AC Dissaray

AXICHEM Pty Ltd

Chemwatch Hazard Alert Code: **3**

Chemwatch: **20-8929**
Version No: **3.1.1.1**
Safety Data Sheet according to WHS and ADG requirements

Issue Date: **04/08/2015**
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L.GHS.AUS.EN

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

Product Identifier

Product name	AC Dissaray
Synonyms	Not Available
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 certain broadleaf weeds in winter cereals, pastures, turf and non-crop areas.

Details of the supplier of the safety data sheet

Registered company name	AXICHEM Pty Ltd
Address	18 Conquest Way Wangara WA 6065 Australia
Telephone	+61 8 9302 4666
Fax	Not Available
Website	www.axichem.com.au
Email	msds@axichem.com.au

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	1800 039 008 (all hours)
Other emergency telephone numbers	Not Available

CHEMWATCH EMERGENCY RESPONSE

Primary Number	Alternative Number 1	Alternative Number 2
1800 039 008	1800 039 008	+612 9186 1132

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

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

Poisons Schedule	S5
Classification [1]	Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Serious Eye Damage Category 1
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI*

Label elements

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Hazard pictogram(s)	

SIGNAL WORD	**DANGER**

Hazard statement(s)

H302	Harmful if swallowed.
H315	Causes skin irritation.
H318	Causes serious eye damage.

Precautionary statement(s) Prevention

P280	Wear protective gloves/protective clothing/eye protection/face protection.
P270	Do not eat, drink or smoke when using this product.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P310	Immediately call a POISON CENTER or doctor/physician.
P362	Take off contaminated clothing and wash before reuse.
P301+P312	IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P330	Rinse mouth.
P332+P313	If skin irritation occurs: Get medical advice/attention.

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

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

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
2039-46-5	30	MCPA, dimethylamine salt
		(340 g/L)
2300-66-5	7	dicamba, dimethylamine salt
		(80 g/L)
7732-18-5	>60	water

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.

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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, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary.
Ingestion	▸ For advice, contact a Poisons Information Centre or a doctor at once. ▸ Urgent hospital treatment is likely to be needed. ▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Transport to hospital or doctor without delay.

Indication of any immediate medical attention and special treatment needed

Following 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

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known

Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves in the event of a fire. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ 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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Fire/Explosion Hazard	▸ 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. Decomposition may produce toxic fumes of: , carbon dioxide (CO_2) , hydrogen chloride , phosgene , nitrogen oxides (NOx)
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	▸ 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	Moderate hazard. ▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves. ▸ 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	▸ 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. ▸ **DO NOT** allow clothing wet with material to stay in contact with skin
Other information	▸ Store in original containers. ▸ Keep containers securely sealed.

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	▸ 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	▸ Polyethylene or polypropylene container. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Avoid contamination of water, foodstuffs, feed or seed.

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
AC Dissaray	Not Available	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
MCPA, dimethylamine salt	Not Available	Not Available
dicamba, dimethylamine salt	Not Available	Not Available
water	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. Local exhaust ventilation may be required in specific circumstances. If risk of overexposure exists, wear 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

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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.
Personal protection	
Eye and face protection	▸ 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	▸ 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.
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ P.V.C. apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.
Thermal hazards	Not Available

Recommended material(s)

GLOVE SELECTION INDEX

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

Material	CPI
BUTYL	C
NATURAL RUBBER	C
NEOPRENE	C
PVA	C
VITON	C

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

Respiratory protection

Particulate. (AS/NZS 1716 & 1715, EN 143:2000 & 149:001, ANSI Z88 or national equivalent)

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	-AUS / Class1 P2	-
up to 50	1000	-	-AUS / Class 1 P2
up to 50	5000	Airline *	-
up to 100	5000	-	-2 P2
up to 100	10000	-	-3 P2
100+			Airline**

* - Continuous Flow ** - Continuous-flow or positive pressure demand
A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid

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long-term or frequent use. A qualified practitioner should be consulted.

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)

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Amber liquid with mild amine odour; mixes with water.

Physical state	Liquid	**Relative density (Water = 1)**	1.128
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**	>100
Melting point / freezing point (°C)	<0	**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)**	~50
Vapour pressure (kPa)	Not Applicable	**Gas group**	Not Available
Solubility in water (g/L)	Miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

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

Inhaled	Not normally a hazard due to non-volatile nature of product
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. Chlorophenoxy compounds may cause irritation of the mouth, throat, and gastrointestinal tract, nausea, vomiting, chest and abdominal pain, and diarrhea. Ingestion of very large doses may produce metabolic acidosis, fever or subnormal temperature, hyperventilation, hypotension, vasodilation, flushing, sweating, cardiac arrhythmias, tachycardia, lethargy, weakness, intercostal paralysis, renal and hepatic disorders, myotonia, coma, and convulsions. Skeletal muscle damage may produce muscle twitching, aching and elevated serum enzymes and myoglobin in both blood and urine. Circulatory

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	collapse may be fatal. Acute exposure to 2,4-dichlorophenoxyacetic acid (2,4-D) and its derivatives and analogues may produce headache, dizziness, nausea, vomiting, raised temperature, low blood pressure, leucocytotoxic heart and liver injury and convulsions. All animal species tested seem to react similarly and there is only a minor difference in potency between various salts and esters of 2,4-D either as pure chemicals or as commercial preparations although the free acid exhibits a somewhat higher toxicity. In several species systemic intoxication after massive doses produces ventricular fibrillation or, if death is delayed, motor disturbances. A disinclination to move progresses to rigidity of skeletal muscles (myotonia) and ataxia (involuntary muscle movement). Severe cases show progressive apathy, depression, muscle weakness of the hind limbs, periodic clonic spasms and coma. Subacute poisonings are characterised by anorexia, eye and nose irritation, and possible epistaxis or bleeding from the mouth. Clinical reports of poisonings are rare although protracted peripheral neuropathies with myopathy appear to be characteristic. Significant cumulative toxicity does not occur with 2,4-D and most of its congeners are not metabolised and do not accumulate in body fat or in the food chain. Urinary excretion is slow with a plasma half-life of about 33 hours.
Skin Contact	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. Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. Corneal injury resulting from 2,4-D exposure may be slow to heal.
Chronic	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. 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-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

AC Dissaray	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

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MCPA, dimethylamine salt	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Not Available
	Oral (rat) LD50: 1200 mg/kg[2]	
dicamba, dimethylamine salt	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Not Available
	Inhalation (rat) LC50: >200 mg/l/4h*[2]	
	Oral (rat) LD50: 2629 mg/kg[2]	
water	**TOXICITY**	**IRRITATION**
	Not Available	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*	

MCPA, DIMETHYLAMINE 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

Continued...

	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 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 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 Data for 52% aqueous solution:
DICAMBA, DIMETHYLAMINE SALT	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. for dicamba: Dicamba is moderately toxic by ingestion and slightly toxic by inhalation or dermal exposure. Symptoms of poisoning with dicamba include loss of appetite (anorexia), vomiting, muscle weakness, slowed heart rate, shortness of breath, central nervous system effects (victim may become excited or depressed), benzoic acid in the urine, incontinence, cyanosis (bluing of the skin and gums), and exhaustion following repeated muscle spasms. In addition to these symptoms, inhalation can cause irritation of the linings of the nasal passages and the lungs, and loss of voice. Most individuals who have survived severe poisoning from dicamba have recovered within 2 to 3 days with no permanent effects. Dicamba is very irritating and corrosive and can cause severe and permanent damage to the eyes. In some individuals, dicamba is a skin sensitiser. It may cause skin burns. There is no evidence that dicamba is absorbed into the body through the skin. **Reproductive Effects:** In a 3-generation study, dicamba did not affect the reproductive capacity of rats When rabbits were given doses of 0, 0.5, 1, 3, 10 or 20 mg/kg/day of technical dicamba from days 6 through 18 of pregnancy, toxic effects on the mothers, slightly reduced fetal body weights, and increased loss of fetuses occurred at the 10 mg/kg dose. EPA has set the NOAEL for this study at 3 mg/kg/day.**Teratogenic Effects:** Dicamba is suspected of being a human teratogen. No teratogenic effects have been shown in lab animals such as rabbits and rats. **Mutagenic Effects:** Dicamba has not been shown to be a mutagen. **Carcinogenic Effects:** Data from laboratory studies are inadequate for EPA to determine if dicamba can increase the risk of cancer in humans. Rats fed up to 25 mg dicamba/kg/day for 2 years showed no increased incidence of tumors. **Organ Toxicity:** In mice, some enlargement of liver cells has occurred. A similar effect has not been shown in man. **Fate in Humans and Animals:** Dicamba was excreted rapidly by rats, mainly in the urine, when administered orally or subcutaneously. One to 4% was excreted in the faeces. Mice, rats, rabbits and dogs excreted 85% of an oral dose as unmetabolised dicamba in the urine within 48 hours of dosing. Eventually, between 90 and 99% of the dose was excreted unmetabolised in the urine. This indicates that dicamba is rapidly absorbed into the bloodstream from the gastrointestinal tract. Like most organic acids, dicamba is joined to glycine, or glucuronic acid in the liver. When dicamba was ingested daily in the feed, the concentrations in different organs reached a steady state within 2 weeks. When daily intake stopped, storage in the organs declined rapidly. It is therefore concluded that dicamba does not bioaccumulate in mammalian tissues. Following an attempted suicide with a mixture of dicamba and 2,4-D, dicamba levels in the blood serum and urine of the victim became undetectable within 2 weeks [* Sandoz]
WATER	No significant acute toxicological data identified in literature search.

Acute Toxicity	✔	**Carcinogenicity**	⊘
Skin Irritation/Corrosion	✔	**Reproductivity**	⊘
Serious Eye Damage/Irritation	✔	**STOT - Single Exposure**	⊘
Respiratory or Skin sensitisation	⊘	**STOT - Repeated Exposure**	⊘

Continued...

Mutagenicity	⊘	**Aspiration Hazard**	⊘

Legend: ✗ *– Data available but does not fill the criteria for classification*
✓ *– Data available to make classification*
⊘ *– Data Not Available to make classification*

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

AC Dissaray

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

MCPA, dimethylamine salt

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	>10mg/L	4
EC50	96	Algae or other aquatic plants	71mg/L	4

dicamba, dimethylamine salt

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	>1000mg/L	4
EC50	48	Crustacea	1600mg/L	4

water

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

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*

DO NOT discharge into sewer or waterways.

Persistence and degradability

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

Bioaccumulative potential

Ingredient	Bioaccumulation
water	LOW (LogKOW = -1.38)

Mobility in soil

Ingredient	Mobility
water	LOW (KOC = 14.3)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal

- Recycle wherever possible.
- Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified.
- Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible material).
- Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.
- 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.

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SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

SECTION 15 REGULATORY INFORMATION

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

MCPA, DIMETHYLAMINE SALT(2039-46-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists | Australia Inventory of Chemical Substances (AICS)

DICAMBA, DIMETHYLAMINE SALT(2300-66-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists | Australia Inventory of Chemical Substances (AICS)

WATER(7732-18-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

National Inventory	Status
Australia - AICS	Y
Canada - DSL	N (dicamba, dimethylamine salt; MCPA, dimethylamine salt)
Canada - NDSL	N (water; dicamba, dimethylamine salt; MCPA, dimethylamine salt)
China - IECSC	N (dicamba, dimethylamine salt; MCPA, dimethylamine salt)
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	N (MCPA, dimethylamine salt)
Korea - KECI	N (dicamba, dimethylamine salt; MCPA, dimethylamine salt)
New Zealand - NZIoC	Y
Philippines - PICCS	N (dicamba, dimethylamine salt; MCPA, dimethylamine salt)
USA - TSCA	N (dicamba, dimethylamine salt; MCPA, dimethylamine salt)
Legend:	*Y = All ingredients are on the inventory* *N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 OTHER INFORMATION

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。

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IDLH: Immediately Dangerous to Life or Health Concentrations
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index