421A Liquid Tin

Mektronics

Version No: **A-2.00**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **02/11/2021**
Revision Date:**13/04/2023**
L.GHS.AUS.EN

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

Product Identifier

Product name	421A Liquid Tin
Synonyms	SDS Code: 421A-liquid; 421A-125ML, 421A-500ML; UFI: UDA0-4056-900Y-FEY7
Other means of identification	03032020421a | UFI:UDA0-4056-900Y-FEY7

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

Relevant identified uses	Electroless tin plating solution

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**Mektronics**	**MG Chemicals (Head office)**
Address	Unit 3 8 Bonz Place, Seven Hills NSW 2147 Australia	1210 Corporate Drive Ontario L7L 5R6 Canada
Telephone	1300 788 701	+(1) 800-340-0772
Fax	1300 722 004	+(1) 800-340-0773
Website	www.mektronics.com.au	www.mgchemicals.com
Email	sales@mektronics.com.au	Info@mgchemicals.com

Emergency telephone number

Association / Organisation	**Verisk 3E (Access Code: 335388)**
Emergency telephone numbers	+61 1 800 686 951
Other emergency telephone numbers	+61 280363166

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Label elements

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

Hazard statement(s)

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

Precautionary statement(s) Prevention

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

Page 1 continued...

P264	Wash all exposed external body areas thoroughly after handling.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P273	Avoid release to the environment.
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 [or shower].
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
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.
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.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
62-56-6	10	thiourea
53408-94-9	5	stannous methanesulfonate
75-75-2	4	methanesulfonic acid
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available*	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs: ▸ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▸ Quickly remove all contaminated clothing, including footwear. ▸ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▸ Transport to hospital, or doctor.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay. ▸ Inhalation of vapours or aerosols (mists, fumes) may cause lung oedema. ▸ Corrosive substances may cause lung damage (e.g. lung oedema, fluid in the lungs). ▸ As this reaction may be delayed up to 24 hours after exposure, affected individuals need complete rest (preferably in semi-recumbent posture) and must be kept under medical observation even if no symptoms are (yet) manifested. ▸ Before any such manifestation, the administration of a spray containing a dexamethasone derivative or beclomethasone derivative may be considered. **This must definitely be left to a doctor or person authorised by him/her.** (ICSC13719)
Ingestion	▸ For advice, contact a Poisons Information Centre or a doctor at once. ▸ Urgent hospital treatment is likely to be needed. ▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.

Continued...

	▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Transport to hospital or doctor without delay.

Indication of any immediate medical attention and special treatment needed

For acute or short term repeated exposures to strong acids:
- Airway problems may arise from laryngeal edema and inhalation exposure. Treat with 100% oxygen initially.
- Respiratory distress may require cricothyroidotomy if endotracheal intubation is contraindicated by excessive swelling
- Intravenous lines should be established immediately in all cases where there is evidence of circulatory compromise.
- Strong acids produce a coagulation necrosis characterised by formation of a coagulum (eschar) as a result of the dessicating action of the acid on proteins in specific tissues.

INGESTION:
- Immediate dilution (milk or water) within 30 minutes post ingestion is recommended.
- **DO NOT** attempt to neutralise the acid since exothermic reaction may extend the corrosive injury.
- Be careful to avoid further vomit since re-exposure of the mucosa to the acid is harmful. Limit fluids to one or two glasses in an adult.
- Charcoal has no place in acid management.
- Some authors suggest the use of lavage within 1 hour of ingestion.

SKIN:
- Skin lesions require copious saline irrigation. Treat chemical burns as thermal burns with non-adherent gauze and wrapping.
- Deep second-degree burns may benefit from topical silver sulfadiazine.

EYE:
- Eye injuries require retraction of the eyelids to ensure thorough irrigation of the conjuctival cul-de-sacs. Irrigation should last at least 20-30 minutes. **DO NOT** use neutralising agents or any other additives. Several litres of saline are required.
- Cycloplegic drops, (1% cyclopentolate for short-term use or 5% homatropine for longer term use) antibiotic drops, vasoconstrictive agents or artificial tears may be indicated dependent on the severity of the injury.
- Steroid eye drops should only be administered with the approval of a consulting ophthalmologist).

[Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	▸ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water 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	▸ Combustible. ▸ Slight fire hazard when exposed to heat or flame. ▸ 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 acrid smoke and corrosive fumes. Combustion products include: carbon monoxide (CO) carbon dioxide (CO_2) sulfur oxides (SOx) hydrogen sulfide (H_2S) metal oxides other pyrolysis products typical of burning organic material.

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	▸ Drains for storage or use areas should have retention basins for pH adjustments and dilution of spills before discharge or disposal of material. ▸ Check regularly for spills and leaks.

Continued...

- 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

Chemical Class:acidic compounds, organic
For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS

LAND SPILL - SMALL

wood fiber - pillow	1	throw	pitchfork	R, P, DGC, RT
cross-linked polymer - particulate	1	shovel	shovel	R,W,SS
cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT
sorbent clay - particulate	2	shovel	shovel	R, I, P
foamed glass - pillow	2	throw	pitchfork	R, P, DGC, RT
wood fiber - particulate	3	shovel	shovel	R, W, P, DGC

LAND SPILL - MEDIUM

cross-linked polymer -particulate	1	blower	skiploader	R, W, SS
polypropylene - particulate	2	blower	skiploader	W, SS, DGC
sorbent clay - particulate	2	blower	skiploader	R, I, P
cross-linked polymer - pillow	3	throw	skiploader	R, DGC, RT
polypropylene - mat	3	throw	skiploader	W, SS, DGC
expanded mineral - particulate	3	blower	skiploader	R, I, W, P, DGC

Legend
DGC: Not effective where ground cover is dense
R; Not reusable
I: Not incinerable
P: Effectiveness reduced when rainy
RT:Not effective where terrain is rugged
SS: Not for use within environmentally sensitive sites
W: Effectiveness reduced when windy
Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;
R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988
Chemical Class: sulfates and sulfites
For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS

LAND SPILL - SMALL

cross-linked polymer - particulate	1	shovel	shovel	R, W, SS
wood-fiber - pillow	1	throw	pitchfork	R, P, DGC, RT
treated wood fiber - pillow	1	throw	pitchfork	DGC, RT
cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT
sorbent clay - particulate	2	shovel	shovel	R, I, P
foamed glass - pillow	2	throw	pitchfork	R, P, DGC, RT

LAND SPILL - MEDIUM

cross-linked polymer - particulate	1	blower	skiploader	R,W, SS
sorbent clay - particulate	2	blower	skiploader	R, I, P
polypropylene - particulate	2	blower	skiploader	R, SS, DGC
expanded mineral - particulate	3	blower	skiploader	R, I, W, P, DGC
wood fiber - particulate	3	blower	skiploader	R, W, P, DGC
polypropylene - mat	3	throw	skiploader	DGC, RT

Legend
DGC: Not effective where ground cover is dense
R; Not reusable
I: Not incinerable
P: Effectiveness reduced when rainy
RT:Not effective where terrain is rugged
SS: Not for use within environmentally sensitive sites
W: Effectiveness reduced when windy
Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;
R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988

Continued...

	‣ 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.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	‣ Avoid all personal contact, including inhalation. ‣ Wear protective clothing when risk of exposure occurs. ‣ Use in a well-ventilated area. ‣ **WARNING:** To avoid violent reaction, **ALWAYS** add material to water and **NEVER** water to material. ‣ Avoid smoking, naked lights or ignition sources. ‣ Avoid contact with incompatible materials. ‣ When handling, **DO NOT** eat, drink or smoke. ‣ Keep containers securely sealed when not in use. ‣ Avoid physical damage to containers. ‣ Always wash hands with soap and water after handling. ‣ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ‣ Use good occupational work practice. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS. ‣ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	‣ Store in original containers. ‣ Keep containers securely sealed. ‣ Store in a cool, dry, well-ventilated area. ‣ Store away from incompatible materials and foodstuff containers. ‣ Protect containers against physical damage and check regularly for leaks. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	‣ **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. For low viscosity materials ‣ Drums and jerricans must be of the non-removable head type. ‣ Where a can is to be used as an inner package, the can must have a screwed enclosure. For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.): ‣ Removable head packaging; ‣ Cans with friction closures and ‣ low pressure tubes and cartridges may be used. - Where combination packages are used, and the inner packages are of glass, porcelain or stoneware, there must be sufficient inert cushioning material in contact with inner and outer packages unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	‣ Contact with acids produces toxic fumes ‣ Reacts with mild steel, galvanised steel / zinc producing hydrogen gas which may form an explosive mixture with air. Thiourea ‣ is basic in aqueous solutions ‣ reacts violently with acrolein, strong acids ‣ is incompatible with acrylaldehyde, hydrogen peroxide, metal salts ‣ aqueous solutions are incompatible with organic anhydrides, acrylates, alcohols, aldehydes, alkylene oxides, substituted allyls, cresols, caprolactam solutions, epichlorohydrin, ethylene dichloride, glycols, hydrogen peroxide, isocyanates, ketones, maleic anhydride, nitrates, nitromethane, phenols, vinyl acetate ‣ Avoid strong bases. ‣ Segregate from alkalies, oxidising agents and chemicals readily decomposed by acids, i.e. cyanides, sulfides, carbonates.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes

Continued...

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	stannous methanesulfonate	Tin, organic compounds (as Sn)	0.1 mg/m3	0.2 mg/m3	Not Available	(g) Some compounds in these groups are classified as carcinogenic or as sensitisers. Check individual classification details on the safety data sheet for information on classification.

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
thiourea	0.38 mg/m3	4.1 mg/m3	25 mg/m3
methanesulfonic acid	0.99 mg/m3	11 mg/m3	65 mg/m3

Ingredient	Original IDLH	Revised IDLH
thiourea	Not Available	Not Available
stannous methanesulfonate	25 mg/m3	Not Available
methanesulfonic acid	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
thiourea	E	≤ 0.01 mg/m³
methanesulfonic acid	E	≤ 0.1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

Sensory irritants are chemicals that produce temporary and undesirable side-effects on the eyes, nose or throat. Historically occupational exposure standards for these irritants have been based on observation of workers' responses to various airborne concentrations. Present day expectations require that nearly every individual should be protected against even minor sensory irritation and exposure standards are established using uncertainty factors or safety factors of 5 to 10 or more. On occasion animal no-observable-effect-levels (NOEL) are used to determine these limits where human results are unavailable. An additional approach, typically used by the TLV committee (USA) in determining respiratory standards for this group of chemicals, has been to assign ceiling values (TLV C) to rapidly acting irritants and to assign short-term exposure limits (TLV STELs) when the weight of evidence from irritation, bioaccumulation and other endpoints combine to warrant such a limit. In contrast the MAK Commission (Germany) uses a five-category system based on intensive odour, local irritation, and elimination half-life. However this system is being replaced to be consistent with the European Union (EU) Scientific Committee for Occupational Exposure Limits (SCOEL); this is more closely allied to that of the USA.
OSHA (USA) concluded that exposure to sensory irritants can:

- cause inflammation
- cause increased susceptibility to other irritants and infectious agents
- lead to permanent injury or dysfunction
- permit greater absorption of hazardous substances and
- acclimate the worker to the irritant warning properties of these substances thus increasing the risk of overexposure.

Exposure controls

Appropriate engineering controls

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

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

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

Within each range the appropriate value depends on:

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

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted,

Continued...

	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.
Individual protection measures, such as personal protective equipment	
Eye and face protection	‣ Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure. ‣ Chemical goggles.whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted. ‣ Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection. ‣ Alternatively a gas mask may replace splash goggles and face shields. ‣ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	‣ Elbow length PVC gloves ‣ When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots. **NOTE:** ‣ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ‣ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.
Body protection	See Other protection below
Other protection	‣ Overalls. ‣ PVC Apron. ‣ PVC protective suit may be required if exposure severe. ‣ Eyewash unit. ‣ Ensure there is ready access to a safety shower.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
'Forsberg Clothing Performance Index'.
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
421A Liquid Tin

Material	CPI
NEOPRENE	C
PE/EVAL/PE	C
PVC	C

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

Respiratory protection

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

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

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

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

76ab-p()

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Yellow

Physical state	Liquid	**Relative density (Water = 1)**	1.25
Odour	Slight sulfur	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	<1	**Decomposition temperature (°C)**	Not Available

Continued...

Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	<20.5
Initial boiling point and boiling range (°C)	Not Available	**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)	Not Available	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. 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.
Ingestion	Ingestion of acidic corrosives may produce circumoral burns with a distinct discolouration of the mucous membranes of the mouth, throat and oesophagus. Immediate pain and difficulties in swallowing and speaking may also be evident. Oedema of the epiglottis may produce respiratory distress and possibly, asphyxia. Nausea, vomiting, diarrhoea and a pronounced thirst may occur. More severe exposures may produce a vomitus containing fresh or dark blood and large shreds of mucosa. Shock, with marked hypotension, weak and rapid pulse, shallow respiration and clammy skin may be symptomatic of the exposure. Circulatory collapse may, if left untreated, result in renal failure. Severe cases may show gastric and oesophageal perforation with peritonitis, fever and abdominal rigidity. Stricture of the oesophageal, gastric and pyloric sphincter may occur as within several weeks or may be delayed for years. Death may be rapid and often results from asphyxia, circulatory collapse or aspiration of even minute amounts. Delayed deaths may be due to peritonitis, severe nephritis or pneumonia. Coma and convulsions may be terminal. The material is not thought to produce adverse health effects following ingestion (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum. Skin sensitivity to thiourea derivatives has been demonstrated in several studies. Allergic contact dermatitis and photocontact dermatitis have been described. A Russian study published in 1970 reported that workers handling thiourea products showed ready penetration through the skin which lead to clinical evidence of destructive changes in the thyroid gland. Case reports of contact and photocontact sensitivity to dimethylthiourea have been described. Symptoms include a recurrent itchy dermatitis on the eyelids, nostrils and mouth, which spread to other locations such as the hands and neck. One worker has been reported to have become light-sensitive, even to exposure to neon light, with episodes occurring soon after he returned to work and continuing work for several weeks. Three female patients showed allergic sensitisation to dimethylthiourea after patch testing. Cross-sensitivity was also demonstrated to diethylthiourea Thiourea, diethylthiourea and tetramethylthiourea have been used clinically to produce antithyroid effects in humans. Commonly observed side-effects of treatments include headache, anxiety, fever, skin rash and gastrointestinal disturbance.
Skin Contact	Skin contact with the material may be harmful; systemic effects may result following absorption. 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.

Continued...

	Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. Skin sensitivity to thiourea derivatives has been demonstrated in several studies. Allergic contact dermatitis and photocontact dermatitis have been described. A Russian study published in 1970 reported that workers handling thiourea products showed ready penetration through the skin which lead to clinical evidence of destructive changes in the thyroid gland. Case reports of contact and photocontact sensitivity to dimethylthiourea have been described. Symptoms include a recurrent itchy dermatitis on the eyelids, nostrils and mouth, which spread to other locations such as the hands and neck. One worker has been reported to have become light-sensitive, even to exposure to neon light, with episodes occurring soon after he returned to work and continuing work for several weeks. Three female patients showed allergic sensitisation to dimethylthiourea after patch testing. Cross-sensitivity was also demonstrated to diethylthiourea
Eye	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. When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. Irritation of the eyes may produce a heavy secretion of tears (lachrymation).
Chronic	Repeated or prolonged exposure to acids may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis. The impact of inhaled acidic agents on the respiratory tract depends upon a number of interrelated factors. These include physicochemical characteristics, e.g., gas versus aerosol; particle size (small particles can penetrate deeper into the lung); water solubility (more soluble agents are more likely to be removed in the nose and mouth). Given the general lack of information on the particle size of aerosols involved in occupational exposures to acids, it is difficult to identify their principal deposition site within the respiratory tract. Acid mists containing particles with a diameter of up to a few micrometers will be deposited in both the upper and lower airways. They are irritating to mucous epithelia, they cause dental erosion, and they produce acute effects in the lungs (symptoms and changes in pulmonary function). AsthmatIcs appear to be at particular risk for pulmonary effects. On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects. Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Thiourea is a sensitiser in persons who exhibit photosensitivity. Chronic exposure may result in damage to the blood, liver and thyroid. Thiourea inhibits utilisation of Iodine and has a haemolytic effect (impedes blood clotting). Thiourea has produced goiter and bone marrow depression (anaemia, leukopenia, thrombocytopenia and agranulocytosis) in experimental animals. When administered in the drinking water, thiourea induced thyroid adenomas and carcinomas in rats of both sexes and squamous cell carcinomas of the Zymbal gland in male rats. When administered in the diet, thiourea induced hepatocellular adenomas in rats. The mechanism by which thioureas exert the antithyroid effect involves the inhibition of iodine uptake and activation by the thyroid. At low doses, a physiological and biological compensation mechanism maintains normal levels of circulating thyroid hormone. Prolonged exposure to high doses of thyroid inhibitors causes severe hypertrophy and hyperplasia resulting in reduced levels of circulating thyroid hormone. Positive mutagenic effects have been elicited by the use of several thiourea derivatives in various assays. Teratogenic responses have been recorded with alkylated thioureas and ethylene thiourea in various species. Alkyl-substituted sulfonates have mutagenic potential due to their alkylating properties. Alkylating agents may damage the stem cell which acts as the precursor to components of the blood. Loss of the stem cell may result in pancytopenia (a reduction in the number of red and white blood cells and platelets) with a latency period corresponding to the lifetime of the individual blood cells. Granulocytopenia (a reduction in granular leukocytes) develops within days and thrombocytopenia (a disorder involving platelets), within 1-2 weeks, whilst loss of erythrocytes (red blood cells) need months to become clinically manifest. Aplastic anaemia develops due to complete destruction of the stem cells.

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

thiourea	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2800 mg/kg[2]	Eye (rabbit): 14%

Continued...

	Inhalation(Rat) LC50: >0.195 mg/l4h[2]
	Oral (Rat) LD50: 125 mg/kg[2]

stannous methanesulfonate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Not Available
	Oral (Rat) LD50: 1621 mg/kg[1]	

methanesulfonic acid	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >1000 mg/kg[1]	Not Available
	Oral (Rat) LD50: 200 mg/kg[2]	

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

421A Liquid Tin

Goitrogenic:.
Goitrogens are substances that suppress the function of the thyroid gland by interfering with iodine uptake, which can, as a result, cause an enlargement of the thyroid, i.e., a goitre
Goitrogens include:

- Vitexin, a flavanoid, which inhibits thyroid peroxidase thus contributing to goiter.
- Ions such as thiocyanate and perchlorate which decrease iodide uptake by competitive inhibition; as a consequence of reduced thyroxine and triiodothyronine secretion by the gland, at low doses, this causes an increased release of thyrotropin (by reduced negative feedback), which then stimulates the gland.
- Lithium which inhibits thyroid hormone release.
- Certain foods, such as soy and millet (containing vitexins) and vegetables in the genus Brassica (e.g. broccoli, brussels sprouts, cabbage, horseradish).
- Caffeine (in coffee, tea, cola, chocolate) which acts on thyroid function as a suppressant.

THIOUREA

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.
for thiourea:
There are reports on disorders of workers coming into contact with thiourea during the course of, for example, maintenance of machinery or packing, without providing any details as to exposure levels. The symptoms observed were typical of hypothyroidism, as evidenced by facial oedema, hypotonia, bradycardia, electrocardiograph alterations associated with reduced basal metabolism, constipation, flatulence, polyuria, and granulocytopenia, accompanied by lymphocytosis and monocytosis. The first perturbations of the blood count were observed after 5-6 months of exposure, and the highest incidence of the symptoms was evident in those workers who had been in contact with the chemical for 5-15 years
Individual cases of contact dermatitis related to the use or processing of thiourea and thiourea compounds have been reported. Some cases showed increased sensitivity to UV light (photocontact dermatitis). Thiourea derivatives such as dimethyl, diethyl, dibutyl, diphenyl, ethylbutyl, and ethylene thiourea are used as accelerators in the vulcanization process in the rubber industry. Products such as wet suits, swimming goggles, orthopaedic devices, protective gloves, and shoes containing these compounds have been shown to produce allergic contact dermatitis.
Administration of thiourea to healthy animals or humans leads to depression of thyroid function. It acts by inhibiting the peroxidase in the thyroid gland, resulting in decreased thyroid hormone production and increased proliferation due to an increase in the secretion of TSH. This could lead to tumour formation. This is a well recognised mechanism of action for non-genotoxic thyroid carcinogens However, no definite conclusion regarding the mechanism of carcinogenicity can be made for thiourea, since it cannot totally be excluded that the possible genotoxicity of thiourea also plays a role.
In humans and animals, thiourea is rapidly absorbed from the gastrointestinal tract. A single oral dose of 28.57 mg thiourea/kg body weight in humans was completely eliminated within 48 h in urine, while a peak concentration in blood was measured within 30 min. In rats administered 5 mg intravenously, 30% of the thiourea was recovered from the carcasses after 3 h, and only traces after 25 h.
Thiourea is also absorbed to a lesser degree through the skin. Following dermal application of 2000 mg/kg body weight to rabbits in the form of an aqueous solution (26 ml of a 25% w/v solution), approximately 4% of the applied dose was found in the animals urine; when applied in solid form, only 0.1% was found in the urine.
Thiourea is oxidised by thyroid gland peroxidase in the presence of iodine or iodide and hydrogen peroxide to form formamidine disulfide (NH2(NH)CSSC(NH)NH2). Formamidine disulfide is unstable and decomposes at pH values above 3.0, forming cyanamide, elementary sulfur, and thiourea. It was shown *in vitro* and *in vivo* that both cyanamide and thiourea are inhibitors of thyroid peroxidase
The acute toxicity of thiourea varies with the species, strain, and age of the animals exposed to the chemical and with the iodine content of their diet. Oral LD50s are about 1000 mg/kg body weight for mice, 125-1930 mg/kg body weight for rats, depending on the strain, and 10 000 mg/kg body weight for rabbits. The intraperitoneal LD50 for the rat ranges between 4 and 1340 mg/kg body weight, according to the strain. Death at these doses is due to lung oedema, and the survivors exhibit pleural effusion. Accordingly, thiourea at doses between 10 and 500 mg/kg body weight has been employed in experimental animal studies as a model agent for the elicitation of lung oedema and pleural effusion. The pathological effects are prevented by pretreatment 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

Continued...

	(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.
STANNOUS METHANESULFONATE	No significant acute toxicological data identified in literature search.
METHANESULFONIC ACID	Skin corrosion/irritation Skin - In vitro study Result: Causes burns. - 4 h (OECD Test Guideline 435) Serious eye damage/eye irritation Eyes - Rabbit Result: Causes burns. (OECD Test Guideline 405) Respiratory or skin sensitization Buehler Test - Guinea pig (OECD Test Guideline 406) Germ cell mutagenicity Test Type: Ames test Test system: Salmonella typhimurium Method: OECD Test Guideline 471 Test Type: In vitro mammalian cell gene mutation test Test system: Chinese hamster ovary cells Method: OECD Test Guideline 476 Test Type: In vivo micronucleus test Species: Mouse Cell type: Bone marrow Application Route: Oral Method: OECD Test Guideline 474 Repeated dose toxicity - Rat - male - Oral - 7 Days - NOAEL (No observed adverse effect level) - = 1,805 mg/kg **WARNING:** This substance has been classified by the IARC as Group 2A: Probably Carcinogenic to Humans. The material may produce severe irritation to the eye causing pronounced 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 produce severe 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) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.
421A Liquid Tin & STANNOUS METHANESULFONATE & METHANESULFONIC ACID	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
421A Liquid Tin & STANNOUS METHANESULFONATE	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.

Continued...

421A Liquid Tin & METHANESULFONIC ACID	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.
METHANESULFONIC ACID	Result: negative Metabolic activation: with and without metabolic activation

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

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

SECTION 12 Ecological information

Toxicity

421A Liquid Tin

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

thiourea

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1008h	Fish	<0.2	7
NOEC(ECx)	504h	Crustacea	0.1mg/l	4
EC50	72h	Algae or other aquatic plants	3.8-10mg/l	1
LC50	96h	Fish	>100mg/l	1
EC50	96h	Algae or other aquatic plants	>=3.8<=5.4mg/l	2
EC50	48h	Crustacea	35mg/l	1

stannous methanesulfonate

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	0.00843mg/l	2
LC50	96h	Fish	>100mg/l	2
EC50	72h	Algae or other aquatic plants	0.407mg/l	2
EC50	48h	Crustacea	>100mg/l	2

methanesulfonic acid

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	24h	Crustacea	1.7mg/l	1
EC50	96h	Algae or other aquatic plants	7.2-20mg/l	2
EC50	72h	Algae or other aquatic plants	>=12<=24mg/l	2
LC50	96h	Fish	73mg/l	2
EC50	48h	Crustacea	12mg/l	1

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

Ecotoxicity:

The tolerance of water organisms towards pH margin and variation is diverse. Recommended pH values for test species listed in OECD guidelines are between 6.0 and almost 9.
Acute testing with fish showed 96h-LC50 at about pH 3.5
for thiourea:
BOD 5: 0.013
COD: 0.84

Environmental fate:

From its very low vapour pressure, a significant adsorption of thiourea onto airborne particles is not expected. Due to its solubility in water (137 g/litre at 20 C), the washout from the atmosphere by wet deposition (fog, rain, snow) is assumed to be significant.
From water solubility and vapour pressure data, a Henry s law constant in the range of 5.58 × 10-9 - 8.44 × 10-9 Pa·m3/mol can be calculated, indicating that thiourea is not expected to volatilise from aqueous solutions. Based on the physicochemical properties of thiourea and its use pattern, the hydrosphere is expected to be the main target compartment for this compound.
Soil sorption coefficients (*Koc*) in the range of 26-315 were determined in studies conducted according to OECD Guideline 106 (adsorption/desorption). The sorption of thiourea onto organic matter of three different soils may be characterized as low (spodosol) to moderate (entisol/alfisol). Neutral thiourea did not undergo any significant ion exchange or other

Continued...

sorption processes in investigations with sorbents such as pure quartz sand, quartz sand coated with polyvinyl alcohol, and quartz sand coated with a mixture of the clay mineral montmorillonite and polyvinyl alcohol. Based on its physicochemical properties, a significant evaporation of thiourea from soil is not to be expected.
Transformation: Thiourea is hydrolytically stable, as measured according to OECD Guideline A-79.74 D. Experimental data on direct photolysis are not available. From the UV spectrum of the substance, direct photolysis in air and water is not to be expected. The extinction coefficients epsilon(max) at lambda(max) (235 and 238 nm) are in the range of 11,000-12,590/mol per second. However, in the atmosphere, the main degradation pathway is probably the reaction of thiourea with hydroxyl radicals. An estimation of the photo-oxidation of thiourea by hydroxyl radicals revealed a half-life of 2.4 h. For the hydrosphere, specific rate constants for the reaction of thiourea with hydrated electrons and hydroxyl radicals are given as 3.0 × 10+9/mol per second (pH 6.4) and 4.7 × 10+9/mol per second (pH 7). Based on a hydroxyl radical concentration of 1 × 10-16 mol/litre in water, a half-life of 17 days can be calculated.
In two studies on ready biodegradability, no mineralisation of thiourea was observed. On the other hand, removal of up to 97% was reported from laboratory tests on inherent biodegradation (Semi-Continuous Activated Sludge, or SCAS, Test), in which the inoculum was very slowly adapted to increasing thiourea concentrations prior to incubation. Cultures of different fungi isolated from soil and grown on glucose and thiourea were shown to degrade thiourea more or less effectively. Whereas *Aspergillus glaucus*, *Penicillium citrinum*, and *Trichoderma viride* took up only 30–50% of an initial thiourea concentration of 0.01% even after long incubation periods of 46 and 106 days and converted not more than 15-17% of thiourea sulfur to sulfate , concentrations in the range of 0.1-0.5 g thiourea/litre were completely removed within 7 days of incubation by *Penicillium rugulosum* . Degradation of thiourea by soil microorganisms was observed Twenty-two per cent of an initial concentration of 1.5 g/litre was degraded within 1 week and 96% within 15 weeks of incubation. Thiourea concentrations exceeding 7.6 g/litre inhibited microbial transformation. In aerobic batch laboratory microcosm experiments, half-lives of 12.8 days (basic soil) and 18.7 days (acid soil) were determined. Although no abiotic controls were performed, removal of thiourea was attributed mainly to biotic processes, assuming abiotic mechanisms (e.g., oxidation, evaporation) to be of minor importance.
From the available degradation tests and taking into account the expected environmental distribution of thiourea, leaching of this compound from soil to ground-water seems possible, particularly under conditions unfavourable for biotic degradation.
Accumulation: Based on the available data on soil sorption, biodegradation in soil, and the calculated *Koc* value, accumulation of thiourea in the geosphere is unlikely.
Due to the low *n*-octanol/water partition coefficient bioaccumulation of thiourea is expected to be insignificant. This assumption is confirmed by the available experimental data. In a study conducted according to OECD Guideline 305C, bioconcentration factors determined for carp (*Cyprinus carpio*) were in the range of <0.2 to <2 (related to whole fish). In another study reported accumulation factors were in the range of <10-90 for golden orfe (*Leuciscus idus*), algae (*Chlorella fusca*), and activated sludge.
Ecotoxicity
Fish LC50 96 h): *Pimephales promelas* (fathead minnow) >100 mg/l (static test):
Fish NOEC (21 d): *Brachydanio rerio* (zebra fish) =>5000 mg/l (semistatic)
Daphnia magna EC50 (24 h): 5.6 mg/l (immobilisation/ static); (96 h) 1.8 mg/l (immobilisation/ static)
Algae EC50 (96 h) *Scenedesmus subspicatus* 4.8-10 mg/l (biomass reduction): 3.8-5.4 mg/l (growth rate)
Bacterial IC50 microbial culture from nitrifying sewage plant 0.8 mg/l (nitrification inhibition test IC75 (2-4 h)
unadapted nitrifying activated sludge 0.075 mg/l (nitrification inhibition test)
Earthworm LC50 (28 d): *Eisenia fetida* 3550 mg/kg soil dry weight
Among the tested organisms, different stages of the red cotton bug (*Dysdercus similis*) proved to be most sensitive, exhibiting EC50 values of 0.03 and 0.025 mg/litre for egg survival and hatching, respectively.
Different fungi were found to be relatively insensitive to thiourea exposure. Complete growth inhibition was observed for *Penicillium rugulosum* after a 7-day exposure to 2000 mg thiourea/litre and for *Helminthosporium sativum* and *Fusarium oxysporum* after a 15-day exposure to 750 mg/litre and 1000 mg/litre, respectively.
Terrestrial plants proved to be generally more sensitive. Whereas thiourea concentrations below 12 mg/litre increased the growth of excised tomato roots (*Lycopersicum esculentum*) within 4 weeks of exposure in a defined basal medium, 18, 23, and 46 mg/litre reduced growth by about 45%, 60%, and 30%, respectively.
Prevent, by any means available, spillage from entering drains or water courses.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
thiourea	LOW	LOW
methanesulfonic acid	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
thiourea	LOW (BCF = 2)
methanesulfonic acid	LOW (LogKOW = -2.3817)

Mobility in soil

Ingredient	Mobility
thiourea	MEDIUM (KOC = 2.782)
methanesulfonic acid	HIGH (KOC = 1)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. ▸ Recycle wherever possible. ▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▸ Treat and neutralise at an approved treatment plant. Treatment should involve: Neutralisation with soda-ash or soda-lime followed by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus ▸ Decontaminate empty containers with 5% aqueous sodium hydroxide or soda ash, followed by water. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 Transport information

Labels Required

Continued...

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

Land transport (ADG)

UN number or ID number	1760
UN proper shipping name	CORROSIVE LIQUID, N.O.S. (contains methanesulfonic acid and stannous methanesulfonate)
Transport hazard class(es)	Class: 8 Subsidiary risk: Not Applicable
Packing group	II
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: 274 Limited quantity: 1 L

Air transport (ICAO-IATA / DGR)

UN number	1760
UN proper shipping name	Corrosive liquid, n.o.s. * (contains methanesulfonic acid and stannous methanesulfonate)
Transport hazard class(es)	ICAO/IATA Class: 8 ICAO / IATA Subrisk: Not Applicable ERG Code: 8L
Packing group	II
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: A3 A803 Cargo Only Packing Instructions: 855 Cargo Only Maximum Qty / Pack: 30 L Passenger and Cargo Packing Instructions: 851 Passenger and Cargo Maximum Qty / Pack: 1 L Passenger and Cargo Limited Quantity Packing Instructions: Y840 Passenger and Cargo Limited Maximum Qty / Pack: 0.5 L

Sea transport (IMDG-Code / GGVSee)

UN number	1760
UN proper shipping name	CORROSIVE LIQUID, N.O.S. (contains methanesulfonic acid and stannous methanesulfonate)
Transport hazard class(es)	IMDG Class: 8 IMDG Subrisk: Not Applicable
Packing group	II
Environmental hazard	Not Applicable
Special precautions for user	EMS Number: F-A, S-B Special provisions: 274 Limited Quantities: 1 L

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

Not Applicable

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

Product name	Group
thiourea	Not Available
stannous methanesulfonate	Not Available
methanesulfonic acid	Not Available

Transport in bulk in accordance with the IGC Code

Product name	Ship Type
thiourea	Not Available
stannous methanesulfonate	Not Available
methanesulfonic acid	Not Available

Continued...

SECTION 15 Regulatory information

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

thiourea is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

stannous methanesulfonate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

methanesulfonic acid is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

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

SECTION 16 Other information

Revision Date	02/11/2021
Initial Date	02/03/2020

SDS Version Summary

Version	Date of Update	Sections Updated
2.00	02/11/2021	Toxicological information - Acute Health (swallowed), Toxicological information - Chronic Health, Firefighting measures - Fire Fighter (fire/explosion hazard), Exposure controls / personal protection - Personal Protection (Respirator)

Other information

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

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
ES: Exposure Standard
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index
AIIC: Australian Inventory of Industrial Chemicals
DSL: Domestic Substances List
NDSL: Non-Domestic Substances List
IECSC: Inventory of Existing Chemical Substance in China
EINECS: European INventory of Existing Commercial chemical Substances

Continued...

ELINCS: European List of Notified Chemical Substances
NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory
KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals
PICCS: Philippine Inventory of Chemicals and Chemical Substances
TSCA: Toxic Substances Control Act
TCSI: Taiwan Chemical Substance Inventory
INSQ: Inventario Nacional de Sustancias Químicas
NCI: National Chemical Inventory
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances