IgA antibodies to cobalt. Exposure to inhaled cobalt chloride aerosols can precipitate an asthmatic attack in sensitised individuals believed to be the result of an allergic reaction within the lungs.
Allergic dermatitis of an erythematous papular type may also occur following occupational exposure. Dermatitis is a common result of dermal exposure to cobalt in humans that has been verified in a large number of studies. Using patch tests and intradermal injections, it has been demonstrated that the dermatitis is probably caused by an allergic reaction to cobalt. Contact allergy was reported in 22 of 223 (9.9%) nurses who were tested with a patch test of 1.0% cobalt chloride as well as 16 of 79 (20.3%) of examined dentists. Persons with body piercings showed an increased prevalence of allergy to cobalt, with the incidence of contact allergy being proportional to number of piercings The prevalence of sensitivity to cobalt following exposure to cobalt as a component of metal implants is low, with only 3.8% of patients developing a new sensitivity to cobalt following insertion of the implant
Exposure levels associated with the development of dermatitis have not been identified. It appears that the allergic properties of cobalt result mainly from exposure to the metal itself, rather than a salt, as it has been demonstrated that daily repeated exposure to aqueous cobalt salts did not result in hand eczema in patients known to have cobalt allergy.
Occupational exposure to cobalt in humans has been reported to cause several effects on the nervous system, including memory loss , nerve deafness, and a decreased visual acuity. It should be noted though, that both of the studies reporting on these findings, had small numbers of subjects, and exposure characterization was not reported.
Chronic exposure to cobalt produces polycythaemia (increase in blood haemoglobin), increased production of cells of the bone marrow and thyroid gland, pericardial effusion and damage to the alpha cells of the pancreas. Chronic exposure to cobalt compounds may result in pericardial effusion, polycardial effusion, cardiac failure, vomiting, convulsions and thyroid enlargement.
Chronic administration of cobaltous chloride has produced goiter, reduced thyroid activity and lowered synthesis rates and levels of cytochrome P-450, an enzymatic system responsible for chemical detoxification, in the liver. A toxic nephritis (kidney disease) may also develop.
Epidemic cardiomyopathy (heart disease) among heavy beer drinkers in the 1960's in Canada, the USA and Belgium has been attributed to the addition of up to 1.5 ppm of cobalt as a foam restorative and stabiliser. Other factors are probably implicated as therapeutic doses of cobalt, up to 50 mg/day (in the treatment of refractory anaemias) do not produce this effect. Inadequate protein or vitamin intake amongst heavy drinkers, or the effects of alcohol in rendering the heart more susceptible to disease may be important.
Single and repeated subcutaneous or intramuscular injection of cobalt powder and salts to rats may cause sarcoma at the injection site but evidence for carcinogenicity by any other route of exposure does not exist. A number of single cases of malignant tumours, mostly sarcomas, have been reported at the site of orthopedic implants containing cobalt.
Animals, exposed to cobalt compounds also exhibit an increase in respiration, as well as tremor and convulsion. Exposure of rats and mice to aerosols of cobalt (as cobalt sulfate) at concentrations from 0.11 to 1.14 mg cobalt/m3 for 2 years resulted in a spectrum of inflammatory, fibrotic, and proliferative lesions in the respiratory tract of male and female rats and mice. Squamous metaplasia of the larynx occurred in rats and mice at exposure concentrations of .0.11 mg cobalt/m3, with severity of the lesion increasing with increased cobalt concentration. Hyperplastic lesions of the nasal epithelium occurred in rats at concentrations of .0.11 mg cobalt/3, and in mice at concentrations of .0.38 mg cobalt/m3. Both sexes of rats had greatly increased incidences (>90% incidence) of alveolar lesions at all exposure levels, including inflammatory changes, fibrosis, and metaplasia. Similar changes were seen in mice at all exposure levels, though the changes in mice were less severe.
Cobalt metal dust inhalations by miniature swine resulted in early marked decrease in lung compliance and increases in septal collagen. After a one-week "sensitising period", followed by a 10-day lapse period, further exposures resulted in wheezing produced by hypersensitivity reactions.
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.
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.
Chronic inhalation exposure of production workers has caused decreased pulmonary function ad 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

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	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-medicated 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. Chronic copper poisoning is rarely recognised in man although in one instance, at least, symptoms more commonly associated with exposures to mercury, namely infantile acrodynia (pink disease), have been described. Tissue damage of mucous membranes may follow chronic dust exposure. A hazardous situation is exposure of a worker with the rare hereditary condition (Wilson's disease or hereditary hepatolenticular degeneration) to copper exposure which may cause liver, kidney, CNS, bone and sight damage and is potentially lethal. Haemolytic anaemia (a result of red-blood cell damage) is common in cows and sheep poisoned by copper derivatives. Overdosing of copper feed supplements has resulted in pigmentary cirrhosis of the liver. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products]

Li-ion polymer battery in mouthpiece	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
lithium cobaltate	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
graphite	**TOXICITY**	**IRRITATION**
	Oral (rat) LD50: >2000 mg/kg[2]	Not Available
copper	**TOXICITY**	**IRRITATION**
	0.12 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	12 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
	Oral (mouse) LD50: =.7 mg/kg[2]	
	Oral (rat) LD50: 5800 mg/kg[2]	
aluminium	**TOXICITY**	**IRRITATION**
	Not Available	Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
ethylene carbonate	**TOXICITY**	**IRRITATION**
	Not Available	Eye (rabbit): 20 mg - mild
		Eye: adverse effect observed (irritating)[1]
		Skin (rabbit): 660 mg - moderate
		Skin: no adverse effect observed (not irritating)[1]
lithium fluorophosphate	**TOXICITY**	**IRRITATION**
	Oral (rat) LD50: 50-300 mg/kg[1]	Not Available
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*	

LITHIUM COBALTATE	The following information refers to contact allergens as a group and may not be specific to this product.

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	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. Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure. Goitrogenic:. Goitrogens are substances that suppress the function of the thyroid gland by interfering with iodine uptake, which can, as a result, cause an enlargement of the thyroid, i.e., a goitre Goitrogens include: ▸ Vitexin, a 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	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. 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).
ETHYLENE CARBONATE	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 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

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	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 CO_2, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO_2, 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 embyrotoxicity 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.
LITHIUM COBALTATE & GRAPHITE & ALUMINIUM & LITHIUM FLUOROPHOSPHATE	No significant acute toxicological data identified in literature search.
GRAPHITE & ETHYLENE CARBONATE & LITHIUM FLUOROPHOSPHATE	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. 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. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

Acute Toxicity	✔	**Carcinogenicity**	✔
Skin Irritation/Corrosion	✔	**Reproductivity**	✘

Continued...

Serious Eye Damage/Irritation	✔	**STOT - Single Exposure**	✔
Respiratory or Skin sensitisation	✔	**STOT - Repeated Exposure**	✔
Mutagenicity	✘	**Aspiration Hazard**	✘

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

SECTION 12 Ecological information

Toxicity

Li-ion polymer battery in mouthpiece

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

lithium cobaltate

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	1.406mg/L	2
EC50	48	Crustacea	0.241mg/L	2
EC50	72	Algae or other aquatic plants	0.0288mg/L	2
NOEC	168	Algae or other aquatic plants	0.0018mg/L	2

graphite

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	>100mg/L	2
EC50	48	Crustacea	>100mg/L	2
EC50	72	Algae or other aquatic plants	>100mg/L	2
NOEC	72	Algae or other aquatic plants	>=100mg/L	2

copper

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	0.0028mg/L	2
EC50	48	Crustacea	0.001mg/L	2
EC50	72	Algae or other aquatic plants	-0.0108035-0.0171585mg/L	4
BCFD	1344	Not Available	7402.320-mg/L	4
EC25	6	Algae or other aquatic plants	0.001506135-mg/L	4
NOEL	1440	Not Available	-0.0004-0.00122mg/L	4

aluminium

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	0.078242mg/L	2
EC50	48	Crustacea	0.7364mg/L	2
EC50	96	Algae or other aquatic plants	0.0054mg/L	2
BCF	360	Not Available	9mg/L	4
NOEC	72	Algae or other aquatic plants	>=0.004mg/L	2

ethylene carbonate

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	>100mg/L	2
EC50	48	Crustacea	>100mg/L	2
EC50	72	Algae or other aquatic plants	>100mg/L	2
NOEC	72	Algae or other aquatic plants	100mg/L	2

lithium fluorophosphate

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	42mg/L	2
EC50	48	Crustacea	98mg/L	2
EC50	96	Algae or other aquatic plants	43mg/L	2
NOEC	528	Fish	0.2mg/L	2

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 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.

Persistence and degradability

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

Bioaccumulative potential

Continued...

Ingredient	Bioaccumulation
ethylene carbonate	LOW (LogKOW = -0.3388)

Mobility in soil

Ingredient	Mobility
ethylene carbonate	LOW (KOC = 9.168)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	· 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.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	NO

Land transport (DOT)

UN number	3481
UN proper shipping name	Lithium ion batteries packed with equipment including lithium ion polymer batteries
Transport hazard class(es)	Class: 9 Subrisk: Not Applicable
Packing group	Not Applicable
Environmental hazard	Not Applicable
Special precautions for user	Hazard Label: 9 Special provisions: 181, 388, 422, A54

Air transport (ICAO-IATA / DGR)

UN number	3481
UN proper shipping name	Lithium ion batteries packed with equipment (including lithium ion polymer batteries); Lithium ion batteries contained in equipment (including lithium ion polymer batteries)
Transport hazard class(es)	ICAO/IATA Class: 9 ICAO / IATA Subrisk: Not Applicable ERG Code: 12FZ
Packing group	Not Applicable
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: A48 A88 A99 A154 A164 A181 A185 A206 A213; A88 A99 A154 A164 A181 A185 A206 A213 Cargo Only Packing Instructions: 967; 966 Cargo Only Maximum Qty / Pack: 35 kg Passenger and Cargo Packing Instructions: 967; 966 Passenger and Cargo Maximum Qty / Pack: 5 kg Passenger and Cargo Limited Quantity Packing Instructions: Forbidden Passenger and Cargo Limited Maximum Qty / Pack: Forbidden

Sea transport (IMDG-Code / GGVSee)

UN number	3481

Continued...

UN proper shipping name	LITHIUM ION BATTERIES CONTAINED IN EQUIPMENT or LITHIUM ION BATTERIES PACKED WITH EQUIPMENT (including lithium ion polymer batteries)
Transport hazard class(es)	IMDG Class: 9 IMDG Subrisk: Not Applicable
Packing group	Not Applicable
Environmental hazard	Not Applicable
Special precautions for user	EMS Number: F-A , S-I Special provisions: 188 230 310 348 360 376 377 384 387 Limited Quantities: 0

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

Not Applicable

SECTION 15 Regulatory information

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

lithium cobaltate is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B : Possibly carcinogenic to humans
US - California Hazardous Air Pollutants Identified as Toxic Air Contaminants
US ACGIH Threshold Limit Values (TLV)
US ACGIH Threshold Limit Values (TLV) - Carcinogens
US AIHA Workplace Environmental Exposure Levels (WEELs)
US Clean Air Act - Hazardous Air Pollutants
US EPCRA Section 313 Chemical List
US National Toxicology Program (NTP) 14th Report Part B. Reasonably Anticipated to be a Human Carcinogen
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

graphite is found on the following regulatory lists

US ACGIH Threshold Limit Values (TLV)
US AIHA Workplace Environmental Exposure Levels (WEELs)
US DOE Temporary Emergency Exposure Limits (TEELs)
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Levels (PELs) - Table Z1
US OSHA Permissible Exposure Levels (PELs) - Table Z3
US OSHA Permissible Exposure Limits - Annotated Table Z-1
US OSHA Permissible Exposure Limits - Annotated Table Z-3
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances
US TSCA Section 12(b) - List of Chemical Substances Subject to Export Notification Requirements

copper is found on the following regulatory lists

US - California Hazardous Air Pollutants Identified as Toxic Air Contaminants
US ACGIH Threshold Limit Values (TLV)
US AIHA Workplace Environmental Exposure Levels (WEELs)
US ATSDR Minimal Risk Levels for Hazardous Substances (MRLs)
US CWA (Clean Water Act) - Priority Pollutants
US CWA (Clean Water Act) - Toxic Pollutants
US DOE Temporary Emergency Exposure Limits (TEELs)
US EPA Integrated Risk Information System (IRIS)
US EPCRA Section 313 Chemical List
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Levels (PELs) - Table Z1
US OSHA Permissible Exposure Limits - Annotated Table Z-1
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

aluminium is found on the following regulatory lists

US ACGIH Threshold Limit Values (TLV)
US ACGIH Threshold Limit Values (TLV) - Carcinogens
US AIHA Workplace Environmental Exposure Levels (WEELs)
US ATSDR Minimal Risk Levels for Hazardous Substances (MRLs)
US Department of Homeland Security (DHS) - Chemical Facility Anti-Terrorism Standards (CFATS) - Chemicals of Interest
US EPCRA Section 313 Chemical List
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Levels (PELs) - Table Z1
US OSHA Permissible Exposure Limits - Annotated Table Z-1
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

ethylene carbonate is found on the following regulatory lists

Continued...

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

lithium fluorophosphate is found on the following regulatory lists

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

Federal Regulations

Superfund Amendments and Reauthorization Act of 1986 (SARA)

Section 311/312 hazard categories

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

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

Name	Reportable Quantity in Pounds (lb)	Reportable Quantity in kg
Copper	5000	2270

State Regulations

US. California Proposition 65

None Reported

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (lithium fluorophosphate)
Canada - NDSL	No (lithium cobaltate; graphite; copper; aluminium; ethylene carbonate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (graphite; copper; aluminium; lithium fluorophosphate)
Korea - KECI	Yes
New Zealand - NZIoC	No (lithium fluorophosphate)
Philippines - PICCS	No (lithium cobaltate)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (lithium cobaltate; ethylene carbonate; lithium fluorophosphate)
Vietnam - NCI	No (lithium cobaltate)
Russia - ARIPS	No (lithium cobaltate; lithium fluorophosphate)
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

Continued...

SECTION 16 Other information

Revision Date	28/08/2020
Initial Date	28/07/2020

SDS Version Summary

Version	Issue Date	Sections Updated
2.1.1.1	28/07/2020	Classification, Spills (major)
3.1.1.1	28/08/2020	Fire Fighter (fire/explosion hazard), Fire Fighter (fire incompatibility), Handling Procedure, Transport Information

Other information

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

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

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
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

The information contained in the Safety Data Sheet is based on data considered to be accurate, however, no warranty is expressed or implied regarding the accuracy of the data or the results to be obtained from the use thereof.

Other information:
Prepared by: SDI Limited
3-15 Brunsdon Street, Bayswater Victoria, 3153, Australia
Phone Number: +61 3 8727 7111
Department issuing SDS: Research and Development
Contact: Technical Director

end of SDS

Pola Day 9.5% Hydrogen Peroxide Gel
SDI (North America) Inc.

Version No: **6.1.1.1**
Safety Data Sheet according to OSHA HazCom Standard (2012) requirements

Issue Date: **01/11/2019**
Print Date: **23/09/2020**
L.GHS.USA.EN

SECTION 1 Identification

Product Identifier

Product name	Pola Day 9.5% Hydrogen Peroxide Gel
Synonyms	Not Available
Proper shipping name	Hydrogen, peroxide, aqueous solutions with not less than 8 percent but less than 20 percent hydrogen peroxide (stabilized as necessary)
Other means of identification	Not Available

Recommended use of the chemical and restrictions on use

Relevant identified uses	Dental use: To remove discoloration of teeth under the supervision of a dentist.

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

Registered company name	**SDI (North America) Inc.**	**SDI Limited**	**SDI Dental Limited**
Address	1279 Hamilton Parkway Itasca IL 60143 United States	3-15 Brunsdon Street Bayswater VIC 3153 Australia	Block 8, St Johns Court Santry Dublin 9 Ireland
Telephone	+1 630 361 9200 (Business hours)|1 800 228 5166	+61 3 8727 7111 (Business Hours)	+353 1 886 9577 (Business Hours)|800 0225 5734
Fax	+1 630 361 9222	+61 3 8727 7222	Not Available
Website	http://www.sdi.com.au	www.sdi.com.au	http://www.sdi.com.au/
Email	USA.Canada@sdi.com.au	info@sdi.com.au	Ireland@sdi.com.au

Registered company name	**SDi**
Address	Rua Dr. Virgílio de Carvalho Pinto, 612 Pinheiros, Sao Paulo 05415-020 Brazil
Telephone	+55 11 3092 7100 (Business Hours)
Fax	+55 11 3092 7101
Website	http://www.sdi.com.au/
Email	Brasil@sdi.com.au

Emergency phone number

Association / Organisation	**SDI Limited**	**SDI Dental Limited**	**SDi**
Emergency telephone numbers	+61 3 8727 7111	+61 3 8727 7111	+61 3 8727 7111
Other emergency telephone numbers	ray.cahill@sdi.com.au	Not Available	Not Available

SECTION 2 Hazard(s) identification

Classification of the substance or mixture

NFPA 704 diamond

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

Classification	Skin Corrosion/Irritation Category 1A, Serious Eye Damage Category 1

Label elements

Hazard pictogram(s)	

Page 1 continued...

Signal word	**Danger**

Hazard statement(s)

H314	Causes severe skin burns and eye damage.

Hazard(s) not otherwise classified

Not Applicable

Precautionary statement(s) Prevention

P260	Do not breathe mist/vapours/spray.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Precautionary statement(s) Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353	IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/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 or doctor/physician.
P321	Specific treatment (see advice on this label).
P363	Wash contaminated clothing before reuse.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position 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.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
7722-84-1	9.5	hydrogen peroxide

SECTION 4 First-aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Wash out immediately with fresh 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. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary.
Ingestion	▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice.

Most important symptoms and effects, both acute and delayed

See Section 11

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Fire-fighting measures

Extinguishing media

- Water spray or fog.
- Foam.

Continued...

- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.

Special hazards arising from the substrate or mixture

Fire Incompatibility	44rnk1

Special protective equipment and precautions for fire-fighters

Fire Fighting	- Alert Fire Brigade and tell them location and nature of hazard. - Wear breathing apparatus plus protective gloves in the event of a fire. - Prevent, by any means available, spillage from entering drains or water courses. - Use fire fighting procedures suitable for surrounding area. - **DO NOT** approach containers suspected to be hot. - Cool fire exposed containers with water spray from a protected location. - If safe to do so, remove containers from path of fire. - Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	- Non combustible. - Not considered a significant fire risk, however containers may burn. May emit poisonous fumes. May emit corrosive fumes. Decomposes on heating and produces: carbon dioxide (CO_2) carbon monoxide (CO)

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	- Clean up all spills immediately. - Avoid contact with skin and eyes. - Wear impervious gloves and safety goggles. - Trowel up/scrape up. - Place spilled material in clean, dry, sealed container. - Flush spill area with water.
Major Spills	Minor hazard. - 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.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	- Avoid all personal contact, including inhalation. - Wear protective clothing when risk of exposure occurs. - Use in a well-ventilated area. - Prevent concentration in hollows and sumps. - **DO NOT** enter confined spaces until atmosphere has been checked. - **DO NOT** allow material to contact humans, exposed food or food utensils. - Avoid contact with incompatible materials. - When handling, **DO NOT** eat, drink or smoke. - Keep containers securely sealed when not in use. - Avoid physical damage to containers. - Always wash hands with soap and water after handling. - Work clothes should be laundered separately. Launder contaminated clothing before re-use. - Use good occupational work practice. - Observe manufacturer's storage and handling recommendations contained within this SDS. - Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	**Do not** store in direct sunlight. Store between 2 and 25 deg C.

Conditions for safe storage, including any incompatibilities

Suitable container	- **DO NOT** repack. Use containers supplied by manufacturer only.
Storage incompatibility	- Avoid storage with reducing agents. 4base

Continued...

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US NIOSH Recommended Exposure Limits (RELs)	hydrogen peroxide	High-strength hydrogen peroxide, Hydrogen dioxide, Hydrogen peroxide (aqueous), Hydroperoxide, Peroxide	1 ppm / 1.4 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Levels (PELs) - Table Z1	hydrogen peroxide	Hydrogen peroxide	1 ppm / 1.4 mg/m3	Not Available	Not Available	Not Available
US ACGIH Threshold Limit Values (TLV)	hydrogen peroxide	Hydrogen peroxide	1 ppm	Not Available	Not Available	Eye, URT, & skin irr

Emergency Limits

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
hydrogen peroxide	Hydrogen peroxide	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
hydrogen peroxide	75 ppm	Not Available

MATERIAL DATA

Exposure controls

Appropriate engineering controls

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

General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in specific circumstances. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. 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.

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)

Within each range the appropriate value depends on:

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

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

Personal protection

Eye and face protection

- Safety glasses with side shields.
- Chemical goggles.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection

See Hand protection below

Continued...

Hands/feet protection	▸ Wear chemical protective gloves, e.g. PVC. ▸ Wear safety footwear or safety gumboots, e.g. Rubber ▸ Rubber Gloves
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ P.V.C apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.

Respiratory protection

Type B 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	B-AUS	-	B-PAPR-AUS / Class 1
up to 50 x ES	-	B-AUS / Class 1	-
up to 100 x ES	-	B-2	B-PAPR-2 ^

^ - 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)

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Clear gel with spearmint odour, mixes with water.

Physical state	Gel	**Relative density (Water = 1)**	1.1
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	5.9-6.9	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	The material is not thought to produce respiratory irritation (as classified by EC Directives using animal models). Nevertheless inhalation, of the material, especially for prolonged periods, may produce respiratory discomfort and occasionally, distress.
Ingestion	The material has **NOT** been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.

Continued...

Skin Contact	Limited evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. 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.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

Pola Day 9.5% Hydrogen Peroxide Gel	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

hydrogen peroxide	**TOXICITY**	**IRRITATION**
	50 mg/kg[2]	Not Available
	500 mg/kg[2]	
	Dermal (rabbit) LD50: 4060 mg/kg[2]	
	Inhalation (rat) LC50: 2 mg/l/4H[2]	
	Oral (rat) LD50: =1193-1270 mg/kg[2]	
	Oral (rat) LD50: >225 mg/kg[2]	
	Oral (rat) LD50: >5000 mg/kg[2]	
	Oral (rat) LD50: 1270 mg/kg[1]	

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

HYDROGEN PEROXIDE

No significant acute toxicological data identified in literature search.

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. 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.
Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
For hydrogen peroxide**:**
Hazard increases with peroxide concentration, high concentrations contain an additive stabiliser.
Pharmacokinetics
Hydrogen peroxide is a normal product of metabolism. It is readily decomposed by catalase in normal cells. In experimental animals exposed to hydrogen peroxide, target organs affected include the lungs, intestine, thymus, liver, and kidney, suggesting its distribution to those sites.
Hydrogen peroxide has been detected in breath.

- Absorption: Hydrogen peroxide is decomposed in the bowel before absorption. When applied to tissue, solutions of hydrogen peroxide have poor penetrability.
- Distribution Hydrogen peroxide is produced metabolically in intact cells and tissues. It is formed by reduction of oxygen either directly in a two-electron transfer reaction, often catalysed by flavoproteins, or by an initial one-electron step to O2 followed by dismutation to hydrogen peroxide.
- Hydrogen peroxide has been detected in serum and in intact liver. based on the results of toxicity studies, the lungs, intestine, thymus, liver, and kidney may be distribution sites. In rabbits and cats that died after intravenous administration of hydrogen peroxide, the lungs were pale and emphysematous. Following intraperitoneal injection of hydrogen peroxide in mice, pyknotic nuclei were induced in the intestine and thymus (IARC 1985). Degeneration of hepatic and renal tubular epithelial tissue was observed following oral administration of hydrogen peroxide to mice.
- Metabolism Glutathione peroxidase, responsible for decomposing hydrogen peroxide, is present in normal human tissues (IARC 1985). When hydrogen peroxide comes in contact with catalase, an enzyme found in blood and most tissues, it rapidly decomposes into oxygen and water.
- Excretion Hydrogen peroxide has been detected in human breath at levels ranging from 1.0+/-.5 g/L to 0.34+/-0.17 g/L.

Carcinogenicity
Gastric and duodenal lesions including adenomas, carcinomas, and adenocarcinomas have been observed in mice treated orally with hydrogen peroxide. Marked strain differences in the incidence of tumors have been observed. Papilloma development has been observed in mice treated by dermal application.
Genotoxicity
Hydrogen peroxide induced DNA damage, sister chromatid exchanges and chromosomal aberrations in mammalian cells *in vitro* . Hydrogen peroxide induced DNA damage in bacteria (*E. coli*), and was mutagenic to bacteria (*Salmonella typhimurium)* and the fungi, *Neurospora crassa* and *Aspergillis chevallieri*, but not to *Streptomyces griseoflavus*. It was not mutagenic to *Drosophila melanogaster* or to mammalian cells *in vitro*.
Developmental Toxicity
Malformations have been observed in chicken embryos treated with hydrogen peroxide, but experiments with mice and rats have been negative.
Female rats that received 0.45% hydrogen peroxide (equivalent to approximately 630 mg/kg/day)7 as the sole drinking fluid for five weeks produced normal litters when mated with untreated males.
Doses of 1.4 to 11 mol/egg hydrogen peroxide (purity 30%) dissolved in water were injected into the airspace of groups of 20-30 white leghorn chicken eggs on day 3 of incubation.
Embryos were examined on day 14. The incidence of embryonic deaths and malformations was dose-related and detected at doses of 2.8 mol/egg and above. The combined ED50 was 2.7 mol/egg.

Continued...

	Reproductive Toxicity A 1% solution of hydrogen peroxide (equivalent to 1900 mg/kg/day) given as the sole drinking fluid to three-month-old male mice for 7-28 days did not cause infertility. 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*

SECTION 12 Ecological information

Toxicity

Pola Day 9.5% Hydrogen Peroxide Gel

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

hydrogen peroxide

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	16.4mg/L	2
EC50	48	Crustacea	2mg/L	2
EC50	72	Algae or other aquatic plants	0.85mg/L	2
NOEC	72	Algae or other aquatic plants	=0.1mg/L	1

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 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*

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
hydrogen peroxide	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
hydrogen peroxide	LOW (LogKOW = -1.571)

Mobility in soil

Ingredient	Mobility
hydrogen peroxide	LOW (KOC = 14.3)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	Consult State Land Waste Management Authority for disposal. Bury residue in an authorised landfill.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	NO

Land transport (DOT)

UN number	2984
UN proper shipping name	Hydrogen, peroxide, aqueous solutions with not less than 8 percent but less than 20 percent hydrogen peroxide (stabilized as necessary)

Continued...

Transport hazard class(es)	Class: 5.1 Subrisk: Not Applicable
Packing group	III
Environmental hazard	Not Applicable
Special precautions for user	Hazard Label: 5.1 Special provisions: A1, IB2, IP5, T4, TP1, TP6, TP24, TP37

Air transport (ICAO-IATA / DGR)

UN number	2984
UN proper shipping name	Hydrogen peroxide, aqueous solution with 8% or more but less than 20% hydrogen peroxide (stabilized as necessary)
Transport hazard class(es)	ICAO/IATA Class: 5.1 ICAO / IATA Subrisk: Not Applicable ERG Code: 5L
Packing group	III
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: A803 Cargo Only Packing Instructions: 555 Cargo Only Maximum Qty / Pack: 30 L Passenger and Cargo Packing Instructions: 551 Passenger and Cargo Maximum Qty / Pack: 2.5 L Passenger and Cargo Limited Quantity Packing Instructions: Y541 Passenger and Cargo Limited Maximum Qty / Pack: 1 L

Sea transport (IMDG-Code / GGVSee)

UN number	2984
UN proper shipping name	HYDROGEN PEROXIDE, AQUEOUS SOLUTION with not less than 8% but less than 20% hydrogen peroxide (stabilized as necessary)
Transport hazard class(es)	IMDG Class: 5.1 IMDG Subrisk: Not Applicable
Packing group	III
Environmental hazard	Not Applicable
Special precautions for user	EMS Number: F-H , S-Q Special provisions: 65 Limited Quantities: 5 L

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

Not Applicable

If packed as Chemical kits the following classification may be considered if all ICAO/IATA transport requirements are met: Chemical Kit UN3316 - Class 9, SP A44 & A163.

SECTION 15 Regulatory information

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

hydrogen peroxide is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
US ACGIH Threshold Limit Values (TLV)
US AIHA Workplace Environmental Exposure Levels (WEELs)
US Department of Homeland Security (DHS) - Chemical Facility Anti-Terrorism Standards (CFATS) - Chemicals of Interest
US DOE Temporary Emergency Exposure Limits (TEELs)
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Levels (PELs) - Table Z1
US OSHA Permissible Exposure Limits - Annotated Table Z-1
US SARA Section 302 Extremely Hazardous Substances
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

Federal Regulations

Superfund Amendments and Reauthorization Act of 1986 (SARA)

Section 311/312 hazard categories

Flammable (Gases, Aerosols, Liquids, or Solids)	No
Gas under pressure	No
Explosive	No
Self-heating	No

Continued...

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

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

None Reported

State Regulations

US. California Proposition 65

None Reported

National Inventory Status

National Inventory	Status
Australia - AIIC	Yes
Australia Non-Industrial Use	No (hydrogen peroxide)
Canada - DSL	Yes
Canada - NDSL	No (hydrogen peroxide)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - ARIPS	Yes
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 Other information

Revision Date	01/11/2019
Initial Date	09/11/2015

SDS Version Summary

Version	Issue Date	Sections Updated
5.1.1.1	18/03/2016	Acute Health (inhaled), Acute Health (swallowed), Classification, Fire Fighter (fire incompatibility), Storage (storage incompatibility), Transport
6.1.1.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification

Other information

Continued...

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

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

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
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

The information contained in the Safety Data Sheet is based on data considered to be accurate, however, no warranty is expressed or implied regarding the accuracy of the data or the results to be obtained from the use thereof.

Other information:
Prepared by: SDI Limited
3-15 Brunsdon Street, Bayswater Victoria, 3153, Australia
Phone Number: +61 3 8727 7111
Department issuing SDS: Research and Development
Contact: Technical Director

end of SDS

Pola Night 22% Carbamide Peroxide Gel
SDI (North America) Inc.

Version No: **10.1.1.1**
Safety Data Sheet according to OSHA HazCom Standard (2012) requirements

Issue Date: **01/11/2019**
Print Date: **01/10/2020**
L.GHS.USA.EN

SECTION 1 Identification

Product Identifier

Product name	Pola Night 22% Carbamide Peroxide Gel
Synonyms	Not Available
Other means of identification	Not Available

Recommended use of the chemical and restrictions on use

Relevant identified uses	To remove discoloration of teeth, to be performed by a dentist.

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

Registered company name	**SDI (North America) Inc.**	**SDI Limited**	**SDi**
Address	1279 Hamilton Parkway Itasca IL 60143 United States	3-15 Brunsdon Street Bayswater VIC 3153 Australia	Rua Dr. Virgílio de Carvalho Pinto, 612 Pinheiros, Sao Paulo 05415-020 Brazil
Telephone	+1 630 361 9200 (Business hours)|1 800 228 5166	+61 3 8727 7111 (Business Hours)	+55 11 3092 7100 (Business Hours)
Fax	+1 630 361 9222	+61 3 8727 7222	+55 11 3092 7101
Website	http://www.sdi.com.au	www.sdi.com.au	http://www.sdi.com.au/
Email	USA.Canada@sdi.com.au	info@sdi.com.au	Brasil@sdi.com.au

Registered company name	**SDI Dental Limited**
Address	Block 8, St Johns Court Santry Dublin 9 Ireland
Telephone	+353 1 886 9577 (Business Hours)|800 0225 5734
Fax	Not Available
Website	http://www.sdi.com.au/
Email	Ireland@sdi.com.au

Emergency phone number

Association / Organisation	**SDI Limited**	**SDi**	**SDI Dental Limited**
Emergency telephone numbers	+61 3 8727 7111	+61 3 8727 7111	+61 3 8727 7111
Other emergency telephone numbers	ray.cahill@sdi.com.au	Not Available	Not Available

SECTION 2 Hazard(s) identification

Classification of the substance or mixture

NFPA 704 diamond

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

Classification	Eye Irritation Category 2A

Label elements

Hazard pictogram(s)	
Signal word	**Warning**

Page 1 continued...

Hazard statement(s)

H319	Causes serious eye irritation.

Hazard(s) not otherwise classified

Not Applicable

Precautionary statement(s) Prevention

P280	Wear protective gloves/protective clothing/eye protection/face protection.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

Not Applicable

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
124-43-6	22	urea hydrogen peroxide
Not Available		equivalent to:
7722-84-1	7.3	hydrogen peroxide

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

SECTION 4 First-aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Wash out immediately with fresh 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. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs: ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary.
Ingestion	▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice.

Most important symptoms and effects, both acute and delayed

See Section 11

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Fire-fighting measures

Extinguishing media

- Foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.
- Water spray or fog - Large fires only.

Special hazards arising from the substrate or mixture

Fire Incompatibility	▸ Avoid any contamination of this material as it is very reactive and any contamination is potentially hazardous

Continued...

Special protective equipment and precautions for fire-fighters

Fire Fighting	‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear full body protective clothing with breathing apparatus. ‣ Prevent, by any means available, spillage from entering drains or water courses. ‣ Fight fire from a safe distance, with adequate cover. ‣ Extinguishers should be used only by trained personnel. ‣ Use water delivered as a fine spray to control fire and cool adjacent area. ‣ Avoid spraying water onto liquid pools. ‣ **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. ‣ If fire gets out of control withdraw personnel and warn against entry. ‣ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	‣ Non combustible. ‣ Not considered a significant fire risk, however containers may burn. Decomposition may produce toxic fumes of: nitrogen oxides (NOx) carbon monoxide (CO) carbon dioxide (CO2)

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	Wipe with absorbent towel. Wash with water (15 mins).
Major Spills	‣ Clear area of personnel and move upwind. ‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear full body protective clothing with breathing apparatus. ‣ Prevent, by any means available, spillage from entering drains or water courses. ‣ No smoking, flames or ignition sources. Increase ventilation. ‣ Contain spill with sand, earth or other clean, inert materials. ‣ **NEVER USE** organic absorbents such as sawdust, paper or cloth. ‣ Use spark-free and explosion-proof equipment. ‣ Collect any recoverable product into labelled containers for possible recycling. ‣ Avoid contamination with organic matter to prevent subsequent fire and explosion. ‣ **DO NOT** mix fresh with recovered material. ‣ Collect residues and seal in labelled drums for disposal. ‣ Wash area and prevent runoff into drains. ‣ Decontaminate equipment and launder all protective clothing before storage and re-use. ‣ If contamination of drains or waterways occurs advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	For oxidisers, including peroxides. · Avoid personal contact and inhalation of dust, mist or vapours. · Provide adequate ventilation. · Always wear protective equipment and wash off any spillage from clothing. · Keep material away from light, heat, flammables or combustibles. · Keep cool, dry and away from incompatible materials. · Avoid physical damage to containers. · **DO NOT** repack or return unused portions to original containers. Withdraw only sufficient amounts for immediate use. · Use only minimum quantity required. · Avoid using solutions of peroxides in volatile solvents. Solvent evaporation should be controlled to avoid dangerous concentration of the peroxide. · Do NOT allow oxidisers to contact iron or compounds of iron, cobalt, or copper, metal oxide salts, acids or bases. · Do NOT use metal spatulas to handle oxidisers · Do NOT use glass containers with screw cap lids or glass stoppers. · Store peroxides at the lowest possible temperature, consistent with their solubility and freezing point. · CAUTION: Do NOT store liquids or solutions of peroxides at a temperature below that at which the oxidiser freezes or precipitates. Peroxides, in particular, in this form are extremely shock and heat-sensitive. Refrigerated storage of peroxides must ONLY be in explosion-proof units. · The hazards and consequences of fires and explosions during synthesis and use of oxidisers is widely recognised; spontaneous or induced decomposition may culminate in a variety of ways, ranging from moderate gassing to spontaneous ignition or explosion. The heat released from spontaneous decomposition of an energy-rich compound causes a rise in the surrounding temperature; the temperature will rise until thermal balance is established or until the material heats to decomposition, · The most effective means for minimising the consequences of an accident is to limit quantities to a practical minimum. Even gram-scale explosions can be serious. Once ignited the burning of peroxides cannot be controlled and the area should be evacuated. · Unless there is compelling reason to do otherwise, peroxide concentration should be limited to 10% (or less with vigorous reactants). Peroxide concentration is rarely as high as 1% in the reaction mixture of polymerisation or other free-radical reactions, · Oxidisers should be added slowly and cautiously to the reaction medium. This should be completed prior to heating and with good agitation. · Addition oxidisers to the hot monomer is extremely dangerous. A violent reaction (e.g., fire or explosion) can result from inadvertent mixing of promoters (frequently used with peroxides in polymerisation systems) with full-strength oxidisers

Continued...

	· Organic peroxides are very sensitive to contamination (especially heavy-metal compounds, metal oxide salts, alkaline materials including amines, strong acids, and many varieties of dust and dirt). This can initiate rapid, uncontrolled decomposition of peroxides and possible generation of intense heat, fire or explosion The consequences of accidental contamination from returning withdrawn material to the storage container can be disastrous. · When handling **NEVER** smoke, eat or drink. · Always wash hands with soap and water after handling. · Use only good occupational work practice. · Observe manufacturer's storage and handling recommendations contained within this MSDS.
Other information	Store between 2 and 25 deg C. **Do not** store in direct sunlight. Store in a dry and well ventilated-area, away from heat and sunlight.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ **DO NOT** repack. Use containers supplied by manufacturer only.
Storage incompatibility	▸ Avoid strong bases.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US NIOSH Recommended Exposure Limits (RELs)	hydrogen peroxide	High-strength hydrogen peroxide, Hydrogen dioxide, Hydrogen peroxide (aqueous), Hydroperoxide, Peroxide	1 ppm / 1.4 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Levels (PELs) - Table Z1	hydrogen peroxide	Hydrogen peroxide	1 ppm / 1.4 mg/m3	Not Available	Not Available	Not Available
US ACGIH Threshold Limit Values (TLV)	hydrogen peroxide	Hydrogen peroxide	1 ppm	Not Available	Not Available	Eye, URT, & skin irr

Emergency Limits

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
urea hydrogen peroxide	Urea peroxide; (Urea hydrogen peroxide)	1.2 mg/m3	13 mg/m3	79 mg/m3
hydrogen peroxide	Hydrogen peroxide	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
urea hydrogen peroxide	Not Available	Not Available
hydrogen peroxide	75 ppm	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
urea hydrogen peroxide	E	≤ 0.01 mg/m³
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

Exposure controls

Appropriate engineering controls

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

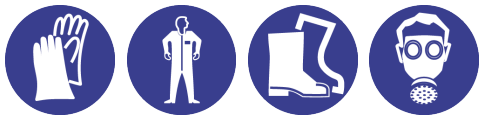
General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in specific circumstances. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. 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.

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range

Continued...

	1: Room air currents minimal or favourable to capture / 1: Disturbing room air currents 2: Contaminants of low toxicity or of nuisance value only. / 2: Contaminants of high toxicity 3: Intermittent, low production. / 3: High production, heavy use 4: Large hood or large air mass in motion / 4: Small hood-local control only Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 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.
Personal protection	
Eye and face protection	▸ Safety glasses with side shields. ▸ Chemical goggles. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	▸ Wear chemical protective gloves, e.g. PVC. ▸ Wear safety footwear or safety gumboots, e.g. Rubber 72rub2
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ PVC Apron. ▸ PVC protective suit may be required if exposure severe. ▸ Eyewash unit. ▸ Ensure there is ready access to a safety shower.

Respiratory protection

Type B 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	B-AUS	-	B-PAPR-AUS / Class 1
up to 50 x ES	-	B-AUS / Class 1	-
up to 100 x ES	-	B-2	B-PAPR-2 ^

^ - 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)

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Clear gel with spearmint odour, soluble in water.

Physical state	Gel	**Relative density (Water = 1)**	1.1
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	5.9-6.9	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (1%)**	Not Available

Continued...

Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable under normal handling conditions. ▸ Prolonged exposure to heat. ▸ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	The material is not thought to produce adverse health effects or skin irritation following contact (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting.
Eye	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

Pola Night 22% Carbamide Peroxide Gel	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
urea hydrogen peroxide	**TOXICITY**	**IRRITATION**
	Not Available	Eye: adverse effect observed (irreversible damage)[1]
		Skin: adverse effect observed (irritating)[1]
hydrogen peroxide	**TOXICITY**	**IRRITATION**
	50 mg/kg[2]	Not Available
	500 mg/kg[2]	
	Dermal (rabbit) LD50: 4060 mg/kg[2]	
	Inhalation (rat) LC50: 2 mg/l/4H[2]	
	Oral (rat) LD50: =1193-1270 mg/kg[2]	
	Oral (rat) LD50: >225 mg/kg[2]	
	Oral (rat) LD50: >5000 mg/kg[2]	
	Oral (rat) LD50: 1270 mg/kg[1]	
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*	

UREA HYDROGEN PEROXIDE	No chronic human exposure data is available
HYDROGEN PEROXIDE	For hydrogen peroxide**:** Hazard increases with peroxide concentration, high concentrations contain an additive stabiliser. **Pharmacokinetics** Hydrogen peroxide is a normal product of metabolism. It is readily decomposed by catalase in normal cells. In experimental animals exposed to hydrogen peroxide, target organs affected include the lungs, intestine, thymus, liver, and kidney, suggesting its distribution to those sites. Hydrogen peroxide has been detected in breath. ▸ Absorption: Hydrogen peroxide is decomposed in the bowel before absorption. When applied to tissue, solutions of hydrogen peroxide have poor penetrability. ▸ Distribution Hydrogen peroxide is produced metabolically in intact cells and tissues. It is formed by reduction of oxygen either directly in a two-electron transfer reaction, often catalysed by flavoproteins, or by an initial one-electron step to O2 followed by dismutation to hydrogen peroxide. ▸ Hydrogen peroxide has been detected in serum and in intact liver. based on the results of toxicity studies, the lungs, intestine, thymus, liver, and kidney may be distribution sites. In rabbits and cats that died after intravenous administration of hydrogen peroxide, the lungs were pale and emphysematous. Following intraperitoneal injection of hydrogen peroxide in mice, pyknotic nuclei were induced in the intestine and thymus (IARC 1985). Degeneration of hepatic and renal tubular epithelial tissue was observed following oral administration of hydrogen

Continued...

peroxide to mice.
- Metabolism Glutathione peroxidase, responsible for decomposing hydrogen peroxide, is present in normal human tissues (IARC 1985). When hydrogen peroxide comes in contact with catalase, an enzyme found in blood and most tissues, it rapidly decomposes into oxygen and water.
- Excretion Hydrogen peroxide has been detected in human breath at levels ranging from 1.0+/-.5 g/L to 0.34+/-0.17 g/L.

Carcinogenicity
Gastric and duodenal lesions including adenomas, carcinomas, and adenocarcinomas have been observed in mice treated orally with hydrogen peroxide. Marked strain differences in the incidence of tumors have been observed. Papilloma development has been observed in mice treated by dermal application.
Genotoxicity
Hydrogen peroxide induced DNA damage, sister chromatid exchanges and chromosomal aberrations in mammalian cells *in vitro* . Hydrogen peroxide induced DNA damage in bacteria (*E. coli*), and was mutagenic to bacteria (*Salmonella typhimurium)* and the fungi, *Neurospora crassa* and *Aspergillis chevallieri*, but not to *Streptomyces griseoflavus*. It was not mutagenic to *Drosophila melanogaster* or to mammalian cells *in vitro*.
Developmental Toxicity
Malformations have been observed in chicken embryos treated with hydrogen peroxide, but experiments with mice and rats have been negative. Female rats that received 0.45% hydrogen peroxide (equivalent to approximately 630 mg/kg/day)7 as the sole drinking fluid for five weeks produced normal litters when mated with untreated males.
Doses of 1.4 to 11 mol/egg hydrogen peroxide (purity 30%) dissolved in water were injected into the airspace of groups of 20-30 white leghorn chicken eggs on day 3 of incubation.
Embryos were examined on day 14. The incidence of embryonic deaths and malformations was dose-related and detected at doses of 2.8 mol/egg and above. The combined ED50 was 2.7 mol/egg.
Reproductive Toxicity
A 1% solution of hydrogen peroxide (equivalent to 1900 mg/kg/day) given as the sole drinking fluid to three-month-old male mice for 7-28 days did not cause infertility.
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.

UREA HYDROGEN PEROXIDE & HYDROGEN PEROXIDE

No significant acute toxicological data identified in literature search.

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. 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. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

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

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

SECTION 12 Ecological information

Toxicity

Pola Night 22% Carbamide Peroxide Gel

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

urea hydrogen peroxide

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	9-100mg/L	2
EC50	48	Crustacea	2mg/L	2
EC0	24	Crustacea	0.9mg/L	2
NOEC	48	Crustacea	1.5mg/L	2

hydrogen peroxide

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96	Fish	16.4mg/L	2
EC50	48	Crustacea	2mg/L	2
EC50	72	Algae or other aquatic plants	0.85mg/L	2
NOEC	72	Algae or other aquatic plants	=0.1mg/L	1

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 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*

DO NOT discharge into sewer or waterways.

Persistence and degradability

Continued...

Ingredient	Persistence: Water/Soil	Persistence: Air
hydrogen peroxide	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
hydrogen peroxide	LOW (LogKOW = -1.571)

Mobility in soil

Ingredient	Mobility
hydrogen peroxide	LOW (KOC = 14.3)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	Consult manufacturer for recycling options. Consult State Land Waste Management Authority for disposal. Bury residue in an authorised landfill. Decontaminate empty containers.

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO

Land transport (DOT): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

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

Not Applicable
If packed as Chemical kits the following classification may be considered if all ICAO/IATA transport requirements are met: Chemical Kit UN3316 - Class 9, SP A44 & A163.

SECTION 15 Regulatory information

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

urea hydrogen peroxide is found on the following regulatory lists

US DOE Temporary Emergency Exposure Limits (TEELs)
US List of Active Substances Exempt from the TSCA Inventory Notifications (Active-Inactive) Rule
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

hydrogen peroxide is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
US ACGIH Threshold Limit Values (TLV)
US AIHA Workplace Environmental Exposure Levels (WEELs)
US Department of Homeland Security (DHS) - Chemical Facility Anti-Terrorism Standards (CFATS) - Chemicals of Interest
US DOE Temporary Emergency Exposure Limits (TEELs)
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Levels (PELs) - Table Z1
US OSHA Permissible Exposure Limits - Annotated Table Z-1
US SARA Section 302 Extremely Hazardous Substances
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

Federal Regulations

Superfund Amendments and Reauthorization Act of 1986 (SARA)

Section 311/312 hazard categories

Flammable (Gases, Aerosols, Liquids, or Solids)	No
Gas under pressure	No
Explosive	No
Self-heating	No
Pyrophoric (Liquid or Solid)	No
Pyrophoric Gas	No
Corrosive to metal	No
Oxidizer (Liquid, Solid or Gas)	No
Organic Peroxide	No
Self-reactive	No
In contact with water emits flammable gas	No
Combustible Dust	No

Continued...

Carcinogenicity	No
Acute toxicity (any route of exposure)	No
Reproductive toxicity	No
Skin Corrosion or Irritation	No
Respiratory or Skin Sensitization	No
Serious eye damage or eye irritation	Yes
Specific target organ toxicity (single or repeated exposure)	No
Aspiration Hazard	No
Germ cell mutagenicity	No
Simple Asphyxiant	No
Hazards Not Otherwise Classified	No

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

None Reported

State Regulations

US. California Proposition 65

None Reported

National Inventory Status

National Inventory	Status
Australia - AIIC	Yes
Australia - Non-Industrial Use	No (urea hydrogen peroxide; hydrogen peroxide)
Canada - DSL	No (urea hydrogen peroxide)
Canada - NDSL	No (hydrogen peroxide)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (urea hydrogen peroxide)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (urea hydrogen peroxide)
Vietnam - NCI	Yes
Russia - ARIPS	Yes
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 Other information

Revision Date	01/11/2019
Initial Date	04/11/2015

SDS Version Summary

Version	Issue Date	Sections Updated
9.1.1.1	24/05/2016	Acute Health (eye), Classification, Fire Fighter (fire/explosion hazard), Ingredients, Personal Protection (hands/feet), Transport, Transport Information
10.1.1.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification

Other information

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

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

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level

Continued...

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

The information contained in the Safety Data Sheet is based on data considered to be accurate, however, no warranty is expressed or implied regarding the accuracy of the data or the results to be obtained from the use thereof.

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end of SDS