



421 Liquid Tin

MG Chemicals (Head office)

Chemwatch Hazard Alert Code: 4

Version No: 3.4

Safety Data Sheet (Conforms to Regulations (EC) No 2015/830)

Issue Date: 22/01/2016

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L.REACH.GBR.EN

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

1.1. Product Identifier

Product name	421 Liquid Tin
Synonyms	SDS Code: 421 - liquid; Part Numbers: 421-125ML, 421-500ML
Proper shipping name	FLUOROBORIC ACID
Other means of identification	Not Available

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

Relevant identified uses	Tin plates copper circuits
Uses advised against	Not Applicable

1.3. Details of the supplier of the safety data sheet

Registered company name	MG Chemicals (Head office)	MG Chemicals UK Limited
Address	9347 - 193 Street Surrey V4N 4E7 British Columbia Canada	Heame House, 23 Bilston Street, Sedgely Dudley DY3 1JA United Kingdom
Telephone	+1 800 201 8822	+44 1663 362888
Fax	+1 800 708 9888	Not Available
Website	www.mgchemicals.com	Not Available
Email	Info@mgchemicals.com	sales@mgchemicals.com

1.4. Emergency telephone number

Association / Organisation	Not Available	CHEMTREC
Emergency telephone numbers	Not Available	+(44)-870-8200418
Other emergency telephone numbers	Not Available	+(1) 703-527-3887

SECTION 2 HAZARDS IDENTIFICATION

2.1. Classification of the substance or mixture

Considered a dangerous mixture according to Directive 1999/45/EC, Reg. (EC) No 1272/2008 (if applicable) and their amendments. Classified as Dangerous Goods for transport purposes.

Classification according to regulation (EC) No 1272/2008 [CLP] [1]	Acute Toxicity (Oral) Category 4, Skin Sensitizer Category 1B, Carcinogen Category 2, Reproductive Toxicity Category 1B, Metal Corrosion Category 1, Skin Corrosion/Irritation Category 1C
Legend:	1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

2.2. Label elements

CLP label elements	
SIGNAL WORD	DANGER

Hazard statement(s)

H302	Harmful if swallowed
H317	May cause an allergic skin reaction

Continued...

421 Liquid Tin

H351	Suspected of causing cancer
H360	May damage fertility or the unborn child
H290	May be corrosive to metals
H314	Causes severe skin burns and eye damage

Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P234	Keep only in original container.
P270	Do not eat, drink or smoke when using this product.
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): 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/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P363	Wash contaminated clothing before reuse.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P390	Absorb spillage to prevent material damage.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.
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Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.
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2.3. Other hazards

Inhalation may produce health damage*.

Cumulative effects may result following exposure*.

RECh - Art.57-59: The mixture does not contain Substances of Very High Concern (SVHC) at the SDS print date.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS**3.1.Substances**

See 'Composition on ingredients' in Section 3.2

3.2.Mixtures

1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP]
1.16872-11-0 2.240-898-3 3.009-010-00-X 4.01-2119456258-32-XXXX	9-11	<u>fluoboric acid</u>	Skin Corrosion/Irritation Category 1B; H314 [3]
1.13814-97-6 2.237-487-6 3.Not Available 4.Not Available	9-11	<u>tin</u> <u>fluoroborate</u>	Metal Corrosion Category 1, Skin Corrosion/Irritation Category 1A, Serious Eye Damage Category 1, Reproductive Toxicity Category 1B; H290, H314, H318, H360 [1]
1.62-56-6 2.200-543-5 3.612-082-00-0 4.Not Available	4-6	<u>thiourea</u>	Carcinogen Category 2, Reproductive Toxicity Category 2, Acute Toxicity (Oral) Category 4, Chronic Aquatic Hazard Category 2; H351, H361d, H302, H411 [3]

Legend: 1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex

Continued...

421 Liquid Tin

VI 4. Classification drawn from C&L

SECTION 4 FIRST AID MEASURES

4.1. Description of first aid measures

General	<ul style="list-style-type: none"> ▶ 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. <p>For massive exposures:</p> <ul style="list-style-type: none"> ▶ If dusts, vapours, aerosols, fumes or combustion products are inhaled, remove from contaminated area. ▶ Lay patient down. ▶ Keep warm and rested. ▶ Prosthesis 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. ▶ Transport to hospital, or doctor, urgently. <p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. <p>If there is evidence of severe skin irritation or skin burns:</p> <ul style="list-style-type: none"> ▶ 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.
Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If there is evidence of severe skin irritation or skin burns:</p> <ul style="list-style-type: none"> ▶ 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	<p>For massive exposures:</p> <ul style="list-style-type: none"> ▶ If dusts, vapours, aerosols, fumes or combustion products are inhaled, remove from contaminated area. ▶ Lay patient down. ▶ Keep warm and rested. ▶ Prosthesis 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. ▶ Transport to hospital, or doctor, urgently.
Ingestion	<ul style="list-style-type: none"> ▶ 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.

4.2 Most important symptoms and effects, both acute and delayed

See Section 11

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

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.

Continued...

421 Liquid Tin

- ▶ 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

5.1. Extinguishing media

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

5.2. Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.
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5.3. Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear full body protective clothing with breathing apparatus. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ 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	<ul style="list-style-type: none"> ▶ 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

SECTION 6 ACCIDENTAL RELEASE MEASURES

6.1. Personal precautions, protective equipment and emergency procedures

See section 8

6.2. Environmental precautions

See section 12

6.3. Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear full body protective clothing with breathing apparatus. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Consider evacuation (or protect in place). ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Neutralise/decontaminate residue (see Section 13 for specific agent). ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▶ If contamination of drains or waterways occurs, advise emergency services.

421 Liquid Tin

6.4. Reference to other sections

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

SECTION 7 HANDLING AND STORAGE

7.1. Precautions for safe handling

Safe handling	<p>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.</p> <ul style="list-style-type: none"> ▶ 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.
Fire and explosion protection	See section 5
Other information	<ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

7.2. Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ DO NOT use aluminium or galvanised containers ▶ Check regularly for spills and leaks ▶ 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. <p>For low viscosity materials</p> <ul style="list-style-type: none"> ▶ 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. <p>For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.):</p> <ul style="list-style-type: none"> ▶ Removable head packaging; ▶ Cans with friction closures and ▶ low pressure tubes and cartridges <p>may be used.</p> <p>-</p> <p>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.</p>
Storage incompatibility	<p>Salts of inorganic fluoride:</p> <ul style="list-style-type: none"> ▶ 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 <p>Fluoboric acid:</p> <ul style="list-style-type: none"> ▶ is a strong inorganic acid; reacts violently with strong bases ▶ reacts slowly with water forming hydroxyfluoroborate 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. ▶ Reacts with mild steel, galvanised steel / zinc producing hydrogen gas which may form an explosive mixture with air. <p>Thiourea</p> <ul style="list-style-type: none"> ▶ is basic in aqueous solutions ▶ reacts violently with acrolein, strong acids

421 Liquid Tin

- ▶ 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
- ▶ Segregate from alkalis, oxidising agents and chemicals readily decomposed by acids, i.e. cyanides, sulfides, carbonates.

7.3. Specific end use(s)

See section 1.2

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION**8.1. Control parameters****DERIVED NO EFFECT LEVEL (DNEL)**

Not Available

PREDICTED NO EFFECT LEVEL (PNEC)

Not Available

OCCUPATIONAL EXPOSURE LIMITS (OEL)**INGREDIENT DATA**

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	tin fluoroborate	Tin compounds, inorganic except SnH4, (as Sn)	2 mg/m3	4 mg/m3	Not Available	Not Available
EU Directive 91/322/EEC Indicative Occupational Exposure Limit Values (IOELVs)	tin fluoroborate	Tin (inorganic compounds as Sn) (6)	2 mg/m3	Not Available	Not Available	Not Available
EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	tin fluoroborate	Tin and inorganic tin compounds	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
tin fluoroborate	Tin fluoroborate	15 mg/m3	170 mg/m3	990 mg/m3
thiourea	Thiourea	0.84 mg/m3	9.3 mg/m3	80 mg/m3

Ingredient	Original IDLH	Revised IDLH
fluoboric acid	Not Available	Not Available
tin fluoroborate	400 mg/m3	100 mg/m3
thiourea	Not Available	Not Available

MATERIAL DATA

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.

8.2. Exposure controls**8.2.1. 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.)

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421 Liquid Tin

	<p>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</p> <p>2.5-10 m/s (500-2000 f/min.)</p> <p>Within each range the appropriate value depends on:</p> <table border="1"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 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.</p>	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
Lower end of the range	Upper end of the range										
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents										
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3: Intermittent, low production.	3: High production, heavy use										
4: Large hood or large air mass in motion	4: Small hood-local control only										
8.2.2. Personal protection											
Eye and face protection	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ Elbow length PVC gloves ▶ When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots. <p>NOTE:</p> <ul style="list-style-type: none"> ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. 										
Body protection	See Other protection below										
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ PVC Apron. ▶ PVC protective suit may be required if exposure severe. ▶ Eyewash unit. ▶ Ensure there is ready access to a safety shower. 										
Thermal hazards	Not Available										

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:

421 Liquid Tin

Material	CPI
PE/EVAL/PE	A

* 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.

Respiratory protection

Not Available

Not Applicable

8.2.3. Environmental exposure controls

See section 12

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

9.1. Information on basic physical and chemical properties

Appearance	clear
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Continued...

421 Liquid Tin

Physical state	Liquid	Relative density (Water = 1)	1.12
Odour	Not Available	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
Vapour pressure (kPa)	2.40	Gas group	Not Available
Solubility in water (g/L)	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

9.2. Other information

Not Available

SECTION 10 STABILITY AND REACTIVITY

10.1.Reactivity	See section 7.2
10.2.Chemical stability	<ul style="list-style-type: none"> ▶ Contact with alkaline material liberates heat ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.
10.3. Possibility of hazardous reactions	See section 7.2
10.4. Conditions to avoid	See section 7.2
10.5. Incompatible materials	See section 7.2
10.6. Hazardous decomposition products	See section 5.3

SECTION 11 TOXICOLOGICAL INFORMATION

11.1. Information on toxicological effects

Inhaled	<p>Acidic corrosives produce respiratory tract irritation with coughing, choking and mucous membrane damage. Symptoms of exposure may include dizziness, headache, nausea and weakness. In more severe exposures, pulmonary oedema may be evident either immediately or after a latent period of 5-72 hours. Symptoms of pulmonary oedema include a tightness in the chest, dyspnoea, frothy sputum and cyanosis. Examination may reveal hypotension, a weak and rapid pulse and moist rates. Death, due to anoxia, may occur several hours after onset of the pulmonary oedema.</p> <p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Acute effects of fluoride inhalation include irritation of nose and throat, coughing and chest discomfort.</p> <p>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</p> <p>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.</p> <p>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.</p> <p>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).</p> <p>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.</p>
Ingestion	<p>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.</p> <p>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.</p> <p>Fluoride is a general protoplasmic poison which appears to produce at least four major functional derangements;</p>

Continued...

421 Liquid Tin

	<p>(1) enzyme inhibition, (2) hypocalcaemia, (3) cardiovascular collapse and (4) specific organ damage.</p> <p>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.</p> <p>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.</p>					
Skin Contact	<p>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.</p> <p>Skin contact is not thought to produce harmful health effects (as classified under EC Directives using animal models). Systemic harm, however, has been identified following exposure of animals by at least one other route and the material may still produce health damage following entry through wounds, lesions or abrasions. Good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>					
Eye	<p>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.</p>					
Chronic	<p>Repeated or prolonged exposure to acids may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis.</p> <p>The impact of inhaled acidic agents on the respiratory tract depends upon a number of interrelated factors. These include physicochemical characteristics, e.g., gas versus aerosol; particle size (small particles can penetrate deeper into the lung); water solubility (more soluble agents are more likely to be removed in the nose and mouth). Given the general lack of information on the particle size of aerosols involved in occupational exposures to acids, it is difficult to identify their principal deposition site within the respiratory tract. Acid mists containing particles with a diameter of up to a few micrometers will be deposited in both the upper and lower airways. They are irritating to mucous epithelia, they cause dental erosion, and they produce acute effects in the lungs (symptoms and changes in pulmonary function). Asthmatics appear to be at particular risk for pulmonary effects.</p> <p>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.</p> <p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: - clear evidence in animal studies 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 is not a secondary non-specific consequence of other toxic effects.</p> <p>There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of: - clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>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.</p> <p>Chronic poisoning by borates may be characterised gastrointestinal disturbances and skin rash. Chronic absorption of small amounts of borax causes mild gastroenteritis and dermatitis.</p> <p>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).</p> <p>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.</p> <p>Thiourea is a sensitizer in persons who exhibit photosensitivity.</p> <p>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.</p> <p>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.</p> <p>Fluoroborates stand apart from other inorganic fluorides in that they are not stored in the bone and excretion (mainly in the urine) exceeds hydrolysis leaving little opportunity for fluoride ion storage. However fluoroborates do accumulate in the thyroid gland preventing uptake of iodine. Animal testing (rat) shows evidence that chronic boron trifluoride exposure may cause elevated levels of bone fluoride and dental fluorosis.</p>					
421 Liquid Tin	<table border="1"> <thead> <tr> <th>TOXICITY</th> <th>IRRITATION</th> </tr> </thead> <tbody> <tr> <td>Not Available</td> <td>Not Available</td> </tr> </tbody> </table>	TOXICITY	IRRITATION	Not Available	Not Available	
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TOXICITY	IRRITATION					

421 Liquid Tin

Oral (rat) LD50: 125 mg/kg^[2]

Eye (rabbit): 14%

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.

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. 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.

FLUOBORIC ACID

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. 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.

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*.

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.

* Megachem SDS

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.

421 Liquid Tin

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 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.

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 available but does not fill the criteria for classification

Continued...

421 Liquid Tin

✔ – Data required to make classification available

⊘ – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

12.1. Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
fluoboric acid	LC50	96	Fish	2600mg/L	2
fluoboric acid	EC50	48	Crustacea	>100mg/L	2
fluoboric acid	EC50	72	Algae or other aquatic plants	>100mg/L	2
fluoboric acid	NOEC	72	Algae or other aquatic plants	100mg/L	2
tin fluoroborate	LC50	96	Fish	78mg/L	2
tin fluoroborate	EC50	672	Fish	>1.56mg/L	2
tin fluoroborate	NOEC	672	Fish	0.78mg/L	2
tin fluoroborate	EC50	48	Crustacea	87mg/L	2
tin fluoroborate	EC50	72	Algae or other aquatic plants	>100mg/L	2
thiourea	EC50	48	Crustacea	=35mg/L	1
thiourea	BCF	24	Algae or other aquatic plants	0.05mg/L	4
thiourea	EC10	72	Algae or other aquatic plants	0.3-0.7mg/L	1
thiourea	LC50	96	Fish	>100mg/L	1
thiourea	NOEC	504	Crustacea	>=0.1- <=0.25mg/L	2
thiourea	EC50	96	Algae or other aquatic plants	>=3.8- <=5.4mg/L	2
Legend:	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 - 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				

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.

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

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

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

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

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

Populations living in areas with high fluoride levels in groundwater may be exposed to higher levels of fluorides in their drinking water or in beverages prepared with the water. Among these populations, outdoor laborers, people living in hot climates, and people with polydipsia will generally have the greatest daily intake of fluorides because they consume greater amounts of water. Foods characteristically high in fluoride content are certain types of fish and seafood (1.9?28.5 mg/kg), especially those types in which the bones are consumed, bone products such as bone meal and gelatin, and tea, which contains approximately 0.52 mg fluoride/cup

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

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.

Tin may exist in either divalent (Sn²⁺) or tetravalent (Sn⁴⁺) cationic (positively charged) ions under environmental conditions. Tin(II) dominates in reduced (oxygen-poor) water, and will readily precipitate as a sulfide (SnS) or as a hydroxide (Sn(OH)₂) in alkaline water. Tin(IV) readily hydrolyses, and can precipitate as a hydroxide. The solubility product of Sn(OH)₄ has been measured at approximately 10 exp(-56) g/L at 25 °C. In general, tin(IV) would be expected to be the only stable ionic species in the weathering cycle.

Tin in water may partition to soils and sediments. Cations such as Sn²⁺ and Sn⁴⁺ will generally be adsorbed by soils to some extent, which reduces their mobility. Tin is generally regarded as being relatively immobile in the environment. However, tin may be transported in water if it partitions to suspended sediments, but the significance of this mechanism has not been studied in detail. Transfer coefficients for tin in a soil-plant system were reported to be 0.01-0.1.

A bioconcentration factor (BCF) relates the concentration of a chemical in plants and animals to the concentration of the chemical in the medium in which they live. It was estimated that the BCFs of inorganic tin were 100, 1,000, and 3,000 for marine and freshwater plants, invertebrates, and fish, respectively. Marine algae can bioconcentrate tin(IV) ion by a factor of 1,900.

Inorganic tin cannot be degraded in the environment, but may undergo oxidation-reduction, ligand exchange, and precipitation reactions. It has been established that inorganic tin can be transformed into organometallic forms by microbial methylation. Inorganic tin may also be converted to stannane (H₄Sn) in extremely anaerobic (oxygen-poor) conditions by macroalgae.

For boron and borates:

Continued...

421 Liquid Tin

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 is 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)₃. 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 (K_{oc}) 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 $3.0 \times 10^{+9}$ /mol per second (pH 6.4) and $4.7 \times 10^{+9}$ /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 K_{oc} 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.

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 (*Lycopersicon 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.

421 Liquid Tin

12.2. Persistence and degradability

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

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
thiourea	LOW (BCF = 2)

12.4. Mobility in soil

Ingredient	Mobility
thiourea	MEDIUM (KOC = 2.782)

12.5. Results of PBT and vPvB assessment

	P	B	T
Relevant available data	Not Available	Not Available	Not Available
PBT Criteria fulfilled?	Not Available	Not Available	Not Available

12.6. Other adverse effects

No data available

SECTION 13 DISPOSAL CONSIDERATIONS

13.1. Waste treatment methods

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

SECTION 14 TRANSPORT INFORMATION

Labels Required

	
Marine Pollutant	NO
HAZCHEM	2X

Land transport (ADR)

14.1.UN number	1775
14.2.Packing group	II
14.3.UN proper shipping name	FLUOROBORIC ACID
14.4.Environmental hazard	Not Applicable

421 Liquid Tin

14.5. Transport hazard class(es)	Class	8
	Subrisk	Not Applicable
14.6. Special precautions for user	Hazard identification (Kemler)	80
	Classification code	C1
	Hazard Label	8
	Special provisions	Not Applicable
	Limited quantity	1 L

Air transport (ICAO-IATA / DGR)

14.1. UN number	1775	
14.2. Packing group	II	
14.3. UN proper shipping name	Fluoroboric acid	
14.4. Environmental hazard	Not Applicable	
14.5. Transport hazard class(es)	ICAO/IATA Class	8
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	8L
14.6. 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)

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

Inland waterways transport (ADN)

14.1. UN number	1775	
14.2. Packing group	II	
14.3. UN proper shipping name	FLUOROBORIC ACID	
14.4. Environmental hazard	Not Applicable	
14.5. Transport hazard class(es)	8	Not Applicable
14.6. Special precautions for user	Classification code	C1
	Special provisions	Not Applicable
	Limited quantity	1 L
	Equipment required	PP, EP
	Fire cones number	0

421 Liquid Tin

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

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

European Customs Inventory of Chemical Substances ECICS (English)	European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)	European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

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

EU Consolidated List of Indicative Occupational Exposure Limit Values (IOELVs)	European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)
EU Directive 91/322/EEC Indicative Occupational Exposure Limit Values (IOELVs)	UK Workplace Exposure Limits (WELs)

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

European Customs Inventory of Chemical Substances ECICS (English)	European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances (updated by ATP: 31) - Carcinogenic Substances
European Trade Union Confederation (ETUC) Priority List for REACH Authorisation	European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances (updated by ATP: 31) - Reprotoxic Substances
European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)	European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - updated by ATP: 31	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : 67/548/EEC, 1999/45/EC, 98/24/EC, 92/85/EC, 94/33/EC, 91/689/EEC, 1999/13/EC, Commission Regulation (EU) 2015/830, Regulation (EC) No 1272/2008 and their amendments as well as the following British legislation: - The Control of Substances Hazardous to Health Regulations (COSHH) 2002 - COSHH Essentials - The Management of Health and Safety at Work Regulations 1999

15.2. Chemical safety assessment

For further information please look at the Chemical Safety Assessment and Exposure Scenarios prepared by your Supply Chain if available.

ECHA SUMMARY

Ingredient	CAS number	Index No	ECHA Dossier
fluoboric acid	16872-11-0	009-010-00-X	01-2119456258-32-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Skin Corr. 1B	GHS05, Dgr	H314
2	Met. Corr. 1, Skin Corr. 1A, Skin Corr. 1B, Eye Dam. 1	GHS05, Dgr	H314

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
tin fluoroborate	13814-97-6	Not Available	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Skin Irrit. 2, Eye Irrit. 2	GHS07, Wng	H315, H319
2	Met. Corr. 1, Acute Tox. 4, Skin Sens. 1, Eye Irrit. 2, Skin Irrit. 2	Wng, GHS05, Dgr	H290, H302, H317, H319, H314

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
thiourea	62-56-6	612-082-00-0	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Acute Tox. 4, Carc. 2, Repr. 2, Aquatic Chronic 2	GHS07, GHS09, GHS08, Wng	H302, H351, H361
2	Acute Tox. 4, Carc. 2, Repr. 2, Aquatic Chronic 2, Skin Irrit. 2, Skin Sens. 1, Acute Tox. 3, Not Classified, STOT RE 2	GHS09, GHS08, Wng, GHS06, Dgr	H351, H315, H317, H301, H331, H373, H360

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (thiourea; fluoboric acid; tin fluoroborate)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	Y

Continued...

421 Liquid Tin

Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
Legend:	Y = All ingredients are on the inventory N = 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**Full text Risk and Hazard codes**

H301	Toxic if swallowed
H315	Causes skin irritation
H318	Causes serious eye damage
H319	Causes serious eye irritation
H331	Toxic if inhaled
H361	Suspected of damaging fertility or the unborn child
H361d	Suspected of damaging the unborn child.
H373	May cause damage to organs.
H411	Toxic to aquatic life with long lasting effects

Other information

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

A list of reference resources used to assist the committee may be found at:

www.chemwatch.net

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

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

EN 166 Personal eye-protection

EN 340 Protective clothing

EN 374 Protective gloves against chemicals and micro-organisms

EN 13832 Footwear protecting against chemicals

EN 133 Respiratory protective devices

Definitions and abbreviations

PC – TWA: Permissible Concentration-Time Weighted Average

PC – STEL: Permissible Concentration-Short Term Exposure Limit

IARC: International Agency for Research on Cancer

ACGIH: American Conference of Governmental Industrial Hygienists

STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit,

IDLH: Immediately Dangerous to Life or Health Concentrations

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