Apparent Barefoot Selective Herbicide

AIRR Apparent Pty Ltd.

Chemwatch Hazard Alert Code: **3**

Chemwatch: **9656-80**
Version No: **2.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Initial Date: **12/11/2026**
Revision Date: **12/11/2026**
Print Date: **23/11/2026**
L.GHS.AUS.EN.E

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

Product Identifier

Product name	Apparent Barefoot Selective Herbicide
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	PESTICIDE, LIQUID, TOXIC, N.O.S. (contains disodium methylarsonate)
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	For the control of Paspalum, Nutgrass, Mullumbimby Couch, Clovers and other weeds in domestic lawns. Use according to manufacturer's directions.

Details of the manufacturer or importer of the safety data sheet

Registered company name	**AIRR Apparent Pty Ltd.**
Address	15/16 Princes Street, Newport NSW 2106 Australia
Telephone	+61 3 5820 8400
Fax	Not Available
Website	www.apparent.com.au
Email	enquiries@apparentag.com.au

Emergency telephone number

Association / Organisation	**AIRR Apparent Pty Ltd.**
Emergency telephone number(s)	1800 033 111 (24 Hours)
Other emergency telephone number(s)	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	S6
Classification [1]	Acute Toxicity (Oral) Category 3, Acute Toxicity (Inhalation) Category 3, Carcinogenicity Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 2
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)

H301	Toxic if swallowed.
H331	Toxic if inhaled.
H351	Suspected of causing cancer.
H411	Toxic to aquatic life with long lasting effects.

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Precautionary statement(s) Prevention

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.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P202	Do not handle until all safety precautions have been read and understood.

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.

No further product hazard information.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
144-21-8	7.4	disodium methylarsonate
3653-48-3	2.4	MCPA sodium salt
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	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 contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes 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.
Ingestion	▸ **IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY.** ▸ For advice, contact a Poisons Information Centre or a doctor. ▸ Urgent hospital treatment is likely to be needed. ▸ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. ▸ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist. ▸ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS. **Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:**

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- **INDUCE** vomiting with fingers down the back of the throat, **ONLY IF CONSCIOUS**. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.

NOTE: Wear a protective glove when inducing vomiting by mechanical means.

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.

* Preplacement and periodic medical examinations are essential for workers exposed to arsenic on a regular basis. Preplacement physical examinations should give particular attention to allergic and chronic skin lesions, eye disease, psoriasis, chronic eczematous dermatitis, hyperpigmentation of the skin, keratosis and warts, baseline weight, baseline blood and haemoglobin counts, baseline urinary arsenic determinations.
Annual physical examinations should give attention to general health, weight, skin condition, and any evidence of excessive exposure or absorption of arsenic
Following exposures to chlorophenoxy compounds:

- Acute toxic reactions are rare. The by-product of production, dioxin, may be implicated in subacute features such as hepatic enlargement, chloracne, neuromuscular symptoms and deranged porphyrin metabolism.
- Large intentional overdoses result in coma, metabolic acidosis, myalgias, muscle weakness, elevated serum creatine kinase, myoglobinuria, irritation of the skin, eyes, respiratory tract and gut and mild renal and hepatic dysfunction.
- Several cases of sensorimotor peripheral neuropathies have been associated with chronic dermal exposure to 2,4-D. For acute exposures the usual methods of gut and skin contamination (lavage, charcoal, cathartic) are recommended in the first several hours. Alkalisation of the urine and generous fluid replacement have the added benefit of treating any myoglobinuria present. Monitor metabolic acidosis, hyperthermia, hyperkalaemia, myoglobinuria and hepatic/renal dysfunction. for 2,4-dichlorophenoxyacetic acid (2,4-D) and its derivatives
- Gastric lavage if there are no signs of impending convulsions.
- Cautious administration of short-acting anticonvulsant drug if convulsions appear imminent.
- General supportive measures for central nervous system depression.
- If hypotension appears, search vigorously for a contributing cause (e.g. dehydration, electrolyte balance, acidosis, myocardial disturbances and hyperpyrexia).
- As appropriate, treat dehydration, electrolyte disturbances, acidosis, and hyperexia.
- To promote excretion of 2,4-D, initiate alkaline diuresis, as in salicylate poisoning by injecting sodium bicarbonate, intravenously, until the urine pH exceeds 7.5 and then infuse mannitol; renal clearance rises sharply as urine pH rises above 7.5 - above pH 8.0, it is said to be 100-fold greater than pH 6.0.
- If cardiac disturbances are suspected, monitor ECG continuously when possible. Prepare to deliver defibrillating shocks in the event of ventricular fibrillation.
- If hypotension intensifies, a trial with a vasopressor drug may be appropriate. Adrenalin (epinephrine) should be avoided because of possible fibrillation.
- If myotonia appears, a trial with quinidine may be helpful.
- Physiotherapy may be necessary for motion disorders associated with peripheral neuritis, myopathy or brain stem dysfunction.

GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products, 5th Ed.
In general, chlorophenoxy herbicides are rapidly absorbed from the gastrointestinal tract and evenly distributed throughout the body; accumulation in human tissues is not expected A steady-state level in the human body will be achieved within 3–5 days of exposure. The herbicides are eliminated mainly in the urine, mostly unchanged, although fenoprop may be conjugated to a significant extent Biological half-lives of chlorophenoxy herbicides in mammals range from 10 to 33 h; between 75% and 95% of the ingested amount is excreted within 96 h. Dogs appear to retain chlorophenoxy acids longer than other species as a result of relatively poor urinary clearance and thus may be more susceptible to their toxic effects. Metabolic conversions occur only at high doses. The salt and ester forms are rapidly hydrolysed and follow the same pharmacokinetic pathways as the free acids

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 substance 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.

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	‣ Slight fire hazard when exposed to heat or flame. ‣ Heating may cause expansion or decomposition leading to violent rupture of containers. ‣ On combustion, may emit toxic fumes of carbon monoxide (CO). ‣ May emit acrid smoke. ‣ Mists containing combustible materials may be explosive. Combustion products include: ‣ carbon dioxide (CO_2) hydrogen chloride phosgene arsenic compounds ‣ other pyrolysis products typical of burning organic material. May emit poisonous fumes.
HAZCHEM	2X

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. ‣ Remove all ignition sources. ‣ 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	Environmental hazard - contain spillage. ‣ 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	‣ Avoid skin contact, including inhalation. ‣ Wear protective clothing when risk of exposure occurs. ‣ Use in a well-ventilated area. ‣ Prevent concentration in hollows and sumps. ‣ **DO NOT** enter confined spaces until atmosphere has been checked. ‣ **DO NOT** allow material to come in direct contact with human skin or eyes. ‣ **DO NOT** allow material to come in contact with exposed food or food contact surfaces. ‣ Suitable PPE must be worn at all times. ‣ 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. ‣ **DO NOT** allow clothing wet with material to stay in contact with skin
Other information	‣ Store in original containers. ‣ Keep containers securely sealed. ‣ Store in a cool, dry, well-ventilated area. ‣ Store away from incompatible materials and foodstuff containers. ‣ Protect containers against physical damage and check regularly for leaks. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	‣ Lined metal can, lined metal pail/ can. ‣ Plastic pail. ‣ Polyliner drum. ‣ Packing as recommended by manufacturer. ‣ Check all containers are clearly labelled and free from leaks. For low viscosity materials ‣ Drums and jerricans must be of the non-removable head type.

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	‣ 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, there must be sufficient inert cushioning material in contact with inner and outer packages *. - In addition, where inner packagings are glass and contain liquids of packing group I and II there must be sufficient inert absorbent to absorb any spillage *. - * unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	The substance may be or contains a "metalloid" The following elements are considered to be metalloids; boron,silicon, germanium, arsenic, antimony, tellurium and (possibly) polonium The electronegativities and ionisation energies of the metalloids are between those of the metals and nonmetals, so the metalloids exhibit characteristics of both classes. The reactivity of the metalloids depends on the element with which they are reacting. For example, boron acts as a nonmetal when reacting with sodium yet as a metal when reacting with fluorine. Unlike most metals, most metalloids are amphoteric- that is they can act as both an acid and a base. For instance, arsenic forms not only salts such as arsenic halides, by the reaction with certain strong acid, but it also forms arsenites by reactions with strong bases. Most metalloids have a multiplicity of oxidation states or valences. For instance, tellurium has the oxidation states +2, -2, +4, and +6. Metalloids react like non-metals when they react with metals and act like metals when they react with non-metals. ‣ Avoid reaction with oxidising agents

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	disodium methylarsonate	Arsenic & soluble compounds (as As)	0.05 mg/m3	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.
Australia Workplace exposure limits for airborne contaminants (WEL list) - Appendix A - Workplace Exposure Limits	disodium methylarsonate	Arsenic and compounds (except arsine)	0.01 mg/m3	Not Available	Not Available	The use, handling and storage of this chemical is subject to restriction or prohibition (see regulations 340, 380 - 384 and Schedule 10 to the model WHS Regulations).

MATERIAL DATA

Use control measures / protective gear to avoid any personal contact. The ES-TWA is based solely on the prevention of systemic effects due to inhalation and is not protective against the substantial risk of cancer produced by exposure to inorganic arsenic. Some jurisdictions require health surveillance be performed on occupationally exposed workers.

Such surveillance should emphasise
- demography, occupational and medical history and health advice
- physical examination with emphasis on the peripheral nervous system and skin
- urinary total arsenic
- records of personal exposure

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

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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.

Individual protection measures, such as personal protective equipment



Eye and face protection

- Safety glasses with side shields.
- Chemical goggles.[AS/NZS 1337.1, EN166 or national equivalent]
- 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].

Skin protection

See Hand protection below

Hands/feet protection

- Wear chemical protective gloves, e.g. PVC.
- Wear safety footwear or safety gumboots, e.g. Rubber

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.

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
· frequency and duration of contact,
· chemical resistance of glove material,
· glove thickness and
· dexterity
Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
· Contaminated gloves should be replaced.
As defined in ASTM F-739-96 in any application, gloves are rated as:
· Excellent when breakthrough time > 480 min
· Good when breakthrough time > 20 min
· Fair when breakthrough time < 20 min
· Poor when glove material degrades
For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.
It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.
Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.
Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:
· Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.
· Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential
Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

Body protection

See Other protection below

Other protection

- Overalls.
- Eyewash unit.
- Barrier cream.
- Skin cleansing cream.

Respiratory protection

Type A 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	A-AUS	-	A-PAPR-AUS / Class 1

Continued...

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

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

- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Slightly cloudy brown liquid.

Physical state	Liquid	**Relative density (Water = 1)**	1.2
Odour	No Odour	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (mm2/s)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**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
Heat of Combustion (kJ/g)	Not Available	**Ignition Distance (cm)**	Not Available
Flame Height (cm)	Not Available	**Flame Duration (s)**	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	**Enclosed Space Ignition Deflagration Density (g/m3)**	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

a) Acute Toxicity	There is sufficient evidence to classify this material as acutely toxic.
b) Skin Irritation/Corrosion	Based on available data, the classification criteria are not met.
c) Serious Eye Damage/Irritation	Based on available data, the classification criteria are not met.
d) Respiratory or Skin sensitisation	Based on available data, the classification criteria are not met.
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	There is sufficient evidence to classify this material as carcinogenic
g) Reproductivity	Based on available data, the classification criteria are not met.
h) STOT - Single Exposure	Based on available data, the classification criteria are not met.

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i) STOT - Repeated Exposure	Based on available data, the classification criteria are not met.
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may produce toxic effects. The material is not classified as causing respiratory irritation under Regulation (EC) No 1272/2008 (CLP), as amended. Nevertheless, inhalation of vapours, fumes or aerosols, particularly following prolonged exposure, may cause respiratory discomfort and, occasionally, respiratory distress.
Ingestion	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. 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. 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.
Skin Contact	The material is not classified as a skin irritant under Regulation (EC) No 1272/2008 (CLP), as amended. However, temporary discomfort may result from prolonged dermal exposure. Good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.
Eye	Although the liquid is not classified as an eye irritant under Regulation (EC) No 1272/2008 (CLP), as amended, direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).
Chronic	On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals. 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. Prolonged inhalation of arsenical dusts are implicated in lung cancer. 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 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 Workers exposed to chlorophenoxy herbicides show a significant increase in soft-tissue sarcoma, malignant lymphomas and bronchial carcinomas. Prolonged or repeated contact with solutions may result in non-allergic dermatoses. Until recently, most epidemiological studies of the effects of chlorophenoxy herbicides dealt with populations exposed in the 1950s and 1960s, when the trichlorophenol-based herbicides 2,4,5-T and fenoprop were contaminated with polychlorinated dioxins and furans, including 2,3,7,8-tetrachlorodibenzodioxin (TCDD); the effects observed may therefore have been a consequence of the presence of the dioxin contaminants. In addition, most epidemiological studies on chlorophenoxy herbicides conducted to date have involved multiple exposures to chemical agents, including other pesticides and synthetic organic compounds. In a series of case–referent studies conducted in Sweden in the late 1970s and early 1980s, strong associations were noted between soft tissue sarcomas (STS) and multiple lymphomas (including Hodgkin disease (HD) and non-

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	Hodgkin lymphoma (NHL)) and the use of chlorophenoxy herbicides by agricultural or forestry workers. The association between STS and chlorophenoxy herbicide use observed in the Swedish studies has not been confirmed in other case–referent studies. Although a number of cohort studies of occupationally exposed workers have been conducted, the small size of many of them limits their usefulness in assessing the relationship between STS and the herbicides. The risk for malignant lymphoma (HD + NHL) was almost five times greater for agricultural and forestry workers exposed to a mixture of chlorophenoxy herbicides than for controls in the case–referent study in Sweden but was not significantly elevated in a Danish cohort study of 3390 workers in a chemical plant manufacturing MCPA, dichlorprop, mecoprop, and 2,4-D, as well as other industrial chemicals and dyes Chronic exposure to 2,4-dichlorophenoxyacetic acid(2,4-D), its salts and its esters and its analogues may result in nausea, liver function changes, contact toxic dermatitis, irritation of the airways and eyes, as well as neurological changes. Persons with chronic diseases of the central nervous system, liver, heart, kidneys, lungs and skin, as well as those with endocrinological or immunological disturbances should not be exposed to herbicides (ILO Encyclopaedia). Groups of rats receiving 2,4-D in their diets for 13 weeks showed growth retardation and decreased food intake at 150 mg/kg/day dosage and an increased serum glutamic pyruvic transaminase (SGPT). A statistically significant incidence of astrocytoma was seen in the brains of male rats receiving 45 mg/kg/day for 104 weeks suggesting a possible carcinogenic effect although the prevalence of naturally occurring tumours in controls makes this result equivocal. A controversial study implicating 2,4-D as the cause of non-Hodgkin's lymphoma among male Kansas residents, aged 21 years or older, was difficult to evaluate because of a number of confounding factors. Agent Orange, a mixture of 2,4-D and 2,4,5-T, with contamination from 2,3,7,8-tetrachlorodibenzo-p-dioxin (also referred to as "dioxin" or TCDD) has been studied due to exposure of military personnel during its use as a herbicide in Vietnam. Neurological, reproductive and carcinogenic effects, purported to have occurred amongst veterans may be related to 2,4-D and 2,4,5-T but given the toxicity of the other components this remains the subject of conjecture. Most, if not all, occupational illnesses associated with 2,4,5-trichlorophenoxyacetic acids (2,4,5,-T) and its derivatives actually result from TCDD contamination. Repeated overexposure to phenoxy herbicides may cause liver, kidney, gastrointestinal and muscular effects. Subchronic exposure by dogs to phenoxy herbicides produced a reduction in circulating lymphocytes Teratogenic response was exhibited in mice (but not rats). Cleft palate was demonstrated. No such findings occurred in non-human primates given up to 10 mg/kg/day (containing 0.05 ppm TCDD) from gestation day 22 to 38. The no-observed effect level (NOAEL) in hamsters was 2 mg/kg 2,4,5-T

Apparent Barefoot Selective Herbicide	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
disodium methylarsonate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 10000 mg/kg[2]	Not Available
	Inhalation (Rat) LC50: >22.1 mg/L4h[2]	
	Oral (Rat) LD50: 821 mg/kg[2]	
MCPA sodium salt	**TOXICITY**	**IRRITATION**
	Oral (Mouse) LD50; 450 mg/kg[2]	Not Available

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

DISODIUM METHYLARSONATE	Toxicity Class: WHO III; EPA III ADI: 0.0005 mg/kg/day NOEL: 0.5 mg/kg/day 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 on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin. 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]***
MCPA SODIUM SALT	Spasticity, diarrhoea, haemorrhage, effects on fertility recorded. Animal Metabolism – MCPA is rapidly absorbed and eliminated from mammalian systems. Rats eliminated nearly all of a single oral dose within 24 hours, mostly in urine with little or no metabolism. In another rat study, three quarters of the dose was eliminated within two days. All was gone the by the eighth day. Humans excreted about half of a 5 mg dose in the urine within a few days. No residues were found after day five. Cattle and sheep fed MCPA in low to moderate doses in the diet for two weeks had no residues from levels less than about 18 mg/kg. The major metabolite of MCPA is 2-methyl-4-chlorophenol in the free and conjugated form, which is formed in the liver
Apparent Barefoot Selective Herbicide & DISODIUM METHYLARSONATE	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. 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. 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

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	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. Within 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 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 carcinogenetic 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.
Apparent Barefoot Selective Herbicide & MCPA SODIUM SALT	For chlorophenoxy pesticides: **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. Side-reactions during manufacture of the parent compound may result in the production of trace amounts of polyhalogenated aromatic hydrocarbon(s). Halogenated phenols, and especially their alkali salts, can condense above 300 deg. C . to form polyphenoxyphenols or, in a very specific reaction, to form dibenzo-p-dioxins Polyhalogenated aromatic hydrocarbons (PHAHs) comprise two major groups. The first group represented by the halogenated derivatives of dibenzodioxins (the chlorinated form is PCDD), dibenzofurans (PCDF) and biphenyls (PCB) exert their toxic effect (as hepatoxicants, reproductive toxicants, immunotoxicants and procarcinogens) by interaction with a cytostolic protein known as the Ah receptor. In guinea pigs the Ah receptor is active in a mechanism which "pumps" PHAH into the cell whilst in humans the reverse appears to true. This, in part, may account for species differences often cited in the literature. This receptor exhibits an affinity for the planar members of this group and carries these to the cellular nucleus where they bind, reversibly, to specific genomes on DNA. This results in the regulation of the production of certain proteins which elicit the toxic response. The potency of the effect is dependent on the strength of the original interaction with the Ah receptor and is influenced by the degree of substitution by the halogen and the position of such substitutions on the parent compound. The most potent molecule is 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) while the coplanar PCBs (including mono-ortho coplanars) possess approximately 1% of this potency. Nevertheless, all are said to exhibit "dioxin-like" behaviour and in environmental and health assessments it has been the practice to assign each a TCDD-equivalence value. The most subtle and important biological effects of the PHAHs are the effects on endocrine hormones and vitamin homeostasis. TCDD mimics the effect of thyroxin (a key metamorphosis signal during maturation) and may disrupt patterns of embryonic development at critical stages. Individuals from exposed wildlife populations have been observed to have altered sexual development, sexual dysfunction as adults and immune system suppression. Immunotoxic effects of the PHAHs (including the brominated congener, PBB) have been the subject of several studies. No clear pattern emerges in human studies however with T-cell numbers and function (a blood marker for immunological response) increasing in some and decreasing in others. Developmental toxicity (e.g. cleft palate, hydronephrosis) occurs in relatively few species; functional alterations following TCDD exposure leads to deficits in cognitive functions in monkeys and to adverse effects in the male reproductive system of rats. Three incidences have occurred which have introduced abnormally high levels of dioxin or dioxin-like congeners to humans. The explosion at a trichlorophenol-manufacturing plant in Seveso, Italy distributed TCDD across a large area of the country-side, whilst rice-oil contaminated with heat-transfer PCBs (and dioxin-like contaminants) has been consumed by two groups, on separate occasions (one in Yusho, Japan and another in Yu-cheng, Taiwan). The only symptom which can unequivocally be related to all these exposures is the development of chloracne, a disfiguring skin condition, following each incident. Contaminated oil poisonings also produced eye-discharge, swelling of eyelids and visual disturbances. The Babies born up to 3 years after maternal exposure (so-called "Yusho-babies") were characteristically brown skinned, coloured gums and nails and (frequently) produced eye-discharges. Delays in intellectual development have been noted. It has been estimated that Yu-cheng patients consumed an average level of 0.06 mg/kg body weight/day total PCB and 0.0002 mg/kg/day of PCDF before the onset of symptoms after 3 months. When the oil was withdrawn after 6 months they had consumed 1 gm total PCB containing 3.8 mg PCDF. Taiwanese patients consumed 10 times as much contaminated oil as the Japanese patients (because of later withdrawal); however since PCB/PCDF concentration in the Japanese oil was 10 times that consumed in Taiwan, patients from both countries consumed about the same amount of PCBs/PCDFs. Preliminary data from the Yusho cohort suggests a six-fold excess of liver cancer mortality in males and a three-fold excess in women. Recent findings from Seveso indicate that the biological effects of low level exposure (BELLEs), experienced by a cohort located at a great distance from the plant, may be hormetic, i.e. may be protective AGAINST the development of cancer. The PHAHs do not appear to be genotoxic - they do not alter the integrity of DNA. This contrasts with the effects of the many polycyclic aromatic hydrocarbons (PAHs) (or more properly, their reactive metabolites). TCDD induces carcinogenic effects in the laboratory in all species, strains and sexes tested. These effects are dose-related and occur in many organs. Exposures as low as 0.001 ug/kg body weight/day produce carcinoma. Several studies implicate PCBs in the development of liver cancer in workers as well as multi-site cancers in animals. The second major group of

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PHAH consists of the non-planar PCB congeners which possess two or more ortho-substituted halogens. These have been shown to produce neurotoxic effects which are thought to reduce the concentration of the brain neurotransmitter, dopamine, by inhibiting certain enzyme-mediated processes. The specific effect elicited by both classes of PHAH seems to depend on the as much on the developmental status of the organism at the time of the exposure as on the level of exposure over a lifetime.

NOTE: Some jurisdictions require that health surveillance be conducted on workers occupationally exposed to polycyclic aromatic hydrocarbons. Such surveillance should emphasise
- demography, occupational and medical history
- health advice, including recognition of photosensitivity and skin changes
- physical examination if indicated
- records of personal exposure including photosensitivity

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

Apparent Barefoot Selective Herbicide

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

disodium methylarsonate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>1839.4mg/L	4
EC50	48h	Crustacea	77.5mg/l	Not Available
EC50(ECx)	48h	Crustacea	77.5mg/l	Not Available
EC50	96h	Algae or other aquatic plants	12.4mg/L	4
LC50	96h	Fish	>51mg/l	Not Available

MCPA sodium salt

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	529.1mg/l	4
EC50	48h	Crustacea	>830mg/L	4
NOEC(ECx)	240h	Fish	1mg/l	4
LC50	96h	Fish	25mg/L	4

Legend: ***Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. US EPA, Ecotox database - Aquatic Toxicity Data 4. ECETOC Aquatic Hazard Assessment Data 5. NITE (Japan) - Bioconcentration Data 6. METI (Japan) - Bioconcentration Data 7. Vendor Data 8. User defined data***

Toxic 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.
Toxic to bees.
For organic arsenicals (as characterised by the herbicidals monosodium methanearsonate (MSMA), disodium methanearsonate (DSMA), calcium acid methanearsonate (CAMA) and dimethylarsinic acid (cacodylic acid))
The toxic effects of arsenicals are significantly modified by numerous biological and abiotic factors. The toxicity of arsenic in the environment is affected by temperature, pH, Eh, organic content, phosphate concentration, adsorption to solid matrices, the presence of other substances and toxicants, duration of exposure and the arsenic species present. In general, inorganic arsenicals are more toxic than organoarsenicals to biota, and trivalent species are more toxic than pentavalent species for both inorganic and organic arsenic compounds
Environmental fate:
Unlike other pesticides that degrade over time, MSMA, DSMA, CAMA, and cacodylic acid contain the element arsenic which does not degrade. Arsenic can, however, transform (i.e., change forms) or be redistributed through runoff, leaching, erosion, volatilisation, or plant uptake. The extent and speed of transformation and redistribution of the organic arsenical herbicides in soil is highly variable and depends mostly on localized environmental conditions. Thus, persistence of applied organic arsenical herbicides can range from days to years, depending on soil properties and ambient conditions such as soil moisture, temperature, chemical concentration, bacterial population, and amount of organic matter.
Although the environmental fate of the organic herbicides is highly variable depending on localized environmental conditions, environmental fate laboratory studies show that organic arsenicals are stable under all tested abiotic conditions; they do not degrade by hydrolysis or by aquatic or soil photolysis. Metabolism rates do not appear to depend linearly on arsenical concentration; the kinetics are therefore not necessarily first-order and so “half-life” may not be an appropriate constant for all concentrations. Despite the uncertainty, first-order half-lives have been calculated for modeling purposes. The estimated half-lives used in the risk assessments may underestimate the faster initial rate of metabolism but adequately portray the overall transformation and so are assumed to be protective for chronic exposure, a major concern for arsenicals.
The modeled aerobic soil half-life for MMA is 240 days; no anaerobic soil half-life was determined. The modeled aerobic soil half-life for DMA is 173 +/- 115 days with a standard upper 90% confidence limit on the mean of 240 days. The anaerobic soil half-life for DMA was calculated to be 128 +/- 38 days with a standard upper 90% confidence limit on the mean of 168 days. The effects of environmental factors on the rate of arsenical metabolism are complex and poorly defined, with different studies leading to conflicting results. An increase in temperature leads to increased metabolism. The observed influences of soil organic matter or applied arsenical concentrations are contradictory. The effect of aerobic versus anaerobic conditions on metabolism rates is also ambiguous.
Potential metabolites of applied organic arsenicals include volatile alkylarsines and inorganic arsenic (as arsenate or arsenite) along with carbon dioxide. Additionally, DMA may be present as a metabolite of MMA as well as applied directly. As with the rate, the metabolism pathway is sensitive to environmental conditions in indeterminate ways with the major metabolites occurring in widely variable proportions. Transformation to volatile alkylarsines, the only metabolism route that would directly reduce soil arsenic loading, has been shown to be possible in certain circumstances but is generally not expected to be a major route of dissipation. Observed metabolism of MMA and DMA to inorganic arsenic has ranged from undetected after several years to more than 80% transformation in several months. Generally, arsenate [As(V)] is expected to be the dominant species of inorganic arsenic, but in reducing conditions, arsenite [As(III)] may be more stable.

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Some of the variability in metabolism processes is associated with variability in sorption, because microbial transformation is only likely to occur while compounds remain dissolved in pore water. Mobility of arsenicals is typically very low to intermediate and appears to be independent of organic matter content. Instead, sorption is higher in soils with higher percentage of clay or with more iron or aluminum content.
Arsenical pesticides and their metabolites may be transported to surface waters and sediments through runoff water, eroding soils, or drift during application. These routes of exposure are likely to lead to elevations above background arsenic levels in surface water bodies.
The relative immobility of arsenicals along with arsenic s elemental nature make buildup in soil after repeated applications an important consideration. Controlled field studies, monitoring targeted to pesticide use areas, and soil modeling results all indicate that soil buildup is a likely result of long term organic arsenical application. Arsenic accumulation is likely to be limited to the top layers of soil, with studies suggesting that it is unlikely to occur at depths greater than 30 cm.
Speciation of arsenic is an important consideration in the fate, movement, and action of this substance. Chemical and biochemical transformations of arsenic include oxidation, reduction and methylation which affects its volatilisation, adsorption, dissolution and biological disposition. The transport of arsenic in the environment is largely controlled by absorption/desorption processes in soils and sediments. Sediment movement is responsible for transport of arsenic soil residues to their ultimate sinks in deep ocean sediments. The clay fraction, plus ferrous and aluminium oxides which coat clay particles, adsorb arsenicals which then undergo transformation as discussed earlier. Conversions of arsenic to volatile alkylarsines leads to air transport losses from soil. Inorganic arsenic occurs in water in different oxidation states depending on the pH and Eh of the water. Arsenate is apparently reduced by bacteria to arsenite in marine environments because the ratio of total arsenate to total arsenic is much lower than that predicted thermodynamically. Methylation of arsenic occurs in both freshwater and marine systems where arsenate occurs as arsenate, arsenite, methanearsonic acid and dimethylarsinic acid. Arsenate predominates because this is the most stable form.
Bioaccumulation occurs in some aquatic species such as seaweeds, freshwater algae and crustaceans. Some arsenic in water flea (Daphnia Magna) and algae occurs as arseno-analogues of phospholipids leading to the mistaken impression that accumulation and utilisation of arsenic takes place at the expense of phosphate. Crabs, lobsters and other marine organisms accumulate organo-arsenicals in the food chain. Although human activity may alter the local picture of environmental arsenic there is little evidence that this affects the global scale arsenic cycle.
Airborne concentrations of arsenic in urban areas may range from a few nanograms to a few tenths of a microgram per cubic meter; airborne arsenic is generally inorganic. In oxygenated soils inorganic arsenic is present in pentavalent form; under reducing conditions it occurs as the trivalent form.
Drinking Water Standards:
arsenic: 50 ug/1 (UK max.) 0.01mg/L (WHO provisional guideline)
chloride:400 mg/l (UK max.)
250 mg/l (WHO guideline)
Soil Guideline:
Dutch Criteria: 29 mg/kg (target)
55 mg/kg (intervention)
Air Quality Standard:
No safe levels recommended due to carcinogenic properties (WHO guideline)

The group of acidic herbicides, including the phenoxy acids, possess functional groups that ionise in aqueous systems yielding pKa values of less than 4. The behaviour of these materials is closely correlated with their acid character. The most significant factor with respect to soil mobility is the organic content of the soil which readily absorbs these compounds. Furthermore in acidic systems these compounds are also absorbed by clay particles. The esters and ethers are expected to behave differently from the acid forms although hydrolysis may influence subsequent binding. In general the esters and ethers are considered non-persistent in the environment.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
disodium methylarsonate	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
disodium methylarsonate	LOW (LogKOW = -5.34)
MCPA sodium salt	LOW (LogKOW = -1.29)

Mobility in soil

Ingredient	Mobility
disodium methylarsonate	LOW (Log KOC = 48.64)

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. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

Continued...

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	2X

Land transport (ADG)

14.1. **UN number or ID number**	2902
14.2. **UN proper shipping name**	PESTICIDE, LIQUID, TOXIC, N.O.S. (contains disodium methylarsonate)
14.3. **Transport hazard class(es)**	Class: 6.1 Subsidiary Hazard: Not Applicable
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: 61 223 274 Limited quantity: 5 L

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	2902
14.2. **UN proper shipping name**	Pesticide, liquid, toxic, n.o.s. * (contains disodium methylarsonate)
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 6.1 ICAO / IATA Subsidiary Hazard: Not Applicable ERG Code: 6L
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: A3 A4 Cargo Only Packing Instructions: 663 Cargo Only Maximum Qty / Pack: 220 L Passenger and Cargo Packing Instructions: 655 Passenger and Cargo Maximum Qty / Pack: 60 L Passenger and Cargo Limited Quantity Packing Instructions: Y642 Passenger and Cargo Limited Maximum Qty / Pack: 2 L

Sea transport (IMDG-Code / GGVSee)

14.1. **UN number**	2902
14.2. **UN proper shipping name**	PESTICIDE, LIQUID, TOXIC, N.O.S. (contains disodium methylarsonate)
14.3. **Transport hazard class(es)**	IMDG Class: 6.1 IMDG Subsidiary Hazard: Not Applicable
14.4. **Packing group**	III
14.5 **Environmental hazard**	Marine Pollutant
14.6. **Special precautions for user**	EMS Number: F-A, S-A Special provisions: 61 223 274 Limited Quantities: 5 L

14.7. Maritime transport in bulk according to IMO instruments

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

Not Applicable

Continued...

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

Product name	Group
disodium methylarsonate	Not Applicable
MCPA sodium salt	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
disodium methylarsonate	Not Applicable
MCPA sodium salt	Not Applicable

SECTION 15 Regulatory information

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

disodium 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 - Not Classified as Carcinogenic

MCPA sodium salt 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

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (disodium methylarsonate; MCPA sodium salt)
Canada - NDSL	No (disodium methylarsonate)
China - IECSC	Yes
Colombia - Colombia National Industrial Chemicals Inventory	No (disodium methylarsonate; MCPA sodium salt)
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	No (disodium methylarsonate; MCPA sodium salt)
New Zealand - NZIoC	No (disodium methylarsonate)
Philippines - PICCS	No (disodium methylarsonate; MCPA sodium salt)
USA - TSCA	TSCA Inventory 'Active' substance(s) (MCPA sodium salt); No (disodium methylarsonate)
Taiwan - TCSI	No (MCPA sodium salt)
Mexico - INSQ	No (disodium methylarsonate; MCPA sodium salt)
Vietnam - NCI	No (disodium methylarsonate)
Russia - FBEPH	No (disodium methylarsonate; MCPA sodium salt)
UAE - Control List (Banned/Restricted Substances)	No (MCPA sodium salt)
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	12/11/2026
Initial Date	12/11/2026

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	12/11/2026	Physical and chemical properties - Appearance, Name

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.

Continued...

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
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration
- MARPOL: International Convention for the Prevention of Pollution from Ships
- IMSBC: International Maritime Solid Bulk Cargoes Code
- IGC: International Gas Carrier Code
- IBC: International Bulk Chemical Code

- 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
- 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

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end of SDS