

AC Militate

AXICHEM Pty Ltd

Chemwatch: 5218-90

Version No: 4.1

Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Chemwatch Hazard Alert Code: 3

Issue Date: 03/09/2020

Print Date: 11/10/2022

L.GHS.AUS.EN

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

Product Identifier

Product name	AC Militate
Chemical Name	Not Applicable
Synonyms	Not Available
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Used for the control of annual and perennial grasses and burrs in cotton, annual and perennial grasses in sugar cane, grass weeds in couch turf, prickly pear and grasses and burrs in non-crop areas.
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Details of the manufacturer or supplier of the safety data sheet

Registered company name	AXICHEM Pty Ltd
Address	9 Palings Court Nerang QLD 4211 Australia
Telephone	07 5596 1736
Fax	Not Available
Website	www.axichem.com.au
Email	msds@axichem.com.au

Emergency telephone number

Association / Organisation	CHEMWATCH EMERGENCY RESPONSE
Emergency telephone numbers	+61 1800 951 288
Other emergency telephone numbers	+61 3 9573 3188

Once connected and if the message is not in your preferred language then please dial 01

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	S7
Classification ^[1]	Acute Toxicity (Inhalation) Category 3, Carcinogenicity Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 1, Acute Toxicity (Oral) 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)

H331	Toxic if inhaled.
H351	Suspected of causing cancer.
H373	May cause damage to organs through prolonged or repeated exposure.
H410	Very toxic to aquatic life with long lasting effects.
H301	Toxic if swallowed.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe mist/vapours/spray.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves and protective clothing.
P273	Avoid release to the environment.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P330	Rinse mouth.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P311	Call a POISON CENTER/doctor/physician/first aider.
P391	Collect spillage.

Precautionary statement(s) Storage

P403+P233	Store in a well-ventilated place. Keep container tightly closed.
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.
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SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
2163-80-6	70-80	monosodium methylarsonate
Not Available		(800 g/L)
Not Available	balance	Ingredients determined not to be hazardous

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	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
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Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▸ 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.
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.

Indication of any immediate medical attention and special treatment needed

For acute or short term repeated exposures to arsenic, soluble compounds: Treat as per arsenic poisoning.

- Acute skin lesions such as contact dermatitis usually do not require other treatment than removal from exposure.
- If more severe symptoms of the respiratory system, the skin or the gastro-intestinal tract occur, British Anti-Lewisite (BAL, dimercaprol) may be given. Prompt administration in such cases is vital; to obtain maximum benefit such treatment should be administered within 4 hours of poisoning.
- In addition, general treatment such as prevention of further absorption from the gastro-intestinal tract are mandatory.
- General supportive therapy such as maintenance of respiration and circulation, maintenance of water and electrolyte balance and control of nervous system effects, as well as elimination of absorbed poison through dialysis and exchange transfusion, may be used if feasible.
- Dimercaprol is given by deep intramuscular injection as a 5% solution in peanut oil (or a 10% solution with benzyl-benzoate in vegetable oil). It is usually given in a dose of 3 mg/kg, 4-hourly, for the first two days, or twice daily for up to seven days. [ILO Encyclopedia]
- BAL Therapy is effective for haematological manifestations of chronic arsenic poisoning but not for neurological symptoms. Watch for side effects (e.g. urticaria, burning sensation in the lips, mouth and throat, fever, conjunctivitis etc).
- Some relief results from administration of diphenhydramine (Benadryl) (1.5 mg/kg intramuscularly or by mouth every 6 hour). [Ellenhorn and Barceloux: Medical Toxicology]

BIOLOGICAL EXPOSURE INDEX - BEI (Notice of Intent to Establish)

BEIs represent the levels of determinants which are most likely to be observed in specimens collected from a healthy worker who has been exposed to chemicals to the same extent as a worker with inhalation exposure to the

Exposure Standard (ES or TLV):

Determinant	Index	Sampling Time	Comments
Inorganic arsenic metabolites in urine	35 ug/gm creatinine	End of workweek	B

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

SECTION 5 Firefighting measures

Extinguishing media

- There is no restriction on the type of extinguisher which may be used.

Special hazards arising from the substrate or mixture

Fire Incompatibility	▸ Avoid reaction with oxidising agents
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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.
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AC Militate

Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Non combustible. ▶ Not considered to be a significant fire risk. ▶ Expansion or decomposition on heating may lead to violent rupture of containers. ▶ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. <p>Decomposes on heating and produces toxic fumes of: arsenic compounds</p>
HAZCHEM	Not Applicable

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	<ul style="list-style-type: none"> ▶ 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	<p>Environmental hazard - contain spillage.</p> <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. ▶ 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	<ul style="list-style-type: none"> ▶ Limit all unnecessary personal contact. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ 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. ▶ 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	<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.

Conditions for safe storage, including any incompatibilities

AC Militate

Suitable container	Plastic container Polylined drum
Storage incompatibility	Avoid storage with oxidisers

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	monosodium methylarsonate	Arsenic & soluble compounds (as As)	0.05 mg/m ³	Not Available	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
AC Militate	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
monosodium methylarsonate	Not Available	Not Available

MATERIAL DATA

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.	
	General exhaust is adequate under normal operating conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.	
	Type of Contaminant:	Air Speed:
	solvent, vapours, degreasing etc., evaporating from tank (in still air)	0.25-0.5 m/s (50-100 f/min)
	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min)
	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)
	Within each range the appropriate value depends on:	
	Lower end of the range	Upper end of the range
	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only	2: Contaminants of high toxicity	
3: Intermittent, low production.	3: High production, heavy use	
4: Large hood or large air mass in motion	4: Small hood - local control only	
Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the		

	extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
Personal protection	
Eye and face protection	<ul style="list-style-type: none"> ▸ Safety glasses. ▸ Safety glasses with side shields. ▸ Chemical goggles. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	<p>Wear chemical protective gloves, e.g. PVC.</p> <p>Wear safety footwear.</p> <ul style="list-style-type: none"> ▸ Elbow length PVC gloves
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. <p>Full body protecting overalls required for other than spot spraying Consider where spray drift may fall and DO NOT spray into wind</p>

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Pink liquid with a pleasant fruity odour; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable
pH (as supplied)	Not Available	Decomposition temperature (°C)	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 Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (Not Available%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▸ 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	<p>Symptoms of exposure resemble those for ingestion. In addition, inhalation may result in pulmonary oedema, restlessness, breathing difficulties, cyanosis, restlessness, cyanosis, and coughing with foamy sputum and rales.</p> <p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may produce toxic effects.</p> <p>Inhalation of arsenic containing materials resemble those described for ingestion but in addition may produce severe nose and respiratory tract irritation.</p> <p>Acute inhalation exposure can cause cough (with foamy sputum and rales), chest pain, shortness of breath, dizziness, headache, pulmonary oedema, extreme general weakness and cyanosis.</p> <p>High exposures can cause poor appetite, nausea, vomiting and muscle cramps. Heart effects with abnormal EKG can also occur with very high exposures</p> <p>Prolonged or repeated exposures may produce necrosis and perforation of the nasal septum.</p>
Ingestion	<p>Toxic effects may result from the accidental ingestion of the material; animal experiments indicate that ingestion of less than 40 gram may be fatal or may produce serious damage to the health of the individual.</p> <p>Symptoms of acute poisoning by arsenic ingestion, which develop within 4 hours include epigastric pain, vomiting and watery diarrhoea, nausea and vomiting. Blood may appear in vomitus and stools. If amount ingested is sufficiently high, shock, rapid pulse and coma may develop, followed by death within 24 hours.</p> <p>Severe gastritis or gastroenteritis may occur as a result of lesions produced by vascular damage from absorbed arsenic (and not local corrosion); symptoms may be delayed for several hours. Eventually a violent haemorrhagic gastroenteritis leads to profound loss of fluid and electrolyte resulting in shock and death. Occasionally alimentary symptoms are mild or absent in which case symptoms are usually referable to the central nervous system, headache, vertigo, muscle spasm or convulsion, delirium and, sometimes, mania. In advanced poisonings by arsenic and its inorganic salts, nervous symptoms are prominent; disorders of the brain (encephalopathies) and peripheral neuritis (more commonly) have been described. A prickling sensation (paresthesia), decreased sensitivity to sensation and pain (hypoesthesia), eventually paralysis and muscular atrophy appear, usually in the legs. "Glove and stocking" distribution of sensory loss may be prominent. The toxic moiety is presumed to be trivalent arsenic in the form of inorganic arsenious acid (arsenite) or an organic arsenoxide. Arsenites are active enzyme inhibitors. Arsenic and its compounds 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.</p>
Skin Contact	<p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>Arsenic and its compounds may irritate the skin. Certain individuals may develop sensitisation dermatitis characterised by eczema with scaling and hyperpigmentation of the skin and hyperkeratosis of the palms of the hands and soles of the feet.</p> <p>Skin contact may cause erythema (an abnormal redness caused by capillary congestion), with burning, itching, swelling and skin eruptions.</p> <p>Repeated skin contact can cause thickened skin and/or patchy areas of darkening and loss of pigment. Some persons develop white lines on the nails.</p>
Eye	<p>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
Chronic	<p>Symptoms of chronic arsenic poisoning, by inhalation, include weight loss, nausea and diarrhoea alternating with constipation, pigmentation and eruption of the skin, loss of hair, peripheral neuritis, blood disorders (anaemia), striations on fingernails and toenails. Long-term exposure can cause an ulcer or hole in the "bone" dividing the inner nose. Hoarseness and sore eyes also occur. anaemia, liver and kidney injury, skin and lung cancer. Renal damage may develop after acute or chronic exposures.</p> <p>Prolonged inhalation of arsenical dusts are implicated in lung cancer.</p> <p>Subacute and chronic exposure to arsenic and its organic salts may produce anorexia, mild gastrointestinal disturbances (nausea and vomiting), low grade fever, persistent headache, pallor, weakness, catarrhal inflammation and neuropathies with severe crippling effects. Stomatitis (oral lesions) and salivation are common. High or repeated exposure can cause nerve damage with "pins and needles" burning, numbness, and later weakness (incoordination) of arms and legs, anaemia, liver and kidney injury, skin and lung cancer. Repeated exposure can also damage the liver, causing narrowing of the blood vessels, or</p>

interfere with bone marrow's ability to make red blood cells.

Skin afflictions are many and varied; erythema, eczema, pigmentation (arsenic melanosis), diffuse alopecia, keratosis (especially of the palms and soles), scaling and desquamation, brittle nails, white lines or bands on the nails (Mees lines), loss of hair and nails and localised subcutaneous oedema (especially of the eye-lids). Signs of renal damage may also develop. Liver enlargement (hepatomegaly) with jaundice (and sometimes pruritus) may develop into cirrhosis with accumulation of body fluids in the abdominal cavity (ascites). Nervous system effects involving the extremities (numbness, tingling, burning pain, weakness, incoordination) may also occur.

Arsenic exposure is linked with increase in diseases including ischaemia, cerebrovascular disease and carotid atherosclerosis. Several cytokines and growth factors involved in inflammation, have been identified in humans after prolonged exposure; these may heighten the risk of atherosclerosis (1). Individual variability in arsenic metabolism may determine susceptibility to arsenic disease. Genetic polymorphism (variability) for enzymes instrumental in arsenic metabolism (purine nucleoside phosphorylase and glutathione S-transferase omega 1-1) has been identified amongst individuals of European and Mexican Hispanic (indigenous) ancestry (2).

Many cases of skin cancer have been reported among people exposed to arsenic through medical treatment with inorganic trivalent arsenic compounds. In some instances skin cancers have occurred in combination with other cancers, such as liver angiosarcoma, intestinal and urinary bladder carcinomas and meningioma. Epidemiological studies of cancer after medical treatment have shown an excess of skin cancers but no clear association with other cancers has been shown.

Prolonged inhalation of arsenical dusts are implicated in lung cancer. Occupational exposure to inorganic arsenic, especially in mining and copper smelting, has consistently been associated with an increased risk of cancer. An almost tenfold increase in the incidence of lung cancer was found in workers most heavily exposed to arsenic and relatively clear dose-response relationships have been obtained with regard to cumulative exposure. Other smelter worker population's have been shown to have consistent increases in lung cancer incidence, as well as increases of about 20% in the incidence of gastrointestinal cancer and of 30% for renal cancer and haematolymphatic malignancies

An association between environmental exposure to arsenic through drinking water and skin cancer has been observed and confirmed. Epidemiological studies in areas where drinking water contained 0.35-1.14 mg/l arsenic elevated risks for cancers of the bladder, kidney, skin, liver, lung and colon in both men and women. Taiwanese exposed to naturally occurring arsenic in well-water have experienced skin cancer. Inorganic and organic arsenics are established carcinogens in man. Chronic human exposure to non-overtly toxic doses of arsenic is associated with carcinogenicity, although the mechanism is not fully understood. Arsenic does not directly cause DNA damage or mutations. Data indicates that nontoxic doses of arsenite can interact with glucocorticoid receptor (GR) complexes and selectively inhibit GR-mediated transcription which is associated with altered nuclear function (3).

(1) Wu et al; Environmental Health Perspectives, 111, 1429-1438, 2003
 (2) Yu et al; Environmental Health Perspectives, 111, 1421-1427, 2003
 (3) Kaltreider et al; Environmental Health Perspectives, 109, pp 245-251, 2001

AC Militate	TOXICITY	IRRITATION
	Not Available	Not Available
monosodium methylarsonate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 2500 mg/kg ^[2]	Eye (rabbit): mild *
	Inhalation(Rabbit) LC50; >20 mg/L4h ^[2]	Skin (rabbit): mild *
	Oral (Rabbit) LD50; 102 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	

MONOSODIUM METHYLARSONATE	<p>Toxicity Class: WHO III; EPA III *</p> <p>Arsenic is a potent toxicant that may exist in several oxidation states and in a number of inorganic and organic forms. Most cases of arsenic-induced toxicity in humans are due to exposure to inorganic arsenic, and there is an extensive database on the human health effects of the common arsenic oxides and oxyacids. Although there may be some differences in the potency of different chemical forms (e.g., arsenites tend to be somewhat more toxic than arsenates), these differences are usually minor. Humans may be exposed to organic arsenicals (mainly methyl and phenyl derivatives of arsenic acid) that are used in agriculture and to organic arsenicals found in fish and shellfish (arsenobetaine and arsenocholine). Although the toxicity of organic arsenicals has not been as extensively investigated as inorganic arsenicals, there are sufficient animal data to evaluate the toxicity of methyl arsenates (e.g., monomethylarsonic acid [MMA] and dimethylarsinic acid [DMA]) and roxarsone. The so-called "fish arsenic" compounds (e.g., arsenobetaine) are not thought to be toxic.</p> <p>The methylation of inorganic arsenic may yield metabolites that alter the cellular oxidation status by potently inhibiting the reduction of glutathione disulfide. The alteration of the oxidation status of the cell by these arsenicals may lead to more serious cytotoxic effects.</p> <p>It is generally accepted that the arsenic-carbon bond is quite strong and most mammalian species do not have the capacity to break this bond; thus, inorganic arsenic is not formed during the metabolism of organic arsenicals. In most species, including humans, ingested (or exogenous) MMA(V) and DMA(V) undergo limited metabolism, do not readily enter the cell, and are primarily excreted unchanged in the urine. This is in contrast to inorganic arsenic, which undergoes sequential reduction and methylation reactions leading to the formation of MMA and DMA. Inorganic As(V) is readily reduced to inorganic As(III), which is taken up by the cell (primarily in the liver), As(III) is methylated to form MMA(V), which is reduced to MMA(III); MMA(III) subsequently undergoes oxidative methylations to form DMA(V). DMA(V) is the primary excretion product in humans. Because inorganic and organic arsenicals exhibit distinct toxicokinetic characteristics, the health effects and MRLs are</p>
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considered separately.

There are limited data on the toxicity of organic arsenicals following inhalation exposure in humans and animals and these data do not allow for identification of critical effects. Keratosis was observed in workers exposed to 0.065 mg/m³ arsanilic acid (i.e., 4-aminophenyl arsenic acid); no alterations in gastrointestinal symptoms or haematological alterations were observed. In animals, very high concentrations (>3,000 mg/m³) of DMA results in respiratory distress, diarrhea, and erythematous lesions on the feet and ears. No adverse effects were observed in rats exposed to DMA concentrations as high as 100 mg DMA/m³ for 95 days.

Similarly, the available dermal toxicity data do not allow for identification of critical effects. Contact dermatitis was observed in workers applying DMA (and its sodium salt) and mild dermal irritation was observed in a Draize test in rabbits (adverse effect level not reported). Intermediate duration (21 days) exposure studies in rabbits did not result in systemic toxicity or skin irritation following 5 day/week exposure to 1,000 mg/kg/day MMA or DMA.

The preponderance of toxicity data for organic arsenicals involves oral exposure. Human data are limited to three case reports of individuals intentionally ingesting pesticides containing organic arsenicals. Gastrointestinal irritation (vomiting, nausea, and diarrhea) were consistently reported in these cases.

Animal data has primarily focused on the toxicity of MMA, DMA, and roxarsone; these data suggest that the targets of toxicity may differ between the compounds. Common signs of toxicity of the organic arsenicals in mice include depression of motility and respiration, irritability, ataxia and convulsions. Death appears to be due to respiratory depression. For DMA and TMAO, a period of increased spontaneous motility preceded the death of the mice. More than half of the mice that received a lethal dose of MMA, DMA or TMAO had diarrhoea, which may have contributed to their deaths.

The LD50s for the organic arsenicals are lower after parenteral administration than after oral administration.

Inorganic arsenic but not organic arsenic induces keratinocyte hyperproliferation and disrupts the process of terminal epidermal differentiation in the epidermis.

Since methylation serves to expedite the excretion of inorganic arsenic, which is more toxic than organoarsenicals, issues such as whether demethylation occurs and if methylation is saturable, inducible, or inhibitable under expected environmental exposure conditions are critical.

Genotoxic effects induced by the organic arsenicals include excess tetraploids (DMA, TMAO), and mitotic arrest (MMA, DMA and TMAO) in Chinese hamster lung (V79) cells. Arsenocholine and arsenobetaine are not genotoxic in V79 cells. In mouse lymphoma cells (L5178Y/TK+/-), incubation of organic arsenicals for 4 h induced cytotoxicity and clastogenicity. MMA was more potent than DMA, but less so than the inorganic arsenicals.

In vitro studies have proven DMA to be a potent clastogenic agent, capable of inducing DNA damage including double strand breaks and cross-link formation.

Reports of successful cancer induction in animals by inorganic arsenic (arsenite and arsenate) have been rare, and most carcinogenic studies have used organic arsenicals such as DMA combined with other tumor initiators. In rats, the methylated arsenicals, dimethylarsinic acid promotes diethylnitrosamine-initiated liver tumors, whereas trimethylarsine oxide induces liver adenomas. There are a variety of potential mechanisms for arsenical-induced hepatocarcinogenesis, such as oxidative DNA damage, impaired DNA damage repair, acquired apoptotic tolerance, hyperproliferation, altered DNA methylation, and aberrant estrogen signaling. Some of these mechanisms may be liver specific/selective. Overall, accumulating evidence clearly indicates that the liver could be an important target of arsenic carcinogenesis.

The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic).

This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

Arsenic compounds are classified by the European Union as toxic by inhalation and ingestion and toxic to aquatic life and long lasting in the environment. IARC classify arsenic in drinking water as a confirmed human carcinogen (IARC 1).

The main inorganic forms of arsenic relevant for human exposures are pentavalent arsenic (also called arsenate, As(V), or As⁺⁵) and trivalent arsenic (also called arsenite, As(III), or As⁺³). These inorganic species undergoes a series of reduction and oxidative/methylation steps in human liver and other tissues to form tri- and pentavalent methylated metabolites of methylarsonite [MA(III)], methylarsonate [MA(V)], dimethylarsinite [DMA(III)], and dimethylarsinate [DMA(V)]. Some mammalian species also produce trimethylated metabolites, trimethylarsine oxide.

The distinction between inorganic and organic forms is important because it is generally accepted that the organic species are excreted more quickly from the body and generally considered less toxic, with a relative rank order of As(III) > As(V) >> MA(V), DMA(V) >> arsenobetaine. However, the methylated trivalent metabolites, MA(III) and DMA(III), are significantly more toxic than their pentavalent counterpart and either As(III) or As(V). In many cases, biomonitoring or environmental occurrence data are reported as total arsenic and do not distinguish between the different species. In those situations, understanding the relevant sources of arsenic is essential to evaluate potential arsenic related health effects, especially those related to inorganic arsenic exposure.

[* The Pesticides Manual, Incorporating The Agrochemicals Handbook, 10th Edition, Editor Clive Tomlin, 1994, British Crop Protection Council]

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

AC Militate

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

SECTION 12 Ecological information

Toxicity

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

monosodium methylarsonate	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	>51mg/l	Not Available
	EC50	48h	Crustacea	77.5mg/l	Not Available
	EC50(ECx)	48h	Crustacea	77.5mg/l	Not Available

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

Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Toxic to bees.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
	No Data available for all ingredients	No Data available for all ingredients

Bioaccumulative potential

Ingredient	Bioaccumulation
	No Data available for all ingredients

Mobility in soil

Ingredient	Mobility
	No Data available for all ingredients


SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	
	<ul style="list-style-type: none"> ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Management Authority for disposal. ▶ Treat and neutralise at an effluent treatment plant. ▶ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

Marine Pollutant	
HAZCHEM	Not Applicable

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

Continued...

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

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

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

Not Applicable

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

Product name	Group
monosodium methylarsonate	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
monosodium methylarsonate	Not Available

SECTION 15 Regulatory information

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

monosodium methylarsonate is found on the following regulatory lists

Australia Chemicals with non-industrial uses removed from the Australian Inventory of Chemical Substances (old Inventory)

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7

Chemical Footprint Project - Chemicals of High Concern List

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (monosodium methylarsonate)
Canada - NDSL	No (monosodium methylarsonate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (monosodium methylarsonate)
Korea - KECI	No (monosodium methylarsonate)
New Zealand - NZIoC	No (monosodium methylarsonate)
Philippines - PICCS	No (monosodium methylarsonate)
USA - TSCA	No (monosodium methylarsonate)
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	No (monosodium methylarsonate)
Russia - FBEPH	No (monosodium methylarsonate)
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	03/09/2020
Initial Date	10/08/2016

SDS Version Summary

Version	Date of Update	Sections Updated
3.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification

Version	Date of Update	Sections Updated
4.1	03/09/2020	Classification change due to full database hazard calculation/update.

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
 ELINCS: European List of Notified Chemical Substances
 NLP: No-Longer Polymers
 ENCS: Existing and New Chemical Substances Inventory
 KECl: 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

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