421 Liquid Tin

Mektronics

Version No: **6.11**
Safety Data Sheet according to WHS and ADG requirements

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SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	421
Synonyms	SDS Code: 421-liquid, 421-125ML, 421-500ML
Other means of identification	Liquid Tin

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

Relevant identified uses	Tin plates copper circuits

Details of the supplier of the safety data sheet

Registered company name	**Mektronics**	**MG Chemicals (Head office)**
Address	Unit 3 8 Bonz Place, Seven Hills NSW 2147 Australia	9347 - 193 Street Surrey V4N 4E7 British Columbia Canada
Telephone	1300 788 701	+(1) 800-201-8822
Fax	1300 722 004	+(1) 800-708-9888
Website	www.mektronics.com.au	www.mgchemicals.com
Email	sales@mektronics.com.au	Info@mgchemicals.com

Emergency telephone number

Association / Organisation	**CHEMTREC Australia**	**Not Available**
Emergency telephone numbers	+(61) 2-9037-2994	Not Available
Other emergency telephone numbers	+(1) 703-527-3887	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 1C, Skin Sensitizer Category 1B, Carcinogenicity Category 2, Reproductive Toxicity Category 2, Chronic Aquatic Hazard Category 3
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HSIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

Hazard pictogram(s)	
SIGNAL WORD	**DANGER**

Hazard statement(s)

H302	Harmful if swallowed.
H314	Causes severe skin burns and eye damage.
H317	May cause an allergic skin reaction.
H351	Suspected of causing cancer.
H361	Suspected of damaging fertility or the unborn child.
H412	Harmful to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.

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P280	Wear protective gloves/protective clothing/eye protection/face protection.
P281	Use personal protective equipment as required.
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

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.
P308+P313	IF exposed or concerned: Get medical advice/attention.
P310	Immediately call a POISON CENTER or doctor/physician.
P363	Wash contaminated clothing before reuse.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
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.
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 in accordance with local regulations.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
16872-11-0	10	fluoboric acid
62-56-6	10	thiourea
13814-97-6	4	tin fluoroborate
10043-35-3	1	boric acid

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If there is evidence of severe skin irritation or skin burns: ▸ Avoid further contact. Immediately remove contaminated clothing, including footwear. ▸ Flush skin under running water for 15 minutes. ▸ Avoiding contamination of the hands, massage calcium gluconate gel into affected areas, pay particular attention to creases in skin. ▸ Contact the Poisons Information Centre. ▸ Continue gel application for at least 15 minutes after burning sensation ceases. ▸ If pain recurs, repeat application of calcium gluconate gel or apply every 20 minutes. ▸ If no gel is available, continue washing for at least 15 minutes, using soap if available. If patient is conscious, give six calcium gluconate or calcium carbonate tablets in water by mouth. ▸ Transport to hospital, or doctor, urgently.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay. For massive exposures: ▸ If dusts, vapours, aerosols, fumes or combustion products are inhaled, remove from contaminated area. ▸ Lay patient down. ▸ Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ If victim is conscious, give six calcium gluconate or calcium carbonate tablets in water by mouth.

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	▸ Transport to hospital, or doctor, urgently.
Ingestion	▸ For advice, contact a Poisons Information Centre or a doctor at once. ▸ Urgent hospital treatment is likely to be needed. ▸ 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. ▸ Transport to hospital or doctor without delay.

Indication of any immediate medical attention and special treatment needed

Following acute or short term repeated exposure to hydrofluoric acid:

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

BIOLOGICAL EXPOSURE INDEX - BEI

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

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

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

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

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

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

- There is no restriction on the type of extinguisher which may be used.
- Use extinguishing media suitable for surrounding area.

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.

Advice for firefighters

Fire Fighting	
Fire/Explosion Hazard	▸ Non combustible. ▸ Not considered to be a significant fire risk. ▸ Acids may react with metals to produce hydrogen, a highly flammable and explosive gas. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ May emit corrosive, poisonous fumes. May emit acrid smoke. Decomposition may produce toxic fumes of: nitrogen oxides (NOx) hydrogen fluoride hydrogen sulfide (H2S) May emit corrosive fumes.
HAZCHEM	2X

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	▸ Drains for storage or use areas should have retention basins for pH adjustments and dilution of spills before discharge or disposal of material. ▸ Check regularly for spills and leaks. ▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Control personal contact with the substance, by using protective equipment. ▸ Contain and absorb spill with sand, earth, inert material or vermiculite. ▸ Wipe up. ▸ Place in a suitable, labelled container for waste disposal.
Major Spills	

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	NOTE: Boron halides react violently with water, and if there is a deficiency of water, a violent explosion may occur. It is therefore highly dangerous to wash ampoules of boron halides (e.g boron tribromide) with water under any circumstances. Only dry non-polar solvents should be used for cleaning or cooling purposes. ▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ **WARNING:** To avoid violent reaction, **ALWAYS** add material to water and **NEVER** water to material. ▸ Avoid smoking, naked lights or ignition sources. ▸ 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.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Lined metal can, lined metal pail/ can. ▸ Plastic pail. ▸ Polyliner drum. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks. For low viscosity materials ▸ Drums and jerricans must be of the non-removable head type. ▸ Where a can is to be used as an inner package, the can must have a screwed enclosure. For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.): ▸ Removable head packaging; ▸ Cans with friction closures and ▸ low pressure tubes and cartridges may be used. - Where combination packages are used, and the inner packages are of glass, porcelain or stoneware, there must be sufficient inert cushioning material in contact with inner and outer packages unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	Salts of inorganic fluoride: ▸ react with water forming acidic solutions. ▸ are violent reactive with boron, bromine pentafluoride,bromine trifluoride, calcium disilicide, calcium hydride, oxygen difluoride, platinum, potassium. ▸ in aqueous solutions are incompatible with sulfuric acid, alkalis, ammonia, aliphatic amines, alkanolamines, alkylene oxides, amides, epichlorohydrin, isocyanates, nitromethane, organic anhydrides, vinyl acetate. ▸ corrode metals in presence of moisture ▸ may be incompatible with glass and porcelain Fluoboric acid: ▸ is a strong inorganic acid; reacts violently with strong bases ▸ reacts slowly with water forming hydroxyfluoborate ions ▸ is incompatible with strong oxidisers, acetic anhydride, sulfuric acid, caustics, carbonates, ammonia, aliphatic amines, alkanolamines, amides, organic anhydrides, isocyanates, vinyl acetate, alkylene oxides, epichlorohydrin, sulfides ▸ attacks metals forming flammable hydrogen gas ▸ Inorganic acids are generally soluble in water with the release of hydrogen ions. The resulting solutions have pH's of less than 7.0. ▸ Inorganic acids neutralise chemical bases (for example: amines and inorganic hydroxides) to form salts - neutralisation can generate dangerously large amounts of heat in small spaces. ▸ The dissolution of inorganic acids in water or the dilution of their concentrated solutions with additional water may generate significant heat. ▸ The addition of water to inorganic acids often generates sufficient heat in the small region of mixing to cause some of the water to boil explosively. The resulting 'bumping' can spatter the acid. ▸ Inorganic acids react with active metals, including such structural metals as aluminum and iron, to release hydrogen, a flammable gas. ▸ Inorganic acids can initiate the polymerisation of certain classes of organic compounds. ▸ Inorganic acids react with cyanide compounds to release gaseous hydrogen cyanide. ▸ Inorganic acids generate flammable and/or toxic gases in contact with dithiocarbamates, isocyanates, mercaptans, nitrides, nitriles, sulfides, and strong reducing agents. Additional gas-generating reactions occur with sulfites, nitrites, thiosulfates (to give H2S and SO3), dithionites (SO2), and even carbonates. ▸ Acids often catalyse (increase the rate of) chemical reactions. ▸ Contact with acids produces toxic fumes ▸ Reacts with mild steel, galvanised steel / zinc producing hydrogen gas which may form an explosive mixture with air. Thiourea ▸ is basic in aqueous solutions ▸ reacts violently with acrolein, strong acids ▸ is incompatible with acrylaldehyde, hydrogen peroxide, metal salts ▸ aqueous solutions are incompatible with organic anhydrides, acrylates, alcohols, aldehydes, alkylene oxides, substituted allyls, cresols, caprolactam solutions, epichlorohydrin, ethylene dichloride, glycols, hydrogen peroxide, isocyanates, ketones, maleic anhydride, nitrates, nitromethane, phenols, vinyl acetate

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SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	tin fluoroborate	Tin oxide & inorganic compounds, except SnH4 (as Sn)	2 mg/m3	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
fluoboric acid	Fluoboric acid; (Tetrafluoroboric acid)	8.7 mg/m3	97 mg/m3	580 mg/m3
thiourea	Thiourea	0.38 mg/m3	4.1 mg/m3	25 mg/m3
tin fluoroborate	Tin fluoroborate	15 mg/m3	170 mg/m3	990 mg/m3
boric acid	Boric acid	6 mg/m3	23 mg/m3	830 mg/m3

Ingredient	Original IDLH	Revised IDLH
fluoboric acid	Not Available	Not Available
thiourea	Not Available	Not Available
tin fluoroborate	100 mg/m3	Not Available
boric acid	Not Available	Not Available

MATERIAL DATA

For inorganic borates and tetraborates:
No data are currently available to establish a causal link between inhalation exposures to sodium tetraborates and chronic respiratory and/or systemic effects.
An occupationally important toxic effect of the sodium tetraborates is their acute irritant effect when in contact with skin and the mucous membranes of the eyes, nose and other sites of the respiratory tract. The irritant properties increase with decreasing water of hydration due to the exothermic effect of hydration. The TLV-TWA of 1 mg/m3 for the anhydrous and pentahydrate forms and 5 mg/m3 for the decahydrate is thought to be protective against the acute irritant effects.

For fluorides:
Based on a study in which the threshold for minimum increase in bone density due to fluoride exposure was 3.38 mg/m3 (as fluoride), the present TLV-TWA has been adopted to prevent irritant effects and disabling bone changes. There is also support for the proposition that occupational exposure below the TLV will have no adverse effect on pregnant women or off-spring. IARC has classified fluorides in drinking water as Group 3 carcinogens; i.e. Not classifiable as to its carcinogenicity to humans. Equivocal evidence of carcinogenic activity (osteosarcoma) has been found in male rats administered sodium fluoride in drinking water. (0-175 ppm) Evidence was not found in female rats or in male or female mice.

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.

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection.
Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.
An approved self contained breathing apparatus (SCBA) may be required in some situations.
Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying 'escape' velocities which, in turn, determine the 'capture velocities' of fresh circulating air required to effectively remove the contaminant.

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.

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Personal protection	
Eye and face protection	▸ Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure. ▸ Chemical goggles.whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted. ▸ Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection. ▸ Alternatively a gas mask may replace splash goggles and face shields. ▸ 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	▸ Elbow length PVC gloves ▸ When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots. **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	▸ Overalls. ▸ PVC Apron. ▸ PVC protective suit may be required if exposure severe. ▸ Eyewash unit. ▸ Ensure there is ready access to a safety shower.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
'Forsberg Clothing Performance Index'.
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
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Material	CPI
BUTYL	C
NEOPREN	C
NITRIL	C
PE/EVAL/PE	C
VITO	C

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as 'feel' or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Water white to light brown

Physical state	Liquid	**Relative density (Water = 1)**	1.12
Odour	Mild	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	<1	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	>100	**Molecular weight (g/mol)**	Not Available
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

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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	▸ Contact with alkaline material liberates heat
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. Acute effects of fluoride inhalation include irritation of nose and throat, coughing and chest discomfort. Even brief exposure to high concentrations of inorganic fluoride may cause sore throat, chest pains, pulmonary oedema,and in rare cases irreparable damage to the lungs, and death A single acute over-exposure may cause nose bleed. Pre-existing respiratory conditions such as emphysema, bronchitis may be aggravated by exposure. Occupational asthma may result from exposure. Acidic corrosives produce respiratory tract irritation with coughing, choking and mucous membrane damage. Symptoms of exposure may include dizziness, headache, nausea and weakness. In more severe exposures, pulmonary oedema may be evident either immediately or after a latent period of 5-72 hours. Symptoms of pulmonary oedema include a tightness in the chest, dyspnoea, frothy sputum and cyanosis. Examination may reveal hypotension, a weak and rapid pulse and moist rates. Death, due to anoxia, may occur several hours after onset of the pulmonary oedema. The material has **NOT** been classified by EC Directives or other classification systems as 'harmful by inhalation'. This is because of the lack of corroborating animal or human evidence. In the absence of such evidence, care should be taken nevertheless to ensure exposure is kept to a minimum and that suitable control measures be used, in an occupational setting to control vapours, fumes and aerosols. Borates, as represented by borax, may act as simple respiratory irritants. In a study of the respiratory effects of borax dust on active borax workers, the incidence of respiratory symptoms, pulmonary function and abnormalities of chest radiographs were related to estimated exposures. Dryness of the mouth, nose or throat, dry cough, nose bleeds, sore throat, productive cough, shortness of breath and chest tightness were related to exposures of 4 mg/m3 or more
Ingestion	Fluoride is a general protoplasmic poison which appears to produce at least four major functional derangements; (1) enzyme inhibition, (2) hypocalcaemia, (3) cardiovascular collapse and (4) specific organ damage. Hypocalcaemia which leads to severe reductions in plasma levels of both total calcium and ionic calcium, may appear several hours after exposure producing painful and involuntary muscular contractions (tetany) initially of the extremities (carpopedal spasm, twitching of limb muscles, laryngo-spasm, cardiospasm etc). Cardiovascular collapse is probably the principal cause of death in acute fluoride poisoning with sinus tachycardia the commonest cardiac finding and serious cardiac arrhythmias also common. Poisonings also cause major adverse effects on the brain and kidneys. Toxic effects may include headache, excessive salivation, rapid movements of the eyeball (nystagmus) and dilated pupils. Convulsions may occur but lethargy, stupor and coma are more common. Renal pathology (acute congestion) has been described in human casualties. Ingestion of acidic corrosives may produce circumoral burns with a distinct discolouration of the mucous membranes of the mouth, throat and oesophagus. Immediate pain and difficulties in swallowing and speaking may also be evident. Oedema of the epiglottis may produce respiratory distress and possibly, asphyxia. Nausea, vomiting, diarrhoea and a pronounced thirst may occur. More severe exposures may produce a vomitus containing fresh or dark blood and large shreds of mucosa. Shock, with marked hypotension, weak and rapid pulse, shallow respiration and clammy skin may be symptomatic of the exposure. Circulatory collapse may, if left untreated, result in renal failure. Severe cases may show gastric and oesophageal perforation with peritonitis, fever and abdominal rigidity. Stricture of the oesophageal, gastric and pyloric sphincter may occur as within several weeks or may be delayed for years. Death may be rapid and often results from asphyxia, circulatory collapse or aspiration of even minute amounts. Delayed deaths may be due to peritonitis, severe nephritis or pneumonia. Coma and convulsions may be terminal. 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. Thiourea, diethylthiourea and tetramethylthiourea have been used clinically to produce antithyroid effects in humans. Commonly observed side-effects of treatments include headache, anxiety, fever, skin rash and gastrointestinal disturbance. Ingestion or percutaneous absorption of boric acid causes nausea, abdominal pain, diarrhoea and violent vomiting, sometimes bloody, which may be accompanied by headache and weakness, and characteristic erythematous (abnormally red) lesions on the skin. In severe cases, shock with fall in arterial pressure, tachycardia (increase in heart rate) and cyanosis (blue skin colour) may occur. Marked central nervous system irritation, oliguria (small volume of urine), and anuria (absence of or defective excretion of urine) may be present. Symptoms of borate poisoning include nausea, vomiting, diarrhoea, epigastric pain. These may be accompanied headache, weakness and a distinctive red skin rash. In severe cases there may be shock, increased heart rate and the skin may appear blue. Vomiting (which may be violent) is often persistent and vomitus and faeces may contain blood. Weakness, lethargy, headache, restlessness, tremors and intermittent convulsions may also occur. Poisoning produces central nervous system stimulation followed by depression, gastrointestinal disturbance (haemorrhagic gastro-enteritis), erythematous skin eruptions (giving rise to a boiled lobster appearance) and may also involve kidneys (producing oliguria, albuminuria, anuria) and, rarely, liver (hepatomegaly, jaundice). Toxic symptoms may be delayed for several hours. Ingested borates are readily absorbed and do not appear to be metabolised via the liver. Excretion occurs mainly through the kidneys in the urine with about half excreted in the first 12 hours and the remainder over 5-12 days. Borates are excreted primarily in the urine regardless of the route of administration. The borates (tetra-, di-, meta, or ortho- salts, in contrast to perborates) once solubilised in the acid of gastric juices, cannot be distinguished from each other on chemical or toxicological grounds. In humans acute gastroenteric (or percutaneous absorption of as little as 1 gm of sodium borate can result in severe gastrointestinal irritation, kidney damage. In adults the mean lethal dose of sodium borate or boric acid probably exceeds 30 gms (Gosselin) and death occurs due to vascular collapse in the early stages or to central nervous system depression in later stages. Children are thought to be more susceptible to the effects of borate intoxication.

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Skin Contact	Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions. Skin contact with acidic corrosives may result in pain and burns; these may be deep with distinct edges and may heal slowly with the formation of scar tissue. Boric acid is not absorbed through intact skin but is readily absorbed through areas of damaged, abraded, burned skin, areas of active dermatitis Contact of the skin with liquid hydrofluoric acid (hydrogen fluoride) may cause severe burns, erythema, and swelling, vesiculation, and serious crusting. With more serious burns, ulceration, blue-gray discoloration, and necrosis may occur. Solutions of hydrofluoric acid, as dilute as 2%, may cause severe skin burns. 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. Direct eye contact with acid corrosives may produce pain, lachrymation, photophobia and burns. Mild burns of the epithelia generally recover rapidly and completely. Severe burns produce long-lasting and possible irreversible damage. The appearance of the burn may not be apparent for several weeks after the initial contact. The cornea may ultimately become deeply vascularised and opaque resulting in blindness.
Chronic	On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. 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. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Long term exposure to vapour or dust with inorganic fluorides may result in fluorosis, with rheumatic symptoms, stiff joints, mottling of tooth enamel. Other signs may include nausea, vomiting, anorexia, diarrhoea or constipation, weight loss, anaemia, weakness and general ill-health. Polyuria and polydipsia may also occur. Exfoliative dermatitis, atopic dermatitis, stomatitis, gastrointestinal and respiratory allergy, and on occasions, central nervous system involvement have all been described. Thiourea is a sensitiser in persons who exhibit photosensitivity. Chronic exposure may result in damage to the blood, liver and thyroid. Thiourea inhibits utilisation of Iodine and has a haemolytic effect (impedes blood clotting). Thiourea has produced goiter and bone marrow depression (anaemia, leukopenia, thrombocytopenia and agranulocytosis) in experimental animals. When administered in the drinking water, thiourea induced thyroid adenomas and carcinomas in rats of both sexes and squamous cell carcinomas of the Zymbal gland in male rats. When administered in the diet, thiourea induced hepatocellular adenomas in rats. The mechanism by which thioureas exert the antithyroid effect involves the inhibition of iodine uptake and activation by the thyroid. At low doses, a physiological and biological compensation mechanism maintains normal levels of circulating thyroid hormone. Prolonged exposure to high doses of thyroid inhibitors causes severe hypertrophy and hyperplasia resulting in reduced levels of circulating thyroid hormone. Positive mutagenic effects have been elicited by the use of several thiourea derivatives in various assays. Teratogenic responses have been recorded with alkylated thioureas and ethylene thiourea in various species. Repeated or prolonged exposure to acids may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis. The impact of inhaled acidic agents on the respiratory tract depends upon a number of interrelated factors. These include physicochemical characteristics, e.g., gas versus aerosol; particle size (small particles can penetrate deeper into the lung); water solubility (more soluble agents are more likely to be removed in the nose and mouth). Given the general lack of information on the particle size of aerosols involved in occupational exposures to acids, it is difficult to identify their principal deposition site within the respiratory tract. Acid mists containing particles with a diameter of up to a few micrometers will be deposited in both the upper and lower airways. They are irritating to mucous epithelia, they cause dental erosion, and they produce acute effects in the lungs (symptoms and changes in pulmonary function). AsthmatIcs appear to be at particular risk for pulmonary effects. Chronic boric acid poisoning is characterized by mild gastrointestinal irritation, loss of appetite, disturbed digestion, nausea, possibly vomiting and a hard blotchy rash. Dryness of skin, reddening of tongue, loss of hair, conjunctivitis, and kidney injury have also been reported. [Occupational Diseases] Long term exposure to boric acid may be of more concern, causes kidney damage and eventually kidney failure. Although it does not appear to be carcinogenic, studies in dogs have reported testicular atrophy after exposure to 32 mg/kg bw/day for 90 days. This level is far lower than the LD50. Boric acid in high doses shows significant developmental toxicity and teratogenicity in rabbit, rat, and mouse foetuses as well as cardiovascular defects, skeletal variations, mild kidney lesions. The mechanism of action by which boric acid causes testicular toxicity has been investigated and it has been proposed that decreased testosterone production arises via a CNS mediated mechanism. It is not likely that hormone changes can explain the testicular atrophy observed at high dose levels since it has been shown that spermatogenesis can be maintained in the presence of significantly decreased intra-testicular testosterone levels. The fact that testicular damage was reversible and less extensive in younger sexually immature males than in mature animals also argues against an endocrine disruptor mechanism because younger animals still in development may be expected to be more sensitive to anti-androgenic effects than adults. Inhibition of spermiation has been investigated and the involvement of Sertoli cells is suggested, as effects on these cells can lead to testicular atrophy. The changes in serum hormone levels may reflect an indirect effect on the CNS mediated by paracrine and/or autocrine influences. Chronic poisoning by borates may be characterised gastrointestinal disturbances and skin rash. Chronic absorption of small amounts of borax causes mild gastroenteritis and dermatitis. Chronic feeding studies involving borate administration to rats and dogs leads to accumulation in the testes, germ cell depletion and testicular atrophy. Hair loss in a young woman was traced to chronic ingestion of boric acid-containing mouthwashes whilst hair loss, dermatitis, gastric ulcer and hypoplastic anaemia in an adult male was attributed to the consumption of an uncharacterised 'boric tartrate' for 20 years (symptoms disappeared following withdrawal). Repeated ingestion or inhalation of sub-acute doses of boric acid produces gastrointestinal irritation and disturbance, loss of appetite, disturbed digestion, nausea and vomiting, erythematous rash which may become hard and purpuric, dryness of the skin and mucous membranes, reddening of the tongue, cracking of the lips, conjunctivitis, palpebral oedema and kidney injury. Workers exposed to dust levels containing in excess of 31 mg/m3 boric acid, showed atrophic and subatrophic changes of the respiratory mucous membranes. Prolonged ingestion by animals produces a variety of reproductive effects including changes to the ovaries, fallopian tubes, the testes, epididymis and sperm ducts.

421 Liquid Tin	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

fluoboric acid	**TOXICITY**	**IRRITATION**
	Oral (rat) LD50: 100 mg/kg[2]	Not Available

Continued...

thiourea	**TOXICITY** / **IRRITATION** dermal (rat) LD50: >6810 mg/kg[2] / Eye (rabbit): 14% Oral (rat) LD50: 125 mg/kg[2] /
tin fluoroborate	**TOXICITY** / **IRRITATION** Dermal (rabbit) LD50: ~3000 mg/kg[1] / Not Available Oral (rat) LD50: 100 mg/kg[2] /
boric acid	**TOXICITY** / **IRRITATION** Dermal (rabbit) LD50: >2000 mg/kg[2] / Skin (human): 15 mg/3d -I- mild Oral (rat) LD50: 2500 mg/kg[2] /
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*

421 Liquid Tin	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
FLUOBORIC ACID	No significant acute toxicological data identified in literature search. 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. The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may produce respiratory tract irritation. Symptoms of pulmonary irritation may include coughing, wheezing, laryngitis, shortness of breath, headache, nausea, and a burning sensation. Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence). The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties. 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 the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
THIOUREA	for thiourea: There are reports on disorders of workers coming into contact with thiourea during the course of, for example, maintenance of machinery or packing, without providing any details as to exposure levels. The symptoms observed were typical of hypothyroidism, as evidenced by facial oedema, hypotonia, bradycardia, electrocardiograph alterations associated with reduced basal metabolism, constipation, flatulence, polyuria, and granulocytopenia, accompanied by lymphocytosis and monocytosis. The first perturbations of the blood count were observed after 5-6 months of exposure, and the highest incidence of the symptoms was evident in those workers who had been in contact with the chemical for 5-15 years Individual cases of contact dermatitis related to the use or processing of thiourea and thiourea compounds have been reported. Some cases showed increased sensitivity to UV light (photocontact dermatitis). Thiourea derivatives such as dimethyl, diethyl, dibutyl, diphenyl, ethylbutyl, and ethylene thiourea are used as accelerators in the vulcanization process in the rubber industry. Products such as wet suits, swimming goggles, orthopaedic devices, protective gloves, and shoes containing these compounds have been shown to produce allergic contact dermatitis. Administration of thiourea to healthy animals or humans leads to depression of thyroid function. It acts by inhibiting the peroxidase in the thyroid gland, resulting in decreased thyroid hormone production and increased proliferation due to an increase in the secretion of TSH. This could lead to tumour formation. This is a well recognised mechanism of action for non-genotoxic thyroid carcinogens However, no definite conclusion regarding the mechanism of carcinogenicity can be made for thiourea, since it cannot totally be excluded that the possible genotoxicity of thiourea also plays a role. In humans and animals, thiourea is rapidly absorbed from the gastrointestinal tract. A single oral dose of 28.57 mg thiourea/kg body weight in humans was completely eliminated within 48 h in urine, while a peak concentration in blood was measured within 30 min. In rats administered 5 mg intravenously, 30% of the thiourea was recovered from the carcasses after 3 h, and only traces after 25 h. Thiourea is also absorbed to a lesser degree through the skin. Following dermal application of 2000 mg/kg body weight to rabbits in the form of an aqueous solution (26 ml of a 25% w/v solution), approximately 4% of the applied dose was found in the animals' urine; when applied in solid form, only 0.1% was found in the urine. Thiourea is oxidised by thyroid gland peroxidase in the presence of iodine or iodide and hydrogen peroxide to form formamidine disulfide (NH2(NH)CSSC(NH)NH2). Formamidine disulfide is unstable and decomposes at pH values above 3.0, forming cyanamide, elementary sulfur, and thiourea. It was shown *in vitro* and *in vivo* that both cyanamide and thiourea are inhibitors of thyroid peroxidase The acute toxicity of thiourea varies with the species, strain, and age of the animals exposed to the chemical and with the iodine content of their diet. Oral LD50s are about 1000 mg/kg body weight for mice, 125-1930 mg/kg body weight for rats, depending on the strain, and 10 000 mg/kg body weight for rabbits. The intraperitoneal LD50 for the rat ranges between 4 and 1340 mg/kg body weight, according to the strain. Death at these doses is due to lung oedema, and the survivors exhibit pleural effusion. Accordingly, thiourea at doses between 10 and 500 mg/kg body weight has been employed in experimental animal studies as a model agent for the elicitation of lung oedema and pleural effusion. The pathological effects are prevented by pretreatment

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	of the animals with cysteine or glutathione, which reduces the irreversible binding of radioactivity to lung proteins after administration of [14C] thiourea. Toxic doses of thiourea also resulted in hyperglycaemia, glucosuria, polyuria, and a reduction in the liver glycogen level in rats. **Irritation and sensitisation:** A 24-h exposure to undiluted thiourea applied to the intact and abraded skin of rabbits resulted in mild to marked erythema with a slight degree of oedema. When rabbit skin was exposed to 0.5 g of thiourea for a period of 4 h, the substance was tolerated without reaction. A single application of a 10% (w/w) aqueous solution of thiourea to the eye was tolerated without reaction. In another study, the application of 100 mg thiourea to the conjunctiva of the rabbit eye resulted in reddening (1-2 using Draize scoring) and swelling (1-2 using Draize scoring). Thiourea yielded negative results in a sensitization test carried out with guinea-pigs according to the method of Magnusson & Kligman. **Short term exposure:** The iodine level of the thyroid gland was reduced from 73 to 13 mg/100 g tissue upon the oral administration of thiourea at 70 mg/kg body weight for 10 days. Thiourea also resulted in a reduction of thyroid iodine uptake when administered in rats at 1% (500 mg/kg body weight per day) in the diet for 2 months. Concomitant with reduced thyroid activity, the weight of the pituitary gland increased and signs of pituitary overactivity were evident both histologically and biochemically; the weights of the ovary, uterus, and prostate gland all declined. Haemosiderosis in the spleen, lymph nodes, and intestinal villi of rats was observed subsequent to the administration of 16-50 daily doses of 1 ml of a 1% aqueous solution of thiourea by gavage. The repeated administration of high doses (no quantitative data given) of thiourea in the diet, in the drinking-water, or by intraperitoneal injection resulted in manifold effects: reduced osmotic resistance of the erythrocytes, congestion, haemosiderosis and atrophy of the spleen, anaemia, leukocytopenia, granulocytopenia, increased erythropoiesis in the bone marrow, reduced clotting times, and increased phospholipid levels of the blood. **Long-term exposure and carcinogenicity:** In a chronic toxicity study, thiourea was administered daily in drinking-water at concentrations of 1.72, 6.88, or 27.5 mg/kg body weight to mice for 2 years and to rats for the duration of their lifetimes or a maximum of 3 years. A reduction in body weight gain and an enlargement of the thyroid gland were observed only in the rats in the highest dose group, and no other changes were detected, either macroscopically or microscopically. A lowest-observed-adverse-effect level (LOAEL) of 27.5 mg/kg body weight per day (reduction of body weight and enlargement of thyroid gland) and a no-observed-adverse-effect level (NOAEL) of 6.88 mg/kg body weight per day for rats can be given. Thiourea has not been tested in a standard bioassay of carcinogenicity in rodents. Several older carcinogenicity studies, of doubtful quality, were carried out prior to the mid-1960s They described the occurrence of tumours at numerous locations other than the thyroid gland, but the distribution of these varied from one study to another. In several studies involving different strains of mice, thyroid hyperplasia, but not thyroid tumours, was reported after oral administration. In rats given thiourea orally, a high incidence of thyroid follicular cell adenomas and carcinomas and increased incidences of hepatocellular adenomas and tumours of the Zymbal or Meibomian gland were reported **Genotoxicity and related end-points:** Thiourea has been tested in numerous assays. It did not induce gene mutations in bacteria. Inconsistent results, the majority of which were negative, were obtained in mammalian cells. Thiourea induced chromosomal recombination in yeast and insects. Thiourea is not considered to be a genotoxic carcinogen. **Mitogenic effects:** Thiourea has mitogenic properties. Older studies with high doses of thiourea (0.4 g, 1-14 times, intraperitoneal; unclear whether per animal or per kg body weight) produced a high mitosis rate in the liver without hepatocellular necrosis. Studies on partially hepatectomized rats showed similar results. **Effects on fertility:** Thiourea can affect fertility as a result of hypothyroidism. Thiourea was included in the diet of rats at concentrations of between 0.01 and 1% for 24 months, which were equivalent to doses ranging from 5 to 500 mg/kg body weight per day. A reduction or cessation of spermatogenesis and effects on the thyroid gland or other organs were observed at doses higher than 35 mg/kg body weight per day. **Developmental toxicity:** Thiourea had neither a maternally toxic nor a teratogenic effect when administered to rats on the 12th or 13th day of gestation as a single oral dose of 480 mg/kg body weight. In a study in which 66 female sheep (18 growing lambs, 18 maiden ewes, 9 pregnant ewes; controls: 9 growing lambs, 9 maiden ewes, 3 pregnant ewes) were orally administered 0 or 50 mg thiourea/kg body weight daily for 2, 4, or 6 months (six treated and three controls per group), external genitalia were infantile and stunted in growing lambs, while they were pale anaemic and dry in maiden ewes. None of the growing lambs showed signs of oestrus. Mammary development was retarded Thiourea was shown to cross the placenta in mice and rats and to be preferentially stored in the thyroid gland, depending on the stage of development of this organ, where it affects iodine metabolism. In a study in which groups of CF4 rats were treated with 0.2% thiourea in the drinking-water on days 1-14 of gestation, growth retardation and malformations of the nervous system and skeleton were present in treated offspring, although specific incidences of foetal effects were not given. **Immunological, neurological, or other effects:** Acute intoxication with thiourea has been linked with an increase in the level of histamine in the lungs and plasma (4.38 ug histamine/100 ml plasma was determined for rats administered thiourea intraperitoneally at 10 mg/kg body weight compared with 2.08 ug/100 ml in the controls) and with an increase in lung vessel permeability. Rats developed tolerance to an otherwise lethal dose of thiourea (10 mg/kg body weight) when pretreated with a non-lethal dose (0.5 mg/kg body weight) over a period of 8 days. This tolerance was accompanied by a reduction in both lung vessel permeability and plasma histamine levels The oedema-inducing effect of thiourea is probably due to the action of its oxidation product cyanamide and can be alleviated by treatment with hydroxyl radical scavengers such as dimethyl sulfoxide, ethanol, or mannitol. The adverse action of thiourea on the lungs of rats injected intraperitoneally with 0.3 mg/kg body weight could also be diminished by intraperitoneal treatment with the antiarrhythmic agents procainamide (at 4 mg/kg body weight), quinidine gluconate (20 mg/kg body weight), and lidocaine (30 mg/kg body weight). Treatment *in vitro* with 75 mmol thiourea/litre results in an inhibition of interleukin-8 production in human whole blood, the toxic effect of which can be suppressed by the administration of glutathione or cysteine. Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [*National Toxicology Program: U.S. Dep. of Health & Human Services 2002*] 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. Product: Oral (rat) >5000 mg/kg Dermal (rabbit) >2800 mg/kg [Orica] Respiratory tract changes, multiple lung effects, haemorrhage, granulocytopenia, specific developmental abnormalities involving central nervous system, musculoskeletal system, endocrine system recorded.
TIN FLUOROBORATE	* Megachem SDS
BORIC ACID	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.
421 Liquid Tin & FLUOBORIC ACID & TIN FLUOROBORATE	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.
FLUOBORIC ACID & TIN FLUOROBORATE	for acid mists, aerosols, vapours Data from assays for genotoxic activity in vitro suggest that eukaryotic cells are susceptible to genetic damage when the pH falls to about 6.5. Cells from the respiratory tract have not been examined in this respect. Mucous secretion may protect the cells of the airways from direct exposure to inhaled acidic mists, just as mucous plays an important role in protecting the gastric epithelium from its auto-secreted hydrochloric acid. In considering whether pH itself induces genotoxic events in vivo in the respiratory system, comparison should be made with the human stomach, in which gastric juice may be at pH 1-2 under fasting or nocturnal conditions, and with the human urinary bladder, in which the pH of urine can range from <5 to > 7 and normally averages 6.2. Furthermore, exposures to low pH in vivo differ from exposures *in vitro* in that, *in vivo*, only a portion of the cell surface is subjected to the adverse conditions, so that perturbation of intracellular homeostasis may be maintained more readily than in vitro.

Acute Toxicity	✔	**Carcinogenicity**	✔

Continued...

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

421 Liquid Tin

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
Not Available	Not Available	Not Available	Not Available	Not Available

fluoboric acid

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	2-600mg/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

thiourea

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	>100mg/L	1
EC50	48	Crustacea	35mg/L	2
EC50	72	Algae or other aquatic plants	3.8-10mg/L	1
BCF	24	Algae or other aquatic plants	0.05mg/L	4
NOEC	504	Crustacea	0.11mg/L	1

tin fluoroborate

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	>1-mg/L	2
EC50	48	Crustacea	87mg/L	2
EC50	72	Algae or other aquatic plants	>100mg/L	2
NOEC	672	Fish	0.78mg/L	2

boric acid

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	74mg/L	2
EC50	48	Crustacea	133mg/L	4
EC50	96	Algae or other aquatic plants	15.4mg/L	2
NOEC	768	Fish	0.009mg/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*

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
Although small amounts of fluorides are conceded to have beneficial effects, two forms of chronic toxic effect, dental fluorosis and skeletal fluorosis may be caused by excessive intake over long periods. Fluorides are absorbed by humans following inhalation of workplace and ambient air that has been contaminated, ingestion of drinking water and foods and dermal contact.
Fluoride accumulates, food-dependently in skeletal tissues of both aquatic and terrestrial vertebrates and invertebrates. Bioaccumulation occurs in marine organisms and, to a lesser extend, fresh water organisms. Reported BCF-values for marine organisms range up to approximately 150 and 60 for fish and crustacea, respectively. The most important exposure route for plants is uptake from the atmosphere. Concentrations in plants in the vicinity of a HF production plant range up to approximately 200 mg/kg, with mean levels between 20 and 50 mg/kg dry weight. Generally, lowest fluoride levels are found in herbivores and (somewhat) higher levels in predators.
Fluorides have been shown to accumulate in animals that consume fluoride-containing foliage However, accumulation is primarily in skeletal tissue and therefore, it is unlikely that fluoride will biomagnify up the food chain.
Both hydrogen fluoride and particulate fluorides will be transported in the atmosphere and deposited on land or water by wet and dry deposition. Non-volatile inorganic fluoride particulates are removed from the atmosphere via condensation or nucleation processes. Fluorides adsorbed on particulate matter in the atmosphere are generally stable and are not readily hydrolysed, although they may be degraded by radiation if they persist in the atmosphere. Fluorine and the silicon fluorides (fluosilicates, silicofluorides) are hydrolysed in the atmosphere to form hydrogen fluoride. Hydrogen fluoride may combine with water vapour to produce an aerosol or fog of aqueous hydrofluoric acid. Based upon available data, inorganic fluoride compounds, with the exception of sulfur hexafluoride, are not expected to remain in the troposphere for long periods or to migrate to the stratosphere. Estimates of the residence time of sulfur hexafluoride in the atmosphere range from 500 to several thousand years. Fluoride in aerosols can be transported over large distances by wind or as a result of atmospheric turbulence. The distance travelled is determined by the deposition velocity of both the gaseous hydrogen fluoride and the fluorides in particulate form. Atmospheric fluorides may be transported to soils and surface waters through both wet and dry deposition processes.
Fluorides undergo transformations in soil and water, forming complexes and binding strongly to soil and sediment.
In water, the transport and transformation of inorganic fluorides are influenced by pH, water hardness and the presence of ion-exchange materials such as clays. In natural water, fluoride forms strong complexes with aluminum in water, and fluorine chemistry in water is largely regulated by aluminum concentration and pH. Below pH 5, fluoride is almost entirely complexed with aluminum and consequently, the concentration of free F- is low. As the pH increases, Al-OH complexes dominate over Al-F complexes and the free F- levels increase. Fluoride forms stable complexes with

Continued...

calcium and magnesium, which are present in sea water. Calcium carbonate precipitation dominates the removal of dissolved fluoride from sea water. The residence time for fluoride in ocean sediment is calculated to be 2-3 million years. Fluorosilicic acid and hydrofluoric acid in high aquatic concentrations such as may be found in industrial waste ponds may volatilise, releasing silicon tetrafluoride and hydrogen fluoride into the atmosphere.
Solubilisation of inorganic fluorides from minerals may also be enhanced by the presence of ion-exchange materials (e.g., bentonite clays and humic acid). Once dissolved, inorganic fluorides remain in solution under conditions of low pH and hardness and in the presence of ion-exchange material. Soluble inorganic fluorides may also form aerosols at the air?water interface or vaporise into the atmosphere whereas undissolved species generally undergo sedimentation.
Factors that influence the mobility of inorganic fluorides in soil are pH and the formation of aluminium and calcium complexes In more acidic soils, concentrations of inorganic fluoride were considerably higher in the deeper horizons. The low affinity of fluorides for organic material results in leaching from the more acidic surface horizon and increased retention by clay minerals and silts in the more alkaline, deeper horizons. The maximum adsorption of fluoride to soil was reported to occur at pH 5.5. In acidic soils with pH below 6, most of the fluoride is in complexes with either aluminium or iron. Fluoride in alkaline soils at pH 6.5 and above is almost completely fixed in soils as calcium fluoride, if sufficient calcium carbonate is available. Fluoride is extremely immobile in soil, as determined by lysimeter experiments.
Populations living in areas with high fluoride levels in groundwater may be exposed to higher levels of fluorides in their drinking water or in beverages prepared with the water. Among these populations, outdoor laborers, people living in hot climates, and people with polydipsia will generally have the greatest daily intake of fluorides because they consume greater amounts of water.
Foods characteristically high in fluoride content are certain types of fish and seafood (1.9-28.5 mg/kg), especially those types in which the bones are consumed, bone products such as bone meal and gelatin, and tea, which contains approximately 0.52 mg fluoride/cup
Fluoride is mainly absorbed by the body in the form of hydrogen fluoride, which has a p*K*a of 3.45. That is, when ionic fluoride enters the acidic environment of the stomach lumen, it is largely converted into hydrogen fluoride. Most of the fluoride that is not absorbed from the stomach will be rapidly absorbed from the small intestine.
For boron and borates:
Environmental fate:
Boron is generally found in nature bound to oxygen and is never found as the free element. Atmospheric boron may be in the form of particulate matter or aerosols as borides, boron oxides, borates, boranes, organoboron compounds, trihalide boron compounds, or borazines. Borates are relatively soluble in water, and will probably be removed from the atmosphere by precipitation and dry deposition. The half-life of airborne particles is usually on the order of days, depending on the size of the particle and atmospheric conditions.
Boron readily hydrolyses in water to form the electrically neutral, weak monobasic acid boric acid (H3BO3) and the monovalent ion, B(OH)4-. In concentrated solutions, boron may polymerise, leading to the formation of complex and diverse molecular arrangements. Because most environmentally relevant boron minerals are highly soluble in water, it is unlikely that mineral equilibria will control the fate of boron in water. Boron was found to not be significantly removed during the conventional treatment of waste water. Boron may, however, be co-precipitated with aluminum, silicon, or iron to form hydroxyborate compounds on the surfaces of minerals.
Waterborne boron may be adsorbed by soils and sediments. Adsorption-desorption reactions are expected to be the only significant mechanism that will influence the fate of boron in water. The extent of boron adsorption depends on the pH of the water and the chemical composition of the soil. The greatest adsorption is generally observed at pH 7.5-9.0. the single most important property of soil that will influence the mobility of boron is the abundance of amorphous aluminum oxide. The extent of boron adsorption has also been attributed to the levels of iron oxide, and to a lesser extent, the organic matter present in the soil, although other studies found that the amount of organic matter present was not important. The adsorption of boron may not be reversible in some soils. The lack of reversibility may be the result of solid-phase formation on mineral surfaces and/or the slow release of boron by diffusion from the interior of clay minerals.
It is unlikely that boron is bioconcentrated significantly by organisms from water. A bioconcentration factor (BCF) relates the concentration of a chemical in the tissues of aquatic and terrestrial animals or plants to the concentration of the chemical in water or soil. The BCFs of boron in marine and freshwater plants, fish, and invertebrates were estimated to be <100. Experimentally measured BCFs for fish have ranged from 52 to 198. These BCFs suggest that boron is not significantly bioconcentrated.
As an element, boron itself cannot be degraded in the environment; however, it may undergo various reactions that change the form of boron (e.g., precipitation, polymerization, and acid-base reactions) depending on conditions such as its concentration in water and pH. In nature, boron in generally found in its oxygenated form. In aqueous solution, boron is normally present as boric acid and borate ions, with the dominant form of inorganic boron in natural aqueous systems as undissociated boric acid. Boric acid acts as an electron acceptor in aqueous solution, accepting an hydroxide ion from water to form (B(OH)4)-ion. In dilute solution, the favored form of boron is B(OH)4. In more concentrated solutions (>0.1 M boric acid) and at neutral to alkaline pH (6–11), polymeric species are formed (e.g., B3O3(OH)4-, B5O6(OH)4-, B3O3(OH)52-, and B4O5(OH)42-)
Most boron compounds are transformed to borates in soil due to the presence of moisture. Borates themselves are not further degraded in soil. However, borates can exist in a variety of forms in soil. Borates are removed from soils by water leaching and by assimilation by plants.
The most appreciable boron exposure to the general population is likely to be ingestion of food and to a lesser extent in water. As boron is a natural component of the environment, individuals will have some exposure from foods and drinking water
Boron-containing salts (borates) are ubiquitous in the environment. Surface soil, unpolluted waterways and seawater all typically contain significant amounts of boron as borate. Boron is an essential micronutrient for healthy growth of plants, however, it can be harmful to boron sensitive plants in higher quantities. In some areas such as the American Southwest, boron occurs naturally in surface waters in concentrations that have been shown to be toxic to commercially important plants.
Based on the collected information regarding aquatic toxicity, boron is not regarded as dangerous to aquatic organisms. The concentration in treated municipal waste water is a factor 100 lower than the NOEC-value for *Daphnia magna*.
No quality criteria exist for the concentration of boron in soil and compost. Boron is added to farmland when sewage sludge is applied as a soil improving agent, but there is not sufficient data to evaluate its effect on soil organisms. Being an essential micro-nutrient, no adverse effects of boron are expected at low concentrations.
Ecotoxicity:
In aquatic environments low concentrations of borates generally promote the growth of algae, whereas higher concentrations inhibited algal growth. In a growth inhibition test with *Scenedesmus subspicatus,* an EC50 value of 34 mg B/l was determined. Boric acid toxicity in Daphnia 48 h-LC50 (static test) was found to be 95 mg B/l. In a separate study it was concluded that chronic effects of boron to Daphnia may occur at a concentration of > 10 mg/l.
The toxicity of boron in fish is often higher in soft water than in hard water. The acute toxicity of boron towards *Danio rerio* (96 h-LC50) has been determined to 14.2 mg B/l . In a fish early life stage test with rainbow trout NOEC levels of boron have been determined in the range between 0.009 and 0.103 mg B/l, whereas the EC50 ranged from 27 to 100 mg B/l dependent on the water hardness.
for thiourea:
BOD 5: 0.013
COD: 0.84
Environmental fate:
From its very low vapour pressure, a significant adsorption of thiourea onto airborne particles is not expected. Due to its solubility in water (137 g/litre at 20 C), the washout from the atmosphere by wet deposition (fog, rain, snow) is assumed to be significant.
From water solubility and vapour pressure data, a Henry's law constant in the range of 5.58 × 10-9 - 8.44 × 10-9 Pa·m3/mol can be calculated, indicating that thiourea is not expected to volatilise from aqueous solutions. Based on the physicochemical properties of thiourea and its use pattern, the hydrosphere is expected to be the main target compartment for this compound.
Soil sorption coefficients (*Koc*) in the range of 26-315 were determined in studies conducted according to OECD Guideline 106 (adsorption/desorption). The sorption of thiourea onto organic matter of three different soils may be characterized as low (spodosol) to moderate (entisol/alfisol). Neutral thiourea did not undergo any significant ion exchange or other sorption processes in investigations with sorbents such as pure quartz sand, quartz sand coated with polyvinyl alcohol, and quartz sand coated with a mixture of the clay mineral montmorillonite and polyvinyl alcohol. Based on its physicochemical properties, a significant evaporation of thiourea from soil is not to be expected.
Transformation: Thiourea is hydrolytically stable, as measured according to OECD Guideline A-79.74 D. Experimental data on direct photolysis are not available. From the UV spectrum of the substance, direct photolysis in air and water is not to be expected. The extinction coefficients epsilon(max) at lambda(max) (235 and 238 nm) are in the range of 11,000-12,590/mol per second. However, in the atmosphere, the main degradation pathway is probably the reaction of thiourea with hydroxyl radicals. An estimation of the photo-oxidation of thiourea by hydroxyl radicals revealed a half-life of 2.4 h. For the hydrosphere, specific rate constants for the reaction of thiourea with hydrated electrons and hydroxyl radicals are given as 3.0 × 10+9/mol per second (pH 6.4) and 4.7 × 10+9/mol per second (pH 7). Based on a hydroxyl radical concentration of 1 × 10-16 mol/litre in water, a half-life of 17 days can be calculated.
In two studies on ready biodegradability, no mineralisation of thiourea was observed. On the other hand, removal of up to 97% was reported from laboratory tests on inherent biodegradation (Semi-Continuous Activated Sludge, or SCAS, Test), in which the inoculum was very slowly adapted to increasing thiourea concentrations prior to incubation. Cultures of different fungi isolated from soil and grown on glucose and thiourea were shown to degrade thiourea more or less effectively. Whereas *Aspergillus glaucus*, *Penicillium citrinum*, and *Trichoderma viride* took up only 30–50% of an initial thiourea concentration of 0.01% even after long incubation periods of 46 and 106 days and converted not more than 15-17% of thiourea sulfur to sulfate , concentrations in the range of 0.1-0.5 g thiourea/litre were completely removed within 7 days of incubation by *Penicillium rugulosum* . Degradation of thiourea by soil microorganisms was observed Twenty-two per cent of an initial concentration of 1.5 g/litre was degraded within 1 week and 96% within 15 weeks of incubation. Thiourea concentrations exceeding 7.6 g/litre inhibited microbial transformation. In aerobic batch laboratory microcosm experiments, half-lives of 12.8 days (basic soil) and 18.7 days (acid soil) were determined. Although no abiotic controls were performed, removal of thiourea was attributed mainly to biotic processes, assuming abiotic mechanisms (e.g., oxidation, evaporation) to be of minor importance.
From the available degradation tests and taking into account the expected environmental distribution of thiourea, leaching of this compound from soil to ground-water seems possible, particularly under conditions unfavourable for biotic degradation.
Accumulation: Based on the available data on soil sorption, biodegradation in soil, and the calculated *Koc* value, accumulation of thiourea in the geosphere is unlikely.
Due to the low *n*-octanol/water partition coefficient bioaccumulation of thiourea is expected to be insignificant. This assumption is confirmed by the available experimental data. In a study conducted according to OECD Guideline 305C, bioconcentration factors determined for carp (*Cyprinus carpio*) were in the range of <0.2 to <2 (related to whole fish). In another study reported accumulation factors were in the range of <10-90 for golden orfe (*Leuciscus idus*), algae (*Chlorella fusca*), and activated sludge.

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Ecotoxicity
Fish LC50 96 h): *Pimephales promelas* (fathead minnow) >100 mg/l (static test):
Fish NOEC (21 d): *Brachydanio rerio* (zebra fish) =>5000 mg/l (semistatic)
Daphnia magna EC50 (24 h): 5.6 mg/l (immobilisation/ static); (96 h) 1.8 mg/l (immobilisation/ static)
Algae EC50 (96 h) *Scenedesmus subspicatus* 4.8-10 mg/l (biomass reduction): 3.8-5.4 mg/l (growth rate)
Bacterial IC50 microbial culture from nitrifying sewage plant 0.8 mg/l (nitrification inhibition test IC75 (2-4 h)
unadapted nitrifying activated sludge 0.075 mg/l (nitrification inhibition test)
Earthworm LC50 (28 d): *Eisenia fetida* 3550 mg/kg soil dry weight
Among the tested organisms, different stages of the red cotton bug (*Dysdercus similis*) proved to be most sensitive, exhibiting EC50 values of 0.03 and 0.025 mg/litre for egg survival and hatching, respectively.
Different fungi were found to be relatively insensitive to thiourea exposure. Complete growth inhibition was observed for *Penicillium rugulosum* after a 7-day exposure to 2000 mg thiourea/litre and for *Helminthosporium sativum* and *Fusarium oxysporum* after a 15-day exposure to 750 mg/litre and 1000 mg/litre, respectively.
Terrestrial plants proved to be generally more sensitive. Whereas thiourea concentrations below 12 mg/litre increased the growth of excised tomato roots (*Lycopersicum esculentum*) within 4 weeks of exposure in a defined basal medium, 18, 23, and 46 mg/litre reduced growth by about 45%, 60%, and 30%, respectively.
Prevent, by any means available, spillage from entering drains or water courses.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
thiourea	LOW	LOW
boric acid	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
thiourea	LOW (BCF = 2)
boric acid	LOW (BCF = 0)

Mobility in soil

Ingredient	Mobility
thiourea	MEDIUM (KOC = 2.782)
boric acid	LOW (KOC = 35.04)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. For small quantities: ▸ Cautiously dissolve in water ▸ Neutralise with sodium carbonate or if product does not dissolve completely add a small quantity of hydrochloric acid followed by sodium carbonate ▸ Add excess calcium chloride to precipitate the fluoride and/ or carbonate ▸ Remove solids to site approved for hazardous waste ▸ Recycle wherever possible. ▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▸ Treat and neutralise at an approved treatment plant. Treatment should involve: Neutralisation with soda-ash or soda-lime followed by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material). ▸ Decontaminate empty containers with 5% aqueous sodium hydroxide or soda ash, followed by water. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 TRANSPORT INFORMATION

Labels Required

	 Limited quantity: 421-125ML, 421-500ML

Land transport (ADG)

UN number	1775
UN proper shipping name	FLUOROBORIC ACID

Continued...

Transport hazard class(es)	Class: 8 Subrisk: Not Applicable
Packing group	II
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: Not Applicable Limited quantity: 1 L

Air transport (ICAO-IATA / DGR)

UN number	1775
UN proper shipping name	Fluoroboric acid
Transport hazard class(es)	ICAO/IATA Class: 8 ICAO / IATA Subrisk: Not Applicable ERG Code: 8L
Packing group	II
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: Not Applicable Cargo Only Packing Instructions: 855 Cargo Only Maximum Qty / Pack: 30 L Passenger and Cargo Packing Instructions: 851 Passenger and Cargo Maximum Qty / Pack: 1 L Passenger and Cargo Limited Quantity Packing Instructions: Y840 Passenger and Cargo Limited Maximum Qty / Pack: 0.5 L

Sea transport (IMDG-Code / GGVSee)

UN number	1775
UN proper shipping name	FLUOROBORIC ACID
Transport hazard class(es)	IMDG Class: 8 IMDG Subrisk: Not Applicable
Packing group	II
Environmental hazard	Not Applicable
Special precautions for user	EMS Number: F-A , S-B Special provisions: Not Applicable Limited Quantities: 1 L

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

FLUOBORIC ACID(16872-11-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)

THIOUREA(62-56-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix F (Part 3)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

TIN FLUOROBORATE(13814-97-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards
Australia Inventory of Chemical Substances (AICS)

BORIC ACID(10043-35-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

Continued...

National Inventory Status

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (thiourea; boric acid; fluoboric acid; tin fluoroborate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Legend:	*Yes = All ingredients are on the inventory* *No = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 OTHER INFORMATION

Revision Date	03/01/2019
Initial Date	04/01/2018

Other information

Ingredients with multiple cas numbers

Name	CAS No
fluoboric acid	16872-11-0, 102931-96-4, 1056477-09-8, 1178564-64-1, 122141-73-5, 1262801-47-7, 127408-00-8, 1303-67-9, 133097-00-4, 133951-38-9, 13767-36-7, 140148-87-4, 1414852-51-9, 172908-18-8, 183135-74-2, 191665-41-5, 350009-05-1, 36835-64-0, 496925-14-5, 547767-06-6, 65814-43-9, 67116-13-6, 72802-72-3, 74618-51-2, 81586-24-5, 845825-70-9, 900178-90-7, 96958-96-2, 148706-30-3
boric acid	10043-35-3, 11113-50-1, 41685-84-1

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.
The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

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