

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

Product Identifier

Product name	Saxon®
Chemical Name	Not Applicable
Synonyms	ACVM approval: P008401
Proper shipping name	CORROSIVE LIQUID, N.O.S. (contains MCPA and mecoprop-P-potassium)
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	Herbicide Use according to manufacturer's directions.
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Details of the manufacturer or importer of the safety data sheet

Registered company name	Arxada NZ Limited
Address	13-15 Hudson Road Bell Block New Plymouth 4312 New Zealand
Telephone	+64 6 755 9234
Fax	+64 6 755 1174
Website	www.arxada.co.nz
Email	office-newplymouth@arxada.com

Emergency telephone number

Association / Organisation	Arxada NZ Limited
Emergency telephone number(s)	0800 243 622
Other emergency telephone number(s)	+64 4 917 9888 (International)

SECTION 2 Hazards identification

Classification of the substance or mixture

Classification ^[1]	Acute Toxicity (Oral) Category 4, Aspiration Hazard Category 1, Skin Corrosion/Irritation Category 1B, Serious Eye Damage/Eye Irritation Category 1, Carcinogenicity Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 2, Hazardous to the Aquatic Environment Acute Hazard Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
Signal word	Danger

Hazard statement(s)

H302	Harmful if swallowed.
H304	May be fatal if swallowed and enters airways.
H314	Causes severe skin burns and eye damage.
H351	Suspected of causing cancer.
H373	May cause damage to organs through prolonged or repeated exposure.
H410	Very toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P260	Do not breathe mist/vapours/spray.
P264	Wash all exposed external body areas thoroughly after handling.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P270	Do not eat, drink or smoke when using this product.
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.
P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P363	Wash contaminated clothing before reuse.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.
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Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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No further product hazard information.

SECTION 3 Composition / information on ingredients**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
1929-86-8	10-30	<u>mecoprop-P-potassium</u>
94-74-6	10-30	<u>MCPA</u>
112-34-5	10-20	<u>diethylene glycol monobutyl ether</u>
64742-94-5	10-15	<u>hydrocarbons, C10, aromatics, >1% naphthalene</u>
81406-37-3	1-10	<u>fluroxypyr methylheptyl ester</u>
141-43-5	1-10	<u>monoethanolamine</u>
37251-69-7	<5	<u>nonylphenol, ethoxylated, propoxylated</u>
1310-58-3	<1	<u>potassium hydroxide</u>
127-08-2	<1	<u>potassium acetate</u>
7732-18-5	10-20	<u>water</u>
Legend:	1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available	

SECTION 4 First aid measures**Description of first aid measures**

Eye Contact	If this product comes in contact with the eyes: ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs: ▶ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▶ Quickly remove all contaminated clothing, including footwear. ▶ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▶ Transport to hospital, or doctor.
Inhalation	▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay. ▶ Inhalation of vapours or aerosols (mists, fumes) may cause lung oedema. ▶ Corrosive substances may cause lung damage (e.g. lung oedema, fluid in the lungs).

Continued...

	- As this reaction may be delayed up to 24 hours after exposure, affected individuals need complete rest (preferably in semi-recumbent posture) and must be kept under medical observation even if no symptoms are (yet) manifested. - Before any such manifestation, the administration of a spray containing a dexamethasone derivative or beclomethasone derivative may be considered. This must definitely be left to a doctor or person authorised by him/her. (ICSC13719)
Ingestion	- For advice, contact a Poisons Information Centre or a doctor at once. - Urgent hospital treatment is likely to be needed. If swallowed do NOT induce vomiting. - If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. - 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. - Avoid giving milk or oils. - Avoid giving alcohol. - If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

Indication of any immediate medical attention and special treatment needed

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.

Treat symptomatically.

For petroleum distillates

- In case of ingestion, gastric lavage with activated charcoal can be used promptly to prevent absorption - decontamination (induced emesis or lavage) is controversial and should be considered on the merits of each individual case; of course the usual precautions of an endotracheal tube should be considered prior to lavage, to prevent aspiration.
- Individuals intoxicated by petroleum distillates should be hospitalized immediately, with acute and continuing attention to neurologic and cardiopulmonary function.
- Positive pressure ventilation may be necessary.
- Acute central nervous system signs and symptoms may result from large ingestions of aspiration-induced hypoxia.
- After the initial episode, individuals should be followed for changes in blood variables and the delayed appearance of pulmonary oedema and chemical pneumonitis. Such patients should be followed for several days or weeks for delayed effects, including bone marrow toxicity, hepatic and renal impairment. Individuals with chronic pulmonary disease will be more seriously impaired, and recovery from inhalation exposure may be complicated.
- Gastrointestinal symptoms are usually minor and pathological changes of the liver and kidneys are reported to be uncommon in acute intoxications.
- Chlorinated and non-chlorinated hydrocarbons may sensitize the heart to epinephrine and other circulating catecholamines so that arrhythmias may occur. Careful consideration of this potential adverse effect should precede administration of epinephrine or other cardiac stimulants and the selection of bronchodilators.

for naphthalene intoxication: Naphthalene requires hepatic and microsomal activation prior to the production of toxic effects. Liver microsomes catalyse the initial synthesis of the reactive 1,2-epoxide intermediate which is subsequently oxidised to naphthalene dihydrodiol and alpha-naphthol. The 2-naphthoquinones are thought to produce haemolysis, the 1,2-naphthoquinones are thought to be responsible for producing cataracts in rabbits, and the glutathione-adducts of naphthalene-1,2-oxide are probably responsible for pulmonary toxicity. Suggested treatment regime:

- Induce emesis and/or perform gastric lavage with large amounts of warm water where oral poisoning is suspected.
- Instill a saline cathartic such as magnesium or sodium sulfate in water (15 to 30g).
- Demulcents such as milk, egg white, gelatin, or other protein solutions may be useful after the stomach is emptied but oils should be avoided because they promote absorption.
- If eyes/skin contaminated, flush with warm water followed by the application of a bland ointment.
- Severe anaemia, due to haemolysis, may require small repeated blood transfusions, preferably with red cells from a non-sensitive individual.
- Where intravascular haemolysis, with haemoglobinuria occurs, protect the kidneys by promoting a brisk flow of dilute urine with, for example, an osmotic diuretic such as mannitol. It may be useful to alkalinise the urine with small amounts of sodium bicarbonate but many researchers doubt whether this prevents blockage of the renal tubules.
- Use supportive measures in the case of acute renal failure. GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products, 5th Ed.

For acute or short-term repeated exposures to highly alkaline materials:

- Respiratory stress is uncommon but present occasionally because of soft tissue edema.
- Unless endotracheal intubation can be accomplished under direct vision, cricothyroidotomy or tracheotomy may be necessary.
- Oxygen is given as indicated.
- The presence of shock suggests perforation and mandates an intravenous line and fluid administration.
- Damage due to alkaline corrosives occurs by liquefaction necrosis whereby the saponification of fats and solubilisation of proteins allow deep penetration into the tissue.

Alkalis continue to cause damage after exposure.

INGESTION:

- Milk and water are the preferred diluents

No more than 2 glasses of water should be given to an adult.

- Neutralising agents should never be given since exothermic heat reaction may compound injury.

* Catharsis and emesis are absolutely contra-indicated.

* Activated charcoal does not absorb alkali.

* Gastric lavage should not be used.

Supportive care involves the following:

- Withhold oral feedings initially.
- If endoscopy confirms transmucosal injury start steroids only within the first 48 hours.
- Carefully evaluate the amount of tissue necrosis before assessing the need for surgical intervention.
- Patients should be instructed to seek medical attention whenever they develop difficulty in swallowing (dysphagia).

SKIN AND EYE:

- Injury should be irrigated for 20-30 minutes.

Eye injuries require saline. [Ellenhorn & Barceloux: Medical Toxicology]

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. Alkalinisation 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. ▶ 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 (CO₂) hydrogen chloride phosgene nitrogen oxides (NO_x) metal oxides ▶ other pyrolysis products typical of burning organic material. May emit corrosive fumes.

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	▶ Drains for storage or use areas should have retention basins for pH adjustments and dilution of spills before discharge or disposal of material. ▶ Check regularly for spills and leaks. ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb spill with sand, earth, inert material or vermiculite. ▶ Wipe up. ▶ Place in a suitable, labelled container for waste disposal.																																																												
Major Spills	Chemical Class: bases For release onto land: recommended sorbents listed in order of priority. <table><thead><tr><th>SORBENT TYPE</th><th>RANK</th><th>APPLICATION</th><th>COLLECTION</th><th>LIMITATIONS</th></tr></thead><tbody><tr><td colspan="5">LAND SPILL - SMALL</td></tr><tr><td>cross-linked polymer - particulate</td><td>1</td><td>shovel</td><td>shovel</td><td>R,W,SS</td></tr><tr><td>cross-linked polymer - pillow</td><td>1</td><td>throw</td><td>pitchfork</td><td>R, DGC, RT</td></tr><tr><td>sorbent clay - particulate</td><td>2</td><td>shovel</td><td>shovel</td><td>R, I, P</td></tr><tr><td>foamed glass - pillow</td><td>2</td><td>throw</td><td>pitchfork</td><td>R, P, DGC, RT</td></tr><tr><td>expanded minerals - particulate</td><td>3</td><td>shovel</td><td>shovel</td><td>R, I, W, P, DGC</td></tr><tr><td>foamed glass - particulate</td><td>4</td><td>shovel</td><td>shovel</td><td>R, W, P, DGC,</td></tr><tr><td colspan="5">LAND SPILL - MEDIUM</td></tr><tr><td>cross-linked polymer -particulate</td><td>1</td><td>blower</td><td>skiploader</td><td>R,W, SS</td></tr><tr><td>sorbent clay - particulate</td><td>2</td><td>blower</td><td>skiploader</td><td>R, I, P</td></tr><tr><td>expanded mineral - particulate</td><td>3</td><td>blower</td><td>skiploader</td><td>R, I,W, P, DGC</td></tr></tbody></table>	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS	LAND SPILL - SMALL					cross-linked polymer - particulate	1	shovel	shovel	R,W,SS	cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT	sorbent clay - particulate	2	shovel	shovel	R, I, P	foamed glass - pillow	2	throw	pitchfork	R, P, DGC, RT	expanded minerals - particulate	3	shovel	shovel	R, I, W, P, DGC	foamed glass - particulate	4	shovel	shovel	R, W, P, DGC,	LAND SPILL - MEDIUM					cross-linked polymer -particulate	1	blower	skiploader	R,W, SS	sorbent clay - particulate	2	blower	skiploader	R, I, P	expanded mineral - particulate	3	blower	skiploader	R, I,W, P, DGC
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Legend

DGC: Not effective where ground cover is dense

R; Not reusable

I: Not incinerable

P: Effectiveness reduced when rainy

RT: Not effective where terrain is rugged

SS: Not for use within environmentally sensitive sites

W: Effectiveness reduced when windy

Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;

R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988

- ▶ Clear area of personnel and move upwind.
- ▶ Alert Fire Brigade and tell them location and nature of hazard.
- ▶ Wear full body protective clothing with breathing apparatus.
- ▶ Prevent, by any means available, spillage from entering drains or water course.
- ▶ Consider evacuation (or protect in place).
- ▶ Stop leak if safe to do so.
- ▶ Contain spill with sand, earth or vermiculite.
- ▶ Collect recoverable product into labelled containers for recycling.
- ▶ Neutralise/decontaminate residue (see Section 13 for specific agent).
- ▶ Collect solid residues and seal in labelled drums for disposal.
- ▶ Wash area and prevent runoff into drains.
- ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.
- ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling

- ▶ Containers, even those that have been emptied, may contain explosive vapours.
 - ▶ Do NOT cut, drill, grind, weld or perform similar operations on or near containers.
 - ▶ **DO NOT allow clothing wet with material to stay in contact with skin**
- The tendency of many ethers to form explosive peroxides is well documented. Ethers lacking non-methyl hydrogen atoms adjacent to the ether link are thought to be relatively safe
- ▶ **DO NOT concentrate by evaporation, or evaporate extracts to dryness, as residues may contain explosive peroxides with DETONATION potential.**
 - ▶ Any static discharge is also a source of hazard.
 - ▶ Before any distillation process remove trace peroxides by shaking with excess 5% aqueous ferrous sulfate solution or by percolation through a column of activated alumina.
 - ▶ Distillation results in uninhibited ether distillate with considerably increased hazard because of risk of peroxide formation on storage.
 - ▶ Add inhibitor to any distillate as required.
 - ▶ When solvents have been freed from peroxides by percolation through columns of activated alumina, the absorbed peroxides must promptly be desorbed by treatment with polar solvents such as methanol or water, which should then be disposed of safely.
- The substance accumulates peroxides which may become hazardous only if it evaporates or is distilled or otherwise treated to concentrate the peroxides. The substance may concentrate around the container opening for example.
- Purchases of peroxidisable chemicals should be restricted to ensure that the chemical is used completely before it can become peroxidised.
- ▶ A responsible person should maintain an inventory of peroxidisable chemicals or annotate the general chemical inventory to indicate which chemicals are subject to peroxidation. An expiration date should be determined. The chemical should either be treated to remove peroxides or disposed of before this date.
 - ▶ The person or laboratory receiving the chemical should record a receipt date on the bottle. The individual opening the container should add an opening date.
 - ▶ Unopened containers received from the supplier should be safe to store for 18 months.
 - ▶ Opened containers should not be stored for more than 12 months.
 - ▶ Avoid skin contact, including inhalation.
 - ▶ Wear protective clothing when risk of exposure occurs.
 - ▶ Use in a well-ventilated area.
 - ▶ Avoid contact with moisture.
 - ▶ Avoid contact with incompatible materials.
 - ▶ **When handling, DO NOT eat, drink or smoke.**
 - ▶ Keep containers securely sealed when not in use.
 - ▶ Avoid physical damage to containers.
 - ▶ Always wash hands with soap and water after handling.
 - ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use.
 - ▶ Use good occupational work practice.
 - ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
 - ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.

Other information

- Consider storage under inert gas.
- ▶ 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.
 - ▶ **DO NOT store near acids, or oxidising agents**
 - ▶ No smoking, naked lights, heat or ignition sources.

Conditions for safe storage, including any incompatibilities

Suitable container

- ▶ Glass container is suitable for laboratory quantities
 - ▶ Lined metal can, lined metal pail/ can.
 - ▶ Plastic pail.
 - ▶ Polyliner drum.
 - ▶ Packing as recommended by manufacturer.
 - ▶ Check all containers are clearly labelled and free from leaks.
- For low viscosity materials

	- Drums and jerricans must be of the non-removable head type. - Where a can is to be used as an inner package, the can must have a screwed enclosure. For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.): - Removable head packaging; - Cans with friction closures and - low pressure tubes and cartridges may be used. - Where combination packages are used, and the inner packages are of glass, porcelain or stoneware, there must be sufficient inert cushioning material in contact with inner and outer packages unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	Monoethanolamine - is a strong organic base - reacts violently with strong oxidisers, strong acids (with spattering) - is incompatible with acetic acid, acetic anhydride, acrolein, acrylates, acrylic acid, acrylonitrile, alcohols, aldehydes, alkali metals, alkylene oxides, substituted allyls, caprolactam solution, cellulose nitrate, chlorosulfonic acid, cresols, epichlorohydrin, glycols, halogenated hydrocarbons, isocyanates, ketones, mesityl oxide, oleum, organic anhydrides, phenols, beta-propiolactone, vinyl acetate - forms explosive mixture with sodium perchlorate - reacts with iron forming tris-ethanolamineiron - may undergo a self-sustaining thermal decomposition when heated in excess of 250 degrees C - attacks aluminium, copper, lead, tin, zinc, and their alloys - attacks plastics, coatings an rubber - Avoid strong acids, acid chlorides, acid anhydrides and chloroformates. - Avoid contact with copper, aluminium and their alloys. - Avoid reaction with oxidising agents



X — Must not be stored together

O — May be stored together with specific preventions

+ — May be stored together

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
New Zealand Workplace Exposure Standards (WES)	MCPA	Respirable dust (not otherwise classified)	3 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	MCPA	Inhalable dust (not otherwise classified)	10 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	monoethanolamine	2-Aminoethanol (Ethanolamine)	0.2 ppm / 0.5 mg/m3	0.5 mg/m3 / 0.2 ppm	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	potassium hydroxide	Potassium hydroxide	Not Available	Not Available	2 mg/m3	Not Available

MATERIAL DATA

Exposure controls

Appropriate engineering controls	Unless written procedures, specific to the workplace are available, the following is intended as a guide: For Laboratory-scale handling of Substances assessed to be toxic by inhalation. Quantities of up to 25 grams may be handled in Class II biological safety cabinets *; Quantities of 25 grams to 1 kilogram may be handled in Class II biological safety cabinets* or equivalent containment systems; Quantities exceeding 1 kg may be handled either using specific containment, a hood or Class II biological safety cabinet*. - HEPA terminated local exhaust ventilation should be considered at point of generation of dust, fumes or vapours. - The need for respiratory protection should also be assessed where incidental or accidental exposure is anticipated. Dependent on levels of contamination, PAPR, full face air purifying devices with P2 or P3 filters or air supplied respirators should be evaluated. When handling: Quantities of up to 25 grams, an approved respirator with HEPA filters or cartridges should be considered; Quantities of 25 grams to 1 kilogram, a half-face negative pressure, full negative pressure, or powered helmet-type air purifying respirator should be considered. Quantities in excess of 1 kilogram, a full face negative pressure, helmet-type air purifying, or supplied air respirator should be considered. Written procedures, specific to a particular work-place, may replace these recommendations * For Class II Biological Safety Cabinets, Types B2 or B3 should be considered. Where only Class I, open fronted Cabinets are available, glove panels may be added, Laminar flow cabinets do not provide sufficient protection when handling these materials unless especially designed to do so. Pilot Plant and Production - Wear appropriate gloves; lab coat, nylon coveralls or disposable Tyvek suit; safety glasses, safety shoes, and disposable booties. Use good manufacturing practices (i.e., cGMPs). - Protective garment (coveralls, Tyvek, lab coat) is not to be worn outside the work area. - Clean/dirty/decontamination areas are to be established. - Negative/positive air pressure relationships and buffer zones required (i.e., ante-room/degowning room/airlock). - Area access is to be restricted. - High-energy operations such as milling, particle sizing, spraying or fluidising should be done within an approved emission control or containment system. - Develop cleaning procedures and techniques that limit potential exposure For potent pharmacological agents:
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	Solutions Handling: - Solutions can be handled outside a containment system or without local exhaust ventilation during procedures with no potential for aerosolisation. If the procedures have a potential for aerosolisation, an air-purifying respirator is to be worn by all personnel in the immediate area. - Solutions used for procedures where aerosolisation may occur (e.g., vortexing, pumping) are to be handled within a containment system or with local exhaust ventilation. - In situations where this is not feasible (may include animal dosing), an air-purifying respirator is to be worn by all personnel in the immediate area. If using a ventilated enclosure that has not been validated, wear a half-mask respirator equipped with HEPA cartridges until the enclosure is validated for use. - Ensure gloves are protective against solvents in use.
Individual protection measures, such as personal protective equipment	
Eye and face protection	When handling very small quantities of the material eye protection may not be required. For laboratory, larger scale or bulk handling or where regular exposure in an occupational setting occurs: - Chemical goggles.[AS/NZS 1337.1, EN166 or national equivalent] - Face shield. Full face shield may be required for supplementary but never for primary protection of eyes. - 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	- Elbow length PVC gloves - When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots. NOTE: - The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. - Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. 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. - Rubber gloves (nitrile or low-protein, powder-free latex, latex/ nitrile). Employees allergic to latex gloves should use nitrile gloves in preference. - Double gloving should be considered. - PVC gloves. - Change gloves frequently and when contaminated, punctured or torn. - Wash hands immediately after removing gloves. - Protective shoe covers. [AS/NZS 2210] - Head covering.
Body protection	See Other protection below
Other protection	- Overalls. - PVC Apron. - PVC protective suit may be required if exposure severe. - Eyewash unit. - Ensure there is ready access to a safety shower.

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:

Saxon®

Material	CPI
BUTYL	A
NEOPRENE	A
BUTYL/NEOPRENE	C
HYPALON	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PVA	C
PVC	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 long-term or frequent use. A qualified practitioner should be consulted.

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

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

^ - Full-face

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

- ▶ 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

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SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	A liquid; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	1.16
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	5.5-7.5	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)	>95 C	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	There is sufficient evidence to classify this material as skin corrosive or irritating.
c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
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.
i) STOT - Repeated Exposure	There is sufficient evidence to classify this material as toxic to specific organs through repeated exposure
j) Aspiration Hazard	There is sufficient evidence to classify this material as an aspiration hazard

Inhaled

Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may produce toxic effects. Inhalation of alkaline corrosives may produce irritation of the respiratory tract with coughing, choking, pain and mucous membrane damage. Pulmonary oedema may develop in more severe cases; this may be immediate or in most cases following a latent period of 5-72 hours. Symptoms may include a tightness in the chest, dyspnoea, frothy sputum, cyanosis and dizziness. Findings may include hypotension, a weak and rapid pulse and moist rales. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.

High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations.

Inhalation of naphthalene vapour has been associated with headache, loss of appetite and nausea. Other conditions associated with exposure to the vapour include optic neuritis, corneal injury and kidney damage. Animals exposed to aerosols of a refined commercial solvent mixture consisting primarily of mono-methylated naphthalenes, exhibited dyspnoea. When animals were exposed to this mixture for 27 daily one-hour exposures over a 35-day period, they showed dyspnoea, listlessness, prostration and marked salivation. Weight loss was evident in mice but not in other species. Pathological changes occurred in the lungs, liver and skin. Pulmonary changes consisted mainly of oedema, bronchopneumonia, emphysema, and thickening of the parabronchiolar alveolar septa. Haematology did not identify significant changes.

Inhalation of chlorophenoxy pesticide dusts or mist may produce a sore throat and burning sensations in the nasopharynx region and chest, coughing, lachrymation, rhinitis, dizziness and ataxia. Toxic effects may result following absorption from the lungs. One case report describes kidney and liver damage in two people working in a closed room with paint containing diethylene glycol monobutyl ether and at the same time consuming large quantities of alcoholic beverages. It has as yet not been established whether the glycol ether and alcohol have synergistic effects but it is possible that oxidation and elimination of both substances probably involves alcohol dehydrogenases; competitive inhibition would be the result. Monoethanolamine vapours, mists and liquid are corrosive to the mouth and throat. When rats were exposed for 8 hours to a highly enriched and/or saturated atmosphere at ambient temperatures, there were no fatalities.

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. Ingestion of alkaline corrosives may produce immediate pain, and circumoral burns. Mucous membrane corrosive damage is characterised by a white appearance and soapy feel; this may then become brown, oedematous and ulcerated. Profuse salivation with an inability to swallow or speak may also result. Even where there is limited or no evidence of chemical burns, both the oesophagus and stomach may experience a burning pain; vomiting and diarrhoea may follow. The vomitus may be thick and may be slimy (mucous) and may eventually contain blood and shreds of mucosa. Epiglottal oedema may result in respiratory distress and asphyxia. Marked hypotension is symptomatic of shock; a weak and rapid pulse, shallow respiration and clammy skin may also be evident. Circulatory collapse may occur and, if uncorrected, may produce renal failure. Severe exposures may result in oesophageal or gastric perforation accompanied by mediastinitis, substernal pain, peritonitis, abdominal rigidity and fever. Although oesophageal, gastric or pyloric stricture may be evident initially, these may occur after weeks or even months and years. Death may be quick and results from asphyxia, circulatory collapse or aspiration of even minute amounts. Death may also be delayed as a result of perforation, pneumonia or the effects of stricture formation. Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result. Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis). At sufficiently high doses the material may be nephrotoxic (i.e. poisonous to the kidney). 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 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. Cyanosis, rapid breathing and heart beat, low blood pressure, muscle tenderness and unconsciousness may follow ingestion of diethylene glycol monobutyl ether. Swallowing large or repeated doses may affect kidney function. Ethanolamine is a normal intermediate in the metabolism of certain animal species producing phospholipids and choline. Major signs in rats poisoned by monoethanolamine included lachrymation, piloerection, kyphosis, unsteady gait, emaciation, pallor, red or brown discharge on perianal, periocular and perigenital fur. Gross pathological changes included discolouration of lungs, stomach, intestines and kidneys, liver to stomach adhesions, and liquid or gas-filled stomachs.
Skin Contact	The material can produce severe chemical burns following direct contact with the skin. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. Workers sensitised to naphthalene and its congeners show exfoliative dermatitis. Hypersensitivity, with positive patch tests, has been demonstrated in certain individuals. Percutaneous absorption is apparently inadequate to produce acute systemic reactions, except in new-born babies. Tests with a refined commercial liquid grade of methylnaphthalene (MN), placed under a patch for 48 hours on human skin produced slight to moderate reactions. In rabbits, a single dermal exposure to MN produced loss of appetite (anorexia). Repeated application of the refined commercial grade of MN to rabbit skin at 1-4 mg/kg/day for up to 21 days produced severe skin irritation and necrosis. Anorexia, moderate weight loss and fatalities were also recorded. Pathological changes in one animal were identified in the liver, stomach, heart, bone marrow, spleen, thyroid and thigh muscle. In another animal there was moderate hyperplasia of the bone marrow and thyroid. Photosensitisation has been recorded amongst workers exposed to naphthalene. An abnormal adverse reaction to ultraviolet (UV) and/or visible radiation results in sun-burn like responses or oedematous, vesiculated lesions or bullae. Open cuts, abraded or irritated skin should not be exposed to this material 2,4-D and its derivatives all penetrate intact skin of laboratory rats and man. Subacute application of 2,4-D esters and of the dimethylamine salt to rabbit skin produced only local irritation due probably to the carrier vehicle (oil). Percutaneous absorption has produced severe peripheral neuropathy in elderly patients exposed to spilled 2,4-D ester, fatigue, nausea, vomiting, anorexia, diarrhoea, swelling and aching of the extremities and muscle fasciculations progressing over a period of days to pain, paraesthesias, and severe limb paralysis. Disability was protracted and continued for several years. 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. There are indications that diethylene glycol monobutyl ether is absorbed through intact skin. Toxic effects only occur at very high doses. When applied to the skin ethanolamine is considerably more toxic than when administered orally. A single prolonged skin exposure may result in the absorption of harmful amounts. Rats poisoned after topical application showed sluggishness, abdominal distension, prostration and emaciation. Gross pathological changes included discoloured lungs, trachea, intestines, thymus and kidneys and liquid or gas-filled stomach and intestines. The undiluted liquid causes swelling and redness on rabbit skin, comparable to a first-degree burn. The necrosis produced is not related to its alkalinity. Following a 4-hour occluded application to rabbit skin, there was severe erythema, oedema and necrosis with subsequent ulceration and scabbing.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. Direct contact with alkaline corrosives may produce pain and burns. Oedema, destruction of the epithelium, corneal opacification and iritis may occur. In less severe cases these symptoms tend to resolve. In severe injuries the full extent of the damage may not be immediately apparent with late complications comprising a persistent oedema, vascularisation and corneal scarring, permanent opacity, staphyloma, cataract, symblepharon and loss of sight. Exposure to naphthalene and its congeners has produced cataracts in animals and workers. In one study, eight of twenty-one workers, exposed to naphthalene for 5-years, showed opacities of the lens. Corneal injury resulting from 2,4-D exposure may be slow to heal. A drop of ethanolamine in the eye causes injury slightly less than that produced by ammonia (grade 9, on a scale 1 to 10). Following instillation to rabbit eyes (0.005 ml), there was severe corneal injury with vascularisation and corneal deformation, severe iritis, and severe conjunctival irritation with necrosis
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. Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers. Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects. Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to

benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses. Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.

Hydrocarbon solvents are liquid hydrocarbon fractions derived from petroleum processing streams, containing only carbon and hydrogen atoms, with carbon numbers ranging from approximately C5-C20 and boiling between approximately 35-370 deg C. Many of the hydrocarbon solvents have complex and variable compositions with constituents of 4 types, alkanes (normal paraffins, isoparaffins, and cycloparaffins) and aromatics (primarily alkylated one- and two-ring species). Despite the compositional complexity, most hydrocarbon solvent constituents have similar toxicological properties, and the overall toxicological hazards can be characterized in generic terms. Hydrocarbon solvents can cause chemical pneumonitis if aspirated into the lung, and those that are volatile can cause acute CNS effects and/or ocular and respiratory irritation at exposure levels exceeding occupational recommendations. Otherwise, there are few toxicologically important effects. The exceptions, n-hexane and naphthalene, have unique toxicological properties

Animal studies:

No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) was observed in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of an alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human.

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

In a two-year inhalation study, groups of mice were exposed at 0, 10 or 30 ppm naphthalene, 6 hours/day, 5 days/week for 103 weeks.

Female mice showed an increase of pulmonary alveolar/bronchiolar adenomas at 30 ppm. There was no increase in the incidence of tumours in male mice. Naphthalene inhalation was associated with an increase in the incidence and severity of chronic inflammation, metaplasia of the olfactory epithelium, and hyperplasia of the respiratory epithelium in the nose, and chronic inflammation of the lungs of both sexes.

Saxon®	TOXICITY	IRRITATION
	Not Available	Not Available
mecoprop-P-potassium	TOXICITY	IRRITATION
	Inhalation (Rat) LC50: 5.6 mg/l4h ^[2]	Not Available
MCPA	Oral (Mouse) LD50: 369 mg/kg ^[2]	
	TOXICITY	IRRITATION
	dermal (rat) LD50: >1000 mg/kg ^[2]	Skin (Rodent - rabbit): 500mg - Mild
	Inhalation (Rat) LC50: 1.37 mg/L4h ^[2]	
	Oral (Rat) LD50: 700 mg/kg ^[2]	

diethylene glycol monobutyl ether	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 4120 mg/kg ^[2]	Eye (Rodent - rabbit): 20mg - Severe
	Oral (Rat) LD50: 5660 mg/kg ^[2]	Eye (Rodent - rabbit): 20mg/24H - Moderate
		Eye: adverse effect observed (irritating) ^[1]
hydrocarbons, C10, aromatics, >1% naphthalene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]
	Inhalation (Rat) LC50: >0.003 mg/L4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Skin (Rodent - rabbit): 500uL/24H - Mild
fluroxypyr methylheptyl ester	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[2]	Not Available
	Inhalation (Rat) LC50: >1 mg/L4h ^[2]	
	Oral (Rat) LD50: >5000 mg/kg ^[2]	
monoethanolamine	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 1000 mg/kg ^[2]	Eye (Rodent - rabbit): 250ug - Severe
	Inhalation(Guinea Pig) LC50; ~0.145 mg/L4h ^[2]	Eye: adverse effect observed (irreversible damage) ^[1]
	Oral (Guinea) LD50; 620 mg/kg ^[2]	Skin (Mammal - species unspecified): 5%
nonylphenol, ethoxylated, propoxylated	TOXICITY	IRRITATION
	Oral (Rat) LD50: 1990 mg/kg ^[2]	Not Available
potassium hydroxide	TOXICITY	IRRITATION
	Oral (Rat) LD50: 273 mg/kg ^[2]	Eye (Rodent - rabbit): 1mg/24H - Moderate
		Eye: adverse effect observed (irritating) ^[1]
		Skin (Human): 50mg/24H - Severe
potassium acetate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 20000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (Rat) LC50: 5.6 mg/L4h ^[2]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: 3250 mg/kg ^[2]	
water	TOXICITY	IRRITATION
	Oral (Rat) LD50: >90000 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

MECOPROP-P-POTASSIUM	Kidney changes, reproductive effects, specific developmental abnormalities (craniofacial, musculoskeletal), foetotoxicity recorded. For mecoprop (syn: 2-(2-methyl-4-chlorophenoxy)propionic acid; MCP) Acute Toxicity: Mecoprop has a low acute toxicity to test animals. Mecoprop is irritating to skin and eyes. It causes redness and swelling and can cause cloudy vision . Teratogenic Effects: Mecoprop is a teratogen in rats at moderate to high doses. Oral doses of 125 mg/kg/day in pregnant rats from days 6 to 15 of gestation caused increased intra-uterine deaths, decreased body lengths, and an increased incidence of delayed or absent bone formation in offspring. Mecoprop is not teratogenic in rabbits . Mutagenic Effects: Studies show that mecoprop may be mutagenic at very high doses. Tests of mecoprop on four strains of salmonella and on S. colelicolor showed no mutagenic effects. However, mecoprop caused an increase in sister chromatid exchange after single oral doses of 470 and 3,800 mg/kg in Chinese hamsters . Occupational exposure however did not appear to increase the frequency of sister chromatid exchanges or chromosomal aberrations in the leukocytes of workers Carcinogenic Effects: A study of people employed in the manufacture of phenoxy herbicides including mecoprop showed an association between these herbicides and cancer of soft tissues and non-Hodgkin's lymphoma . However, other data do not support this conclusion . Thus, it is not clear if occupational exposures to phenoxy herbicides can cause cancer. Organ Toxicity: Oral doses of 9 mg/kg/day to female rats and 27 mg/kg/day to male rats cause kidney damage . Fate in Humans and Animals: Mecoprop is eliminated unchanged in the urine of mammals
	MCPA WARNING : IARC - Human limited evidence. Mutation - DNA inhibition mouse for chlorphenoxy pesticides: ADI: 0.01 mg/kg/day NOEL: 1.1 mg/kg/day

	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
DIETHYLENE GLYCOL MONOBUTYL ETHER	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. For diethylene glycol monoalkyl ethers and their acetates: This category includes diethylene glycol ethyl ether (DGEE), diethylene glycol propyl ether (DGPE) diethylene glycol butyl ether (DGBE) and diethylene glycol hexyl ether (DGHE) and their acetates. Acute toxicity: There are adequate oral, inhalation and/or dermal toxicity studies on the category members. Oral LD50 values in rats for all category members are all > 3000 mg/kg bw, with values generally decreasing with increasing molecular weight. Four to eight hour acute inhalation toxicity studies were conducted for all category members except DGPE in rats at the highest vapour concentrations achievable. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 2000 mg/kg bw (DGHE) to 15000 mg/kg bw (DGEEA). Signs of acute toxicity in rodents are consistent with non-specific CNS depression typical of organic solvents in general. All category members are slightly irritating to skin and slightly to moderately irritating to eyes (with the exception of DGHE, which is highly irritating to eyes). Sensitisation tests with DGEE, DGEEA, DGPE, DGBE and DGBEA in animals and/or humans were negative. Repeat dose toxicity: Valid oral studies conducted using DGEE, DGPE, DGBEA, DGHE and the supporting chemical DGBE ranged in duration from 30 days to 2 years. Effects predominantly included kidney and liver toxicity, absolute and/or relative changes in organ weights, and some changes in haematological parameters. All effects were seen at doses greater than 800-1000 mg/kg bw/day from oral or dermal studies; no systemic effects were observed in inhalation studies with less than continuous exposure regimens. Mutagenicity: DGEE, DGEEA, DGBE, DGBEA and DGHE generally tested negative for mutagenicity in <i>S. typhimurium</i> strains TA98, TA100, TA1535, TA1537 and TA1538 and DGBEA tested negative in <i>E. coli</i> WP2uvrA, with and without metabolic activation. In vitro cytogenicity and sister chromatid exchange assays with DGBE and DGHE in Chinese Hamster Ovary Cells with and without metabolic activation and in vivo micronucleus or cytogenicity tests with DGEE, DGBE and DGHE in rats and mice were negative, indicating that these diethylene glycol ethers are not likely to be genotoxic. Reproductive and developmental toxicity: Reliable reproductive toxicity studies on DGEE, DGBE and DGHE show no effect on fertility at the highest oral doses tested (4,400 mg/kg/day for DGEE in the mouse and 1,000 mg/kg/day for DGBE and DGHE in the rat). The dermal NOAEL for reproductive toxicity in rats administered DGBE also was the highest dose tested (2,000 mg/kg/day). Although decreased sperm motility was noted in F1 mice treated with 4,400 mg/kg/day DGEE in drinking water for 14 weeks, sperm concentrations and morphology, histopathology of the testes and fertility were not affected. Results of the majority of adequate repeated dose toxicity studies in which reproductive organs were examined indicate that DGPE and DGBEA do not cause toxicity to reproductive organs (including the testes). Test material-related testicular toxicity was not noted in the majority of the studies with DGEE or DGEEA. Results of the developmental toxicity studies conducted with DGEE, DGBE and DGHE are almost exclusively negative. In these studies, effects on the foetus are generally not observed (even at concentrations that produced maternal toxicity). Exposure to 102 ppm (560 mg/m3) DGEE by inhalation (maximal achievable vapour concentration) or 1385 mg/kg/day DGEE by the dermal route during gestation did not cause maternal or developmental toxicity in the rat. Maternal toxicity and teratogenesis were not observed in rabbits receiving up to 1000 mg/kg/day DGBE by the dermal route during gestation; however a transient decrease in body weight was observed, which reversed by Day 21. In the mouse, the only concentration of DGEE tested (3500 mg/kg/day by gavage) caused maternal, but no foetal toxicity. Also, whereas oral administration of 2050 mg/kg/day DGBE (gavage) to the mouse and 1000 mg/kg/day DGHE (dietary) caused maternal toxicity, these doses had no effect on the developing foetus
HYDROCARBONS, C10, AROMATICS, >1% NAPHTHALENE	Repeated Dose Oral 90 d ? NOAEL = 300 mg/kg for rats (similar to OECD TG 408) Repeated Dose Inhalation 12 month ? NOAEL = 900 mg/m3 for rats (similar to OECD TG 413), read-across material C9 Aromatics * REACH Dossier For C10-12 aromatic hydrocarbons Genotoxicity: C10-C12 Aromatics are not mutagenic using in vitro or in vivo genotoxicity assays. Further data derived from read across data from the supporting substance (structural analogue or surrogate), the C9 Aromatics also support the conclusion that C10-C12 Aromatics are not genotoxic. In bacterial reverse mutation tests, C10-C12 Aromatics were not mutagenic in the presence or absence of metabolic activation. Likewise, there were no mutagenic effects reported in a read-across in vitro mammalian gene mutation test (HGPRT forward mutation assay) in the supporting substance, C9 Aromatics. No in vitro chromosomal effects were reported in a Chinese hamster ovary assay that examined the C9 Aromatics. The test substance, C10-C12 Aromatics, did not produce chromosomal effects when tested in an in vivo mouse bone marrow micronucleus assay. Likewise, the supporting substance C9 Aromatics, were not clastogenic in an in vivo mammalian bone marrow chromosome aberration test. These data demonstrate that these substances are not categorizable genotoxins either in vitro or in vivo. Furthermore, there is no evidence of hyperplastic responses or pre-neoplastic lesions in sub-chronic and chronic repeat-dose studies in either the C9 aromatics or the C10-C12 Aromatics. All studies were conducted in a manner similar or equivalent to currently established OECD guidelines. C10-C12 Aromatics are a non-genotoxic agent and classification is not warranted. Carcinogenicity: The C10-C12 aromatics did not produce pre-neoplastic changes in any tissues during sub-chronic (90 day) repeat dose testing (IUCLID sections 7.5.1 and 7.5.3). Likewise, the closely related C9 aromatic solvents did not produce hyperplastic or pre-neoplastic changes in any tissues in sub-chronic (90 day) and chronic (12 month) repeat-dose studies. No test article-related lesions, including no cancer-relevant lesions, were found in extensive histological examinations of the tissues of rats chronically exposed to C9 aromatic solvents by inhalation for 6 hours per day, 5 days per week for 12 months (C9 Aromatics IUCLID section 7.5.3). Likewise, no test article-related lesions, including no cancer-relevant lesions, were found in extensive histological examinations of the tissues of rats sub-chronically exposed to C9 Aromatic and C10-C12 Aromatic solvents by inhalation for = 90 days (IUCLID section 7.5.3). Similarly, subchronic oral dosing with C10-C12 aromatic solvent did not produce pre-neoplastic changes and had a NOAEL of 300 mg/kg bw/day (IUCLID section 7.5.1). Notably, studies on the metabolism of 14C-1,2,4-trimethylbenzene, a prototypical C9 aromatic compound and a close relative of the compounds present in C10-C12 aromatic hydrocarbons demonstrated that > 99% of the administered radioactivity was excreted in urine within 24 hours following oral dosing.1 Little or no tissue-bound residual radioactivity was detected in this study, demonstrating that the metabolism of the C9 aromatic molecules, and their close relatives in the C10-C12 Aromatic solvents, do not bioaccumulate and are unlikely to result in the formation of DNA adducts or other associated DNA lesions Developmental toxicity: C10-C12 Aromatic fluids are not developmental toxicants. In a developmental study, pregnant dams were dosed by oral gavage with 75, 150 or 450 mg/kg/day C10-C12 Aromatic fluids during gestational days 6 through 15. At 450 mg/kg/day, maternal body weight gain and food consumption were significantly decreased during the first three days of treatment. No adverse fetal effects were noted at any dose level. Thus, C10-C12 Aromatic fluids did not produce any fetal toxicity or any developmental effects in rats. Based on the study results, the maternal NOAEL is 150 mg/kg/day and the developmental NOAEL is greater than 450 mg/kg/day.
FLUROXYPYR METHYLHEPTYL ESTER	Toxicity Class WHO Table 5 * Steroid Receptor Coactivator (SRC) modulators (like inhibitors or knockouts) affect hormone-driven processes, potentially causing reproductive issues (dwarfism, infertility), immune dysregulation (autoimmunity, T-cell changes), metabolic shifts, and influencing cancer growth (both promoting and inhibiting depending on the context). Side effects often relate to their fundamental role in regulating steroid hormones (estrogen, androgen, progesterone) in tissues like the brain, reproductive organs, and immune cells, leading to broad systemic impacts, though specific inhibitors (like SRC-3 inhibitors in Tregs) show promise with fewer general toxicities than broader therapies Key Adverse Effects & Considerations: Reproductive & Endocrine: Dysfunction: SRC knockout in mice causes reproductive failure, delayed puberty, and dwarfism. Behavioral: SRC-1 inhibition can alter sex behaviors and brain development in rodents. Immune System: Treg Modulation: Inhibiting SRCs (especially SRC-3) in Regulatory T cells (Tregs) can boost anti-cancer immunity but might also impact normal immune balance.

	Potential Autoimmunity: While SRC-3 KO Tregs seem benign, broader immune targeting can lead to severe autoimmunity, though SRC inhibitors aim to avoid this. Cancer: Dual Role: SRCs drive many cancers (breast, ovarian, prostate). Modulators can inhibit tumor growth, but some (like pan-SRC inhibitors) might impair anti-tumor immunity General Systemic Effects: Metabolic: SRC dysregulation is linked to Polycystic Ovary Syndrome (PCOS) and fibroids. Brain Function: SRCs are crucial for neuroendocrine regulation, affecting seasonal cycles and hormone responses in the brain Therapeutic Context Matters: Specific vs. Pan-Inhibitors: Targeting specific SRCs (like SRC-3 in Tregs) or using targeted inhibitors (like SI-2) aims for anti-cancer effects with fewer systemic side effects than broader immune checkpoint inhibitors, avoiding issues like cytokine release syndrome. Drug Development: Natural compounds (gossypol, bufalin) and synthetic molecules (SI-2) are being developed, showing potential for non-hormonal treatments in women's health, but clinical application needs careful study. In essence, SRC modulators are powerful tools affecting fundamental hormone signaling, meaning their "adverse effects" are often the flip side of their therapeutic action, necessitating precise targeting for safe use. The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
MONOETHANOLAMINE	* Bayer While it is difficult to generalise about the full range of potential health effects posed by exposure to the many different amine compounds, characterised by those used in the manufacture of polyurethane and polyisocyanurate foams, it is agreed that overexposure to the majority of these materials may cause adverse health effects. - Many amine-based compounds can induce histamine liberation, which, in turn, can trigger allergic and other physiological effects, including bronchoconstriction or bronchial asthma and rhinitis. - Systemic symptoms include headache, nausea, faintness, anxiety, a decrease in blood pressure, tachycardia (rapid heartbeat), itching, erythema (reddening of the skin), urticaria (hives), and facial edema (swelling). Systemic effects (those affecting the body) that are related to the pharmacological action of amines are usually transient. Typically, there are four routes of possible or potential exposure: inhalation, skin contact, eye contact, and ingestion. Inhalation: Inhalation of vapors may, depending upon the physical and chemical properties of the specific product and the degree and length of exposure, result in moderate to severe irritation of the tissues of the nose and throat and can irritate the lungs. Products with higher vapour pressures have a greater potential for higher airborne concentrations. This increases the probability of worker exposure. Higher concentrations of certain amines can produce severe respiratory irritation, characterised by nasal discharge, coughing, difficulty in breathing, and chest pains. Chronic exposure via inhalation may cause headache, nausea, vomiting, drowsiness, sore throat, bronchopneumonia, and possible lung damage. Also, repeated and/or prolonged exposure to some amines may result in liver disorders, jaundice, and liver enlargement. Some amines have been shown to cause kidney, blood, and central nervous system disorders in laboratory animal studies. While most polyurethane amine catalysts are not sensitizers, some certain individuals may also become sensitized to amines and may experience respiratory distress, including asthma-like attacks, whenever they are subsequently exposed to even very small amounts of vapor. Once sensitised, these individuals must avoid any further exposure to amines. Although chronic or repeated inhalation of vapor concentrations below hazardous or recommended exposure limits should not ordinarily affect healthy individuals, chronic overexposure may lead to permanent pulmonary injury, including a reduction in lung function, breathlessness, chronic bronchitis, and immunologic lung disease. Inhalation hazards are increased when exposure to amine catalysts occurs in situations that produce aerosols, mists, or heated vapors. Such situations include leaks in fitting or transfer lines. Medical conditions generally aggravated by inhalation exposure include asthma, bronchitis, and emphysema. Skin Contact: Skin contact with amine catalysts poses a number of concerns. Direct skin contact can cause moderate to severe irritation and injury-i.e., from simple redness and swelling to painful blistering, ulceration, and chemical burns. Repeated or prolonged exposure may also result in severe cumulative dermatitis. Skin contact with some amines may result in allergic sensitisation. Sensitised persons should avoid all contact with amine catalysts. Systemic effects resulting from the absorption of the amines through skin exposure may include headaches, nausea, faintness, anxiety, decrease in blood pressure, reddening of the skin, hives, and facial swelling. These symptoms may be related to the pharmacological action of the amines, and they are usually transient. Eye Contact: Amine catalysts are alkaline in nature and their vapours are irritating to the eyes, even at low concentrations. Direct contact with the liquid amine may cause severe irritation and tissue injury, and the "burning" may lead to blindness. (Contact with solid products may result in mechanical irritation, pain, and corneal injury.) Exposed persons may experience excessive tearing, burning, conjunctivitis, and corneal swelling. The corneal swelling may manifest itself in visual disturbances such as blurred or "foggy" vision with a blue tint ("blue haze") and sometimes a halo phenomenon around lights. These symptoms are transient and usually disappear when exposure ceases. Some individuals may experience this effect even when exposed to concentrations below doses that ordinarily cause respiratory irritation. Ingestion: The oral toxicity of amine catalysts varies from moderately to very toxic. Some amines can cause severe irritation, ulceration, or burns of the mouth, throat, esophagus, and gastrointestinal tract. Material aspirated (due to vomiting) can damage the bronchial tubes and the lungs. Affected persons also may experience pain in the chest or abdomen, nausea, bleeding of the throat and the gastrointestinal tract, diarrhea, dizziness, drowsiness, thirst, circulatory collapse, coma, and even death. Polyurethane Amine Catalysts: Guidelines for Safe Handling and Disposal; Technical Bulletin June 2000 Alliance for Polyurethanes Industry
NONYLPHENOL, ETHOXYLATED, PROPOXYLATED	For nonylphenol and its compounds: Alkylphenols like nonylphenol and bisphenol A have estrogenic effects in the body. They are known as xenoestrogens. Estrogenic substances and other endocrine disruptors are compounds that have hormone-like effects in both wildlife and humans. Xenoestrogens usually function by binding to estrogen receptors and acting competitively against natural estrogens. Nonylphenol has been found to act as an agonist of GPER (G protein-coupled estrogen receptor). Nonylphenol has been shown to mimic the natural hormone 17beta-estradiol, and it competes with the endogenous hormone for binding with the estrogen receptors ERalpha and ERbeta. Effects in pregnant women. Subcutaneous injections of nonylphenol in late pregnancy causes the expression of certain placental and uterine proteins, namely CaBP-9k, which suggest it can be transferred through the placenta to the fetus. It has also been shown to have a higher potency on the first trimester placenta than the endogenous estrogen 17beta-estradiol. In addition, early prenatal exposure to low doses of nonylphenol cause an increase in apoptosis (programmed cell death) in placental cells. These "low doses" ranged from 10-13-10-9 M, which is lower than what is generally found in the environment. Nonylphenol has also been shown to affect cytokine signaling molecule secretions in the human placenta. In vitro cell cultures of human placenta during the first trimester were treated with nonylphenol, which increase the secretion of cytokines including interferon gamma, interleukin 4, and interleukin 10, and reduced the secretion of tumor necrosis factor alpha. This unbalanced cytokine profile at this part of pregnancy has been documented to result in implantation failure, pregnancy loss, and other complications. Effects on metabolism Nonylphenol has been shown to act as an obesity enhancing chemical or obesogen, though it has paradoxically been shown to have anti-obesity properties. Growing embryos and newborns are particularly vulnerable when exposed to nonylphenol because low-doses can disrupt sensitive processes that occur during these important developmental periods. Prenatal and perinatal exposure to nonylphenol has been linked with developmental abnormalities in adipose tissue and therefore in metabolic hormone synthesis and release. Specifically, by acting as an estrogen mimic, nonylphenol has generally been shown to interfere with hypothalamic appetite control. The hypothalamus responds to

the hormone leptin, which signals the feeling of fullness after eating, and nonylphenol has been shown to both increase and decrease eating behavior by interfering with leptin signaling in the midbrain. Nonylphenol has been shown mimic the action of leptin on neuropeptide Y and anorectic POMC neurons, which has an anti-obesity effect by decreasing eating behavior. This was seen when estrogen or estrogen mimics were injected into the ventromedial hypothalamus. On the other hand, nonylphenol has been shown to increase food intake and have obesity enhancing properties by lowering the expression of these anorexigenic neurons in the brain. Additionally, nonylphenol affects the expression of ghrelin: an enzyme produced by the stomach that stimulates appetite. Ghrelin expression is positively regulated by estrogen signaling in the stomach, and it is also important in guiding the differentiation of stem cells into adipocytes (fat cells). Thus, acting as an estrogen mimic, prenatal and perinatal exposure to nonylphenol has been shown to increase appetite and encourage the body to store fat later in life. Finally, long-term exposure to nonylphenol has been shown to affect insulin signaling in the liver of adult male rats.

Cancer

Nonylphenol exposure has also been associated with breast cancer. It has been shown to promote the proliferation of breast cancer cells, due to its agonistic activity on ERalpha (estrogen receptor alpha) in estrogen-dependent and estrogen-independent breast cancer cells. Some argue that nonylphenol's suggested estrogenic effect coupled with its widespread human exposure could potentially influence hormone-dependent breast cancer disease

Polyethers (such as ethoxylated surfactants and polyethylene glycols) are highly susceptible to being oxidized in the air. They then form complex mixtures of oxidation products.

Animal testing reveals that whole the pure, non-oxidised surfactant is non-sensitizing, many of the oxidation products are sensitizers. The oxidation products also cause irritation.

Human beings have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents, and other cleaning products. Exposure to these chemicals can occur through ingestion, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that volumes well above a reasonable intake level would have to occur to produce any toxic response. Moreover, no fatal case of poisoning with alcohol ethoxylates has ever been reported. Multiple studies investigating the acute toxicity of alcohol ethoxylates have shown that the use of these compounds is of low concern in terms of oral and dermal toxicity.

Clinical animal studies indicate these chemicals may produce gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhea, and lethargy. Similarly, slight to severe irritation of the skin or eye was generated when undiluted alcohol ethoxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotoxin, carcinogen, or mutagen (HERA 2007). No information was available on levels at which these effects might occur, though toxicity is thought to be substantially lower than that of nonylphenol ethoxylates.

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture.

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose allergic contact dermatitis (ACD) to these compounds by patch testing

Overall, alcohol alkoxylates (AAs) are not expected to be systemically toxic, although some short chain ethylene glycol ethers, e.g. methyl and ethyl homologues are of concern for a range of adverse health effects. They include skin and eye irritation, liver and kidney damage, bone marrow and central nervous system (CNS) depression, testicular atrophy, developmental toxicity, and immunotoxicity. For higher propyl and butyl homologues, the toxicity involves haemolysis (anaemia) with secondary effects relating to haemosiderin accumulation in the spleen, liver and kidney, and compensatory haematopoiesis in the bone marrow. Systemic toxicity was shown to decrease with increasing alkyl chain lengths and/or alkoxylation degrees (ECETOC, 2005; US EPA, 2010). The chemicals ethylene glycol hexyl ether (with a longer alkyl chain length, CAS No. 112-25-4) and diethylene glycol butyl ether (with a higher ethoxylation degree, CAS No. 112-34-5) have no evidence of systemic effects including haemolysis.

Commercially available AAs are mixtures of homologues of varying carbon chain lengths and it is possible that some of the chemicals with an average alkyl chain length $C \geq 6$ may also contain shorter alkyl chains $C < 6$. It is not practical to quantify the proportion of shorter $C < 6$ chain lengths present in such chemicals, or these shorter chain lengths may not be present at all. The available data suggest a lack of systemic toxicity for the AE chemicals with potential short alkyl chain presence (NICNASa); therefore, the toxicity of the chemicals in this assessment is unlikely to be significantly affected by the presence of shorter chain alkyl groups.

Alcohol ethoxylates are according to CESIO (2000) classified as Irritant or Harmful depending on the number of EO-units:

EO < 5 gives Irritant (Xi) with R38 (Irritating to skin) and R41 (Risk of serious damage to eyes)

EO > 5-15 gives Harmful (Xn) with R22 (Harmful if swallowed) - R38/41

EO > 15-20 gives Harmful (Xn) with R22-41

>20 EO is not classified (CESIO 2000)

Oxo-AE, C13 EO10 and C13 EO15, are Irritating (Xi) with R36/38 (Irritating to eyes and skin).

AE are not included in Annex 1 of the list of dangerous substances of the Council Directive 67/548/EEC

In general, alcohol ethoxylates (AE) are readily absorbed through the skin of guinea pigs and rats and through the gastrointestinal mucosa of rats. AE are quickly eliminated from the body through the urine, faeces, and expired air (CO₂). Orally dosed AE was absorbed rapidly and extensively in rats, and more than 75% of the dose was absorbed. When applied to the skin of humans, the doses were absorbed slowly and incompletely (50% absorbed in 72 hours). Half of the absorbed surfactant was excreted promptly in the urine and smaller amounts of AE appeared in the faeces and expired air (CO₂). The metabolism of C12 AE yields PEG, carboxylic acids, and CO₂ as metabolites. The LD50 values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.

The ability of nonionic surfactants to cause a swelling of the stratum corneum of guinea pig skin has been studied. The swelling mechanism of the skin involves a combination of ionic binding of the hydrophilic group as well as hydrophobic interactions of the alkyl chain with the substrate. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin.

Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants. A substantial amount of toxicological data and information in vivo and in vitro demonstrates that there is no evidence for alcohol ethoxylates (AEs) being genotoxic, mutagenic or carcinogenic. No adverse reproductive or developmental effects were observed. The majority of available toxicity studies revealed NOAELs in excess of 100 mg/kg bw/d but the lowest NOAEL for an individual AE was established to be 50 mg/kg bw/day. This value was subsequently considered as a conservative, representative value in the risk assessment of AE. The effects were restricted to changes in organ weights with no histopathological organ changes with the exception of liver hypertrophy (indicative of an adaptive response to metabolism rather than a toxic effect). It is noteworthy that there was practically no difference in the NOAEL in oral studies of 90-day or 2 years of duration in rats. A comparison of the aggregate consumer exposure and the systemic NOAEL (taking into account an oral absorption value of 75%) results in a Margin of Exposure of 5,800. Taking into account the conservatism in the exposure assessment and the assigned systemic NOAEL, this margin of exposure is considered more than adequate to account for the inherent uncertainty and variability of the hazard database and inter and intra-species extrapolations.

AEs are not contact sensitizers. Neat AE are irritating to eyes and skin. The irritation potential of aqueous solutions of AEs depends on concentrations. Local dermal effects due to direct or indirect skin contact in certain use scenarios where the products are diluted are not of concern as AEs are not expected to be irritating to the skin at in-use concentrations. Potential irritation of the respiratory tract is not a concern given the very low levels of airborne AE generated as a consequence of spray cleaner aerosols or laundry powder detergent dust.

In summary, the human health risk assessment has demonstrated that the use of AE in household laundry and cleaning detergents is safe and does not cause concern with regard to consumer use.

For high boiling ethylene glycol ethers (typically triethylene- and tetraethylene glycol ethers):

Skin absorption: Available skin absorption data for triethylene glycol ether (TGBE), triethylene glycol methyl ether (TGME), and triethylene glycol ethylene ether (TGEE) suggest that the rate of absorption in skin of these three glycol ethers is 22 to 34 micrograms/cm²/hr, with the methyl ether having the highest permeation constant and the butyl ether having the lowest. The rates of absorption of TGBE, TGEE and TGME are at least 100-fold less than EGME, EGEE, and EGBE, their ethylene glycol monoalkyl ether counterparts, which have absorption rates that range from 214 to 2890 micrograms/cm²/hr. Therefore, an increase in either the chain length of the alkyl substituent or the number of ethylene glycol moieties appears to lead to a decreased rate of percutaneous absorption. However, since the ratio of the change in values of the ethylene glycol to the diethylene glycol series is larger than that of the diethylene glycol to triethylene glycol series, the effect of the length of the chain and number of ethylene glycol moieties on absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetraethylene glycol methyl ether (TetraME) and tetraethylene glycol butyl ether (TetraBE) are expected to be less permeable to skin than TGME and TGBE, the differences in permeation between these molecules may only be slight.

Metabolism: The main metabolic pathway for metabolism of ethylene glycol monoalkyl ethers (EGME, EGEE, and EGBE) is oxidation via alcohol and aldehyde dehydrogenases (ALD/ADH) that leads to the formation of an alkoxy acids. Alkoxy acids are the only toxicologically significant metabolites of glycol ethers that have been detected *in vivo*. The principal metabolite of TGME is believed to be 2-[2-(2-methoxyethoxy)ethoxy] acetic acid. Although ethylene glycol, a known kidney toxicant, has been identified as an impurity or a minor metabolite of glycol ethers in animal studies it does not appear to contribute to the toxicity of glycol ethers.

The metabolites of category members are not likely to be metabolized to any large extent to toxic molecules such as ethylene glycol or the mono alkoxy acids because metabolic breakdown of the ether linkages also has to occur.

Acute toxicity: Category members generally display low acute toxicity by the oral, inhalation and dermal routes of exposure. Signs of toxicity in animals receiving lethal oral doses of TGBE included loss of righting reflex and flaccid muscle tone, coma, and heavy breathing. Animals administered lethal oral doses of TGEE exhibited lethargy, ataxia, blood in the urogenital area and piloerection before death.

Irritation: The data indicate that the glycol ethers may cause mild to moderate skin irritation. TGEE and TGBE are highly irritating to the eyes. Other category members show low eye irritation.

Repeat dose toxicity: Results of these studies suggest that repeated exposure to moderate to high doses of the glycol ethers in this category is required to produce systemic toxicity.

In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME.

Testicular effects included spermatid giant cells, focal tubular hypospermatogenesis, and increased cytoplasmic vacuolisation. Due to a high incidence of similar spontaneous changes

in normal New Zealand White rabbits, the testicular effects were considered not to be related to treatment. Thus, the NOAELs for TGME, TGEE and TGBE were established at 1000 mg/kg/day. Findings from this report were considered unremarkable.

A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day. In this study, significantly-increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500 mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or

haemolysed blood in the stomach. These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500

mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats.

In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity.

Mutagenicity: Mutagenicity studies have been conducted for several category members. All *in vitro* and *in vivo* studies were negative at concentrations up to 5,000 micrograms/plate and 5,000 mg/kg, respectively, indicating that the category members are not genotoxic at the concentrations used in these studies. The uniformly negative outcomes of various mutagenicity studies performed on category members lessen the concern for carcinogenicity.

Reproductive toxicity: Although mating studies with either the category members or surrogates have not been performed, several of the repeated dose toxicity tests with the surrogates have included examination of reproductive organs. A lower molecular weight glycol ether, ethylene glycol methyl ether (EGME), has been shown to be a testicular toxicant. In addition, results of repeated dose toxicity tests with TGME clearly show testicular toxicity at an oral dose of 4,000 mg/kg/day four times greater than the limit dose of 1,000 mg/kg/day recommended for repeat dose studies. It should be noted that TGME is 350 times less potent for testicular effects than EGME. TGBE is not associated with testicular toxicity, TetraME is not likely to be metabolised by any large extent to 2-MAA (the toxic metabolite of EGME), and a mixture containing predominantly methylated glycol ethers in the C5-C11 range does not produce testicular toxicity (even when administered intravenously at 1,000 mg/kg/day).

Developmental toxicity: The bulk of the evidence shows that effects on the foetus are not noted in treatments with 1,000 mg/kg/day during gestation. At 1,250 to 1,650 mg/kg/day TGME (in the rat) and 1,500 mg/kg/day (in the rabbit), the developmental effects observed included skeletal variants and decreased body weight gain.

for nonylphenol:

Nonylphenol was studied for oral toxicity in rats in a 28-day repeat dose toxicity test at doses of 0, 4, 15, 60 and 250 mg/kg/day. Changes suggesting renal dysfunction were mainly noted in both sexes given 250 mg/kg. Liver weights were increased in males given 60 mg/kg and in both sexes given 250 mg/kg group. Histopathologically, hypertrophy of the centrilobular hepatocytes was noted in both sexes given 250 mg/kg. Kidney weights were increased in males given 250 mg/kg and macroscopically, disseminated white spots, enlargement and pelvic dilatation were noted in females given 250 mg/kg. Histopathologically, the following lesions were noted in the 250 mg/kg group: basophilic change of the proximal tubules in both sexes, single cell necrosis of the proximal tubules, inflammatory cell infiltration in the interstitium and casts in females, basophilic change and dilatation of the collecting tubules in both sexes, simple hyperplasia of the pelvic mucosa and pelvic dilatation in females. In the urinary bladder, simple hyperplasia was noted in both sexes given 250 mg/kg. In the caecum, macroscopic dilatation was noted in both sexes given 250 mg/kg. Almost all changes except those in the kidney disappeared after a 14-day recovery period. The NOELs for males and females are considered to be 15 mg/kg/day and 60 mg/kg/day, respectively, under the conditions of the present study.

Nonylphenol was not mutagenic to *Salmonella typhimurium*, TA100, TA1535, TA98, TA1537 and *Escherichia coli* WP2 *uvrA*, with or without an exogenous metabolic activation system.

Nonylphenol induced neither structural chromosomal aberrations nor polyploidy in CHL/IU cells, in the absence or presence of an exogenous metabolic activation system.

POTASSIUM HYDROXIDE

The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis.
Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.

POTASSIUM ACETATE

* REACH Dossier

MECOPROP-P-POTASSIUM & MCPA

For chlorophenoxy pesticides:
551chlph

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 hepatocarcinogens, reproductive toxicants, immunotoxicants and procarcinogens) by interaction with a cytosolic 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 be 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 thyroxine (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 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

MECOPROP-P-POTASSIUM & MCPA & HYDROCARBONS, C10, AROMATICS, >1% NAPHTHALENE & MONOETHANOLAMINE & NONYLPHENOL, ETHOXYLATED, PROPOXYLATED & POTASSIUM HYDROXIDE & POTASSIUM ACETATE	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
MCPA & MONOETHANOLAMINE	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.
NONYLPHENOL, ETHOXYLATED, PROPOXYLATED & 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	✓
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

Saxon®	Endpoint	Test Duration (hr)	Species	Value	Source
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Continued...

	Not Available	Not Available	Not Available	Not Available	Not Available
mecoprop-P-potassium	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	150-220mg/l	Not Available
MCPA	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	672h	Fish	<1<1.3	7
	EC50	72h	Algae or other aquatic plants	0.38mg/l	1
	EC50	48h	Crustacea	>280.8mg/L	4
	EC50	96h	Algae or other aquatic plants	0.21mg/l	1
	EC10(ECx)	336h	Algae or other aquatic plants	0.007mg/l	1
	LC50	96h	Fish	>2.995mg/L	4
diethylene glycol monobutyl ether	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	1101mg/l	2
	EC50	48h	Crustacea	>100mg/l	1
	NOEC(ECx)	96h	Algae or other aquatic plants	>=100mg/l	1
	EC50	96h	Algae or other aquatic plants	>100mg/l	1
	LC50	96h	Fish	1300mg/l	2
hydrocarbons, C10, aromatics, >1% naphthalene	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	<1mg/l	1
	EC50	48h	Crustacea	0.95mg/l	1
	EC50	96h	Algae or other aquatic plants	11.7mg/l	2
	EC50(ECx)	48h	Crustacea	0.95mg/l	1
	LC50	96h	Fish	0.58mg/l	2
fluroxypyr methylheptyl ester	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	96h	Algae or other aquatic plants	>1.864mg/L	4
	LC50	96h	Fish	>0.115mg/L	4
	EC50(ECx)	120h	Algae or other aquatic plants	0.074mg/L	4
monoethanolamine	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	15mg/l	1
	EC50	48h	Crustacea	65mg/l	1
	NOEC(ECx)	72h	Algae or other aquatic plants	4mg/l	1
	EC50	96h	Algae or other aquatic plants	80mg/l	2
	LC50	96h	Fish	75mg/l	1
nonylphenol, ethoxylated, propoxylated	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
potassium hydroxide	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	24h	Fish	28mg/l	2
	LC50	96h	Fish	80mg/l	2
potassium acetate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>1000mg/l	2
	EC50	48h	Crustacea	>919mg/l	2
	EC0(ECx)	24h	Crustacea	564mg/l	2
	LC50	96h	Fish	262-340mg/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. 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				

Very 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.

for chlorophenoxy herbicides:

Environmental fate:

Residues of chlorophenoxy herbicides in the environment are the consequence of the direct application of these compounds to agricultural and non-agricultural areas.

Biodegradation is the primary route of elimination from the environment; photolysis and hydrolysis also contribute to their removal.

The chlorophenoxy herbicides are considered to have only marginal potential for leaching to groundwater. In basic waters, phenoxy herbicide esters are hydrolysed to the anionic forms; in acidic waters, photodegradation or vaporisation predominates, depending on the ester.

Chlorophenoxy herbicides may be transported in the atmosphere in the form of droplets, vapour, or powder following application by spraying.

Chlorophenoxy herbicides may be present in food as a result of their direct application to crops; however, concentrations are normally low

For mecoprop (syn: 2-(2-methyl-4-chlorophenoxy)propionic acid; MCP):

log Kow : 0.1004

DT50 in soil 7-13 d. .

Koc 12-25

Half-life (hr) soil : 1440

Environmental fate:

Breakdown of Chemical in Soil and Groundwater: The duration of mecoprop's residual activity in soil is about two months. Adsorption of mecoprop increases with an increase in organic matter in the soil. Unaged mecoprop and its salts are very mobile in a variety of soils. Because of this high mobility, it may potentially leach into groundwater.

In soil mecoprop is degraded by microorganisms to 4-chloro-2-methylphenol, followed by ring hydroxylation at the 6-position and ring opening

Plants: In plants mecoprop is hydroxylated to form 2-hydroxymethyl-4-chlorophenoxypropionic acid

Ecotoxicity:

Effects on Birds:

Bird LC50: mallard duck >5620 ppm, bobwhite quail 5000 ppm

Bird acute oral LD50: Japanese quail 740 mg/kg, bobwhite quail 700 mg/kg

Mecoprop is practically non-toxic to birds.

Effects on Aquatic Organisms:

Fish LC50 (96 h): rainbow trout 124 ppm, bluegill sunfish >100 ppm

Daphnia EC50 (48 h) 420 mg/L

Mecoprop is virtually non-toxic to fish. Available data indicate a low potential of mecoprop to bioaccumulate in fish.

Effects on Other Animals (Nontarget species):

Mecoprop is not toxic to honeybees LD50 (oral) > 10 ug/bee, (contact) >100 ug/bee

For petroleum distillates:

Environmental fate:

When petroleum substances are released into the environment, four major fate processes will take place: dissolution in water, volatilization, biodegradation and adsorption.

These processes will cause changes in the composition of these UVCB substances. In the case of spills on land or water surfaces, photodegradation-another fate process-can also be significant.

As noted previously, the solubility and vapour pressure of components within a mixture will differ from those of the component alone. These interactions are complex for complex UVCBs such as petroleum hydrocarbons.

Each of the fate processes affects hydrocarbon families differently. Aromatics tend to be more water-soluble than aliphatics of the same carbon number, whereas aliphatics tend to be more volatile. Thus, when a petroleum mixture is released into the environment, the principal water contaminants are likely to be aromatics, whereas aliphatics will be the principal air contaminants. The trend in volatility by component class is as follows: alkenes = alkanes > aromatics = cycloalkanes.

The most soluble and volatile components have the lowest molecular weight; thus there is a general shift to higher molecular weight components in residual materials.

Biodegradation:

Biodegradation is almost always operative when petroleum mixtures are released into the environment. It has been widely demonstrated that nearly all soils and sediments have populations of bacteria and other organisms capable of degrading petroleum hydrocarbons. Degradation occurs both in the presence and absence of oxygen. Two key factors that determine degradation rates are oxygen supply and molecular structure. In general, degradation is more rapid under aerobic conditions. Decreasing trends in degradation rates according to structure are as follows:

(1) n-alkanes, especially in the C10–C25 range, which are degraded readily;

(2) isoalkanes;

(3) alkenes;

(4) benzene, toluene, ethylbenzene, xylenes (BTEX) (when present in concentrations that are not toxic to microorganisms);

(5) monoaromatics;

(6) polynuclear (polycyclic) aromatic hydrocarbons (PAHs); and

(7) higher molecular weight cycloalkanes (which may degrade very slowly).

Three weathering processes-dissolution in water, volatilization and biodegradation-typically result in the depletion of the more readily soluble, volatile and degradable compounds and the accumulation of those most resistant to these processes in residues.

When large quantities of a hydrocarbon mixture enter the soil compartment, soil organic matter and other sorption sites in soil are fully saturated and the hydrocarbons will begin to form a separate phase (a non-aqueous phase liquid, or NAPL) in the soil. At concentrations below the retention capacity for the hydrocarbon in the soil, the NAPL will be immobile; this is referred to as residual NAPL. Above the retention capacity, the NAPL becomes mobile and will move within the soil.

Bioaccumulation:

Bioaccumulation potential was characterized based on empirical and/or modelled data for a suite of petroleum hydrocarbons expected to occur in petroleum substances.

Bioaccumulation factors (BAFs) are the preferred metric for assessing the bioaccumulation potential of substances, as the bioconcentration factor (BCF) may not adequately account for the bioaccumulation potential of substances via the diet, which predominates for substances with log Kow > ~4.5.

In addition to fish BCF and BAF data, bioaccumulation data for aquatic invertebrate species were also considered. Biota-sediment/soil accumulation factors (BSAFs), trophic magnification factors and biomagnification factors were also considered in characterizing bioaccumulation potential.

Overall, there is consistent empirical and predicted evidence to suggest that the following components have the potential for high bioaccumulation, with BAF/BCF values greater than 5000: C13–C15 isoalkanes, C12 alkenes, C12–C15 one-ring cycloalkanes, C12 and C15 two-ring cycloalkanes, C14 polycycloalkanes, C15 one-ring aromatics, C15 and C20 cycloalkane monoaromatics, C12–C13 diaromatics, C20 cycloalkane diaromatics, and C14 and C20 three-ring PAHs.

These components are associated with a slow rate of metabolism and are highly lipophilic. Exposures from water and diet, when combined, suggest that the rate of uptake would exceed that of the total elimination rate. Most of these components are not expected to biomagnify in aquatic or terrestrial foodwebs, largely because a combination of metabolism, low dietary assimilation efficiency and growth dilution allows the elimination rate to exceed the uptake rate from the diet; however, one study suggests that some alkyl-PAHs may biomagnify. While only BSAFs were found for some PAHs, it is possible that BSAFs will be > 1 for invertebrates, given that they do not have the same metabolic competency as fish.

In general, fish can efficiently metabolize aromatic compounds. There is some evidence that alkylation increases bioaccumulation of naphthalene but it is not known if this can be generalized to larger PAHs or if any potential increase in bioaccumulation due to alkylation will be sufficient to exceed a BAF/BCF of 5000.

Some lower trophic level organisms (i.e., invertebrates) appear to lack the capacity to efficiently metabolize aromatic compounds, resulting in high bioaccumulation potential for some aromatic components as compared to fish.

This is the case for the C14 three-ring PAH, which was bioconcentrated to a high level (BCF > 5000) by invertebrates but not by fish. There is potential for such bioaccumulative components to reach toxic levels in organisms if exposure is continuous and of sufficient magnitude, though this is unlikely in the water column following a spill scenario due to relatively rapid dispersal.

Bioaccumulation of aromatic compounds might be lower in natural environments than what is observed in the laboratory. PAHs may sorb to organic material suspended in the water column (dissolved humic material), which decreases their overall bioavailability primarily due to an increase in size. This has been observed with fish.

Ecotoxicity:

Diesel fuel studies in salt water are available. The values varied greatly for aquatic species such as rainbow trout and *Daphnia magna*, demonstrating the inherent variability of diesel fuel compositions and its effects on toxicity. Most experimental acute toxicity values are above 1 mg/L. The lowest 48-hour LC50 for salmonids was 2.4 mg/L. *Daphnia magna* had a 24-hour LC50 of 1.8 mg/L. The values varied greatly for aquatic species such as rainbow trout and *Daphnia magna*, demonstrating the inherent variability of diesel fuel compositions and its effects on toxicity. Most experimental acute toxicity values are above 1 mg/L. The lowest 48-hour LC50 for salmonids was 2.4 mg/L. *Daphnia magna* had a 24-hour LC50 of 1.8 mg/L.

The tropical mysid *Metamysidopsis insularis* was shown to be very sensitive to diesel fuel, with a 96-hour LC50 value of 0.22 mg/L. This species has been shown to be as sensitive as temperate mysids to toxicants. However, this study used nominal concentrations, and therefore was not considered acceptable. In another study involving diesel fuel, the effect on brown or common shrimp (*Crangon crangon*) a 96-hour LC50 of 22 mg/L was determined. A "gas oil" was also tested and a 96-hour LC50 of 12 mg/L was determined.

The steady state cell density of marine phytoplankton decreased with increasing concentrations of diesel fuel, with different sensitivities between species. The diatom *Phaeodactylum tricornutum* showed a 20% decrease in cell density in 24 hours following a 3 mg/L exposure with a 24-hour no-observed effect concentration (NOEC) of 2.5 mg/L. The microalga *Isochrysis galbana* was more tolerant to diesel fuel, with a 24-hour lowest-observed-effect concentration (LOEC) of 26 mg/L (14% decrease in cell density), and a NOEC of 25 mg/L.

Finally, the green algae *Chlorella salina* was relatively insensitive to diesel fuel contamination, with a 24-hour LOEC of 170 mg/L (27% decrease in cell density), and a NOEC of 160 mg/L. All populations of phytoplankton returned to a steady state within 5 days of exposure.

In sandy soils, earthworm (*Eisenia fetida*) mortality only occurred at diesel fuel concentrations greater than 10 000 mg/kg, which was also the concentration at which sub-lethal weight loss was recorded.

Nephrotoxic effects of diesel fuel have been documented in several animal and human studies. Some species of birds (mallard ducks in particular) are generally resistant to the toxic effects of petrochemical ingestion, and large amounts of petrochemicals are needed in order to cause direct mortality.

For Glycol Ethers:

Environmental Fate: Several glycol ethers have been shown to biodegrade however; biodegradation slows as molecular weight increases. No glycol ethers that have been tested demonstrate marked resistance to biodegradative processes. No glycol ethers that have been tested demonstrate marked resistance to biodegradative processes.

Atmospheric Fate: Upon release to the atmosphere by evaporation, high boiling glycol ethers are estimated to undergo photo-degradation (atmospheric half lives = 2.4-2.5 hr).

Aquatic Fate: In water, glycol ethers undergo biodegradation (typically 47-92% after 8-21 days) and have a low potential for bioaccumulation (log Kow ranges from -1.73 to +0.51).

Ecotoxicity: Tri- and tetra ethylene glycol ethers are "practically non-toxic" to aquatic species. No major differences are observed in the order of toxicity going from the methyl- to the butyl ethers. Glycols exert a high oxygen demand for decomposition and once released to the environment death of aquatic organisms occurs if dissolved oxygen is depleted.

for naphthalene:

Environmental fate:

Naphthalene released to the atmosphere may be transported to surface water and/or soil by wet or dry deposition. Since most airborne naphthalene is in the vapor phase, deposition is expected to be very slow (about 0.04–0.06 cm/sec). It has been estimated that about 2–3% of naphthalene emitted to air is transported to other environmental media, mostly by dry deposition.

Naphthalene in surface water may volatilise to the atmosphere. The rate of volatilization also depends upon several environmental conditions, including temperature, wind velocity, and mixing rates of the air and water columns.

Log octanol/water partition coefficients (Kow) for naphthalene range from 3.29 to 3.37 and log organic carbon coefficients (Koc) range from 2.97 to 3.27. The reported experimentally determined log Koc is 3.11. Based on the magnitude of these values, it is expected that only a small fraction (<10%) of naphthalene in typical surface water would be associated with particulate matter. Thus, naphthalene discharged to surface waters would remain largely in solution, with smaller quantities being associated with suspended solids and benthic sediments.

Naphthalene is easily volatilized from aerated soils and is adsorbed to a moderate extent (10%). The extent of sorption depends on the organic carbon content of the soil, with rapid movement expected through sandy soils. The estimated soil adsorption coefficient for naphthalene in a soil with <0.6% organic carbon is 1.8. Because it adsorbs to aquifer material, naphthalene's passage through groundwater will be somewhat retarded. However, sorption of naphthalene to aquifer materials with low organic carbon content (<0.03%) may be enhanced by the presence of nonionic low-polarity organics, such as tetrachloroethene, commonly found at hazardous waste sites. Bioconcentration factors (BCFs) for naphthalene have been measured and calculated from the Kow, Koc, or water solubility. The values reported for log BCF range from 1.6 to 3, indicating moderate bioconcentration in aquatic organisms. Naphthalene is reported to be rapidly eliminated from invertebrates when the organisms are placed in pollutant-free water, and naphthalene is readily metabolized in fish. Based on the magnitude of the Kow, bioaccumulation in the food chain is not expected to occur. However, naphthalene exposure of cows and chickens could lead to the presence of naphthalene in milk and eggs.

Limited data were located on transport and partitioning of methylnaphthalenes in the environment. The respective vapor pressures (0.054 and 0.068 mmHg), water solubilities (25.8 and 24.6 mg/L), and Henry's law constants (3.60x10⁻⁴ and 4.99x10⁻⁴ atm-m³/mol) for 1-methylnaphthalene and 2-methylnaphthalene are of similar magnitude to these properties for naphthalene. Thus, it is likely that loss of methylnaphthalenes from ambient water occurs by volatilization. Based on the magnitude of log Kow for 1-methylnaphthalene and 2-methylnaphthalene (3.87 and 3.86, respectively) and the experimental log Koc for 2-methylnaphthalene (3.93), these chemicals may partition similarly to naphthalene in environmental media and are expected to be slightly mobile to immobile in soils. Log BCFs calculated for 2-methylnaphthalene range from 2 to 2.8 and measured log BCFs for 1-methylnaphthalene and 2-methylnaphthalene in oysters ranged from 2.7 to 4.1. Methylnaphthalenes are also metabolized and excreted rapidly by fish and shellfish when they are removed from polluted waters.

The most important atmospheric removal process for naphthalene is reaction with photochemically produced hydroxyl radicals. The major products of this reaction are 1- and 2-naphthol and 1- and 2-nitronaphthalene. Naphthalene also reacts with N₂O₅, nitrate radicals, and ozone in the atmosphere and photolysis is expected to occur. Methylnaphthalenes also react with hydroxyl radicals. The reported rate constants are 5.30x10⁻¹¹ and 5.23x10⁻¹¹ cm³/molecule-sec for 1-methylnaphthalene and 2-methylnaphthalene, respectively. Based on an atmospheric hydroxyl radical concentration of 1x10⁶/cm³, the corresponding atmospheric half-lives are 3.6 and 3.7 hours. Reactions of 1-methylnaphthalene and 2-methylnaphthalene with N₂O₅ radicals have half-lives of 24 and 19 days, respectively. These chemicals also react with atmospheric ozone.

Naphthalene and methylnaphthalenes are degraded in water by photolysis and biological processes. The half-life for photolysis of naphthalene in surface water is estimated to be about 71 hours, but the half-life in deeper water (5 m) is estimated at 550 days. The half-lives for photolysis of 1-methylnaphthalene and 2-methylnaphthalene were estimated at 22 and 54 hours, respectively.

Biodegradation of naphthalene is sufficiently rapid for it to be a dominant fate process in aquatic systems. Data on biodegradation of naphthalene in biodegradability tests and natural systems suggest that biodegradation occurs after a relatively short period of acclimation. Methylnaphthalenes are biodegraded under aerobic conditions after adaptation. The highest degradation rates were reported in water constantly polluted with petroleum.

Naphthalene biodegradation rates are about 8–20 times higher in sediment than in the water column above the sediment. Methylnaphthalenes biodegrade more slowly. Reported half-lives in sediments were 46 weeks for 1-methylnaphthalene and ranged from 14 to 50 weeks for 2-methylnaphthalene.

In soils, biodegradation potential is important to biological remediation of soil. Studies on biodegradation of PAHs suggest that adsorption to the organic matter significantly reduces the bioavailability for microorganisms, and thus the biodegradability, of PAHs, including naphthalene. Biodegradation is accomplished through the action of aerobic microorganisms and declines precipitously when soil conditions become anaerobic. Studies indicate that naphthalene biodegrades to carbon dioxide in aerobic soils, with salicylate as an intermediate product. Abiotic degradation of naphthalene seldom occurs in soils. The behavior of 1-methylnaphthalene in sandy loam was very similar to that of naphthalene. 1-Methylnaphthalene was easily volatilized from aerated soil, and the biodegradation half-life averaged between 1.7 and 2.2 days.

Ecotoxicity:

Acute toxicity data on naphthalene are available for several fish species (freshwater and marine). If low reliable data (too old, static test, nominal concentrations) are excluded, 96h LC50 values range from 1.8 to 7.8 mg/L. Comparable results were obtained with other vertebrates (amphibians).

From chronic toxicity tests, a precise NOEL is not clearly determined. A NOEC of 0.12 mg/L was observed in a 40 days test on juvenile pink salmon, but 50% mortality at 0.11 mg/L was calculated for trout fry exposed during hatching.

Several data are also available for invertebrates, showing 48h EC50 values ranging from 2.1 to 24 mg/L. Also in this case, higher figures must be taken with care due to nominal concentrations.

Chronic data on freshwater invertebrates are methodologically unclear or questionable.

On algae too, data available are obtained with hardly comparable methodological approaches. 50% photosynthesis reduction was observed at 2.8 mg/L in 4 hours experiments.

QSAR predictions using equations for narcosis give results consistent with experimental short-term data on fish daphnia and algae.

For diethylene glycol monoalkyl ethers and their acetates:

The diethylene glycol ethers are soluble in water. Octanol-water partition coefficients (log Kow values) range from -0.69 to +1.3. Henry's Law Constants range from 8.63 E-10 to 9.91 E-8 atm-m³/mole.

Environmental fate:

Estimated hydroxyl radical-induced atmospheric photodegradation half-lives range from 3.18 to 4.41 hours. DGEE, DGPE, and DGHE possess no functional groups in their molecular structures that are readily subject to hydrolysis in the presence of water. The acetate ester groups of DGEEA and DGBEA will hydrolyse, with the hydrolysis rate dependent on temperature, pH and possible presence of impurities that can act as catalysts for hydrolysis.

Level III fugacity modeling indicates that category members, when released to air and water, will partition to water (48.7-66.6%), soil (30.4-50.7%), air (0.52-2.88%), and sediment (0.08-0.15%). Estimates of soil and sediment partition coefficient (Kocs ranging from 1 - 10) suggest that category members would exhibit high soil mobility.

Estimated bioconcentration factors (log BCF) range from 0.299 to 0.609. Category members for which adequate data are available (DGEE, DGHE and DGBEA) are readily biodegradable. These physicochemical and environmental fate properties indicate that category members will not persist in the environment or bioaccumulate.

Ecotoxicity:

The aquatic toxicity data for the diethylene glycol ethers (DGEE, DGPE and DGHE) and diethylene glycol ether acetates (DGEEA and DGBEA) are considered separately because diethylene glycol ether acetates do not hydrolyse readily in water at environmental conditions. The EC/LC50 values for the diethylene glycol ethers in fish range from 200 (for DGHE) to > 5000 mg/l (for DGPE and DGEE). In invertebrates, values range from 433 for DGHE to > 10,000 mg/l for DGEE and DGPE. No algae data are available for the category members; based on data from supporting chemicals, toxicity is expected to be > 100 mg/l for DGHE and > 500 mg/l for other category members.

The LC50 values for fish are 110 mg/l for DGEEA and 77 mg/l for DGBEA. Measured or estimated values for invertebrates are higher (665 mg/l measured for DGBEA, with higher estimated values for both chemicals). No algae data are available for the acetates; based on experimental data for EGBEA, EC50 values are expected to be > 500 mg/l.

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.

for monoethanolamine:

log Kow : -1.31

Koc : 5

Half-life (hr) air : 11

Henry's atm m³/mol: 4.00E-08

BOD 5: 0.8-1.1,0%
Biodegradability:
BOD5: 800 mg/g
>70%: BOD of the ThOD (OECD 301F)
>90%: DOC reduction (OECD 301A)
COD : 1.27-1.28
ThOD : 2.49
BCF : <1

Environmental fate:
Monoethanolamine will leach into soil. It is expected to exist solely as a vapor in the ambient atmosphere. Models estimate that this material will preferentially partition to water versus air or soil.. Vapour-phase is degraded in the atmosphere by reaction with photochemically produced hydroxyl radicals
The potential for mobility in the soil is high (Koc between 0 and 50).
Log soil organic carbon partition coefficient (log Koc) is estimated to be 0.70.

Degradation and Persistence:
The material is biodegradable, passing the OECD tests for ready biodegradability.
Biodegradation reached in CO2 evolution test after 28 days: 97%* (modified Sturm Test, OECD 301B)
Biodegradation reached in modified OECD Screening Test after 28 days: 94%* (OECD 301E)
Biodegradation reached in manometric Respirometer Test after 28 days:>70%* (OECD 301F)
Biodegradation under aerobic static laboratory conditions is high (BOD20 or BOD28/ThOD >40%)
BOD20 (Biochemical Oxygen Demand after 20 days): 1.5 p/p
ThOD (Theoretical Oxygen Demand)): 2.36 p/p (calc).
IC50 (Inhibitory Concentration): >1000 mg/l
Biodegradation: Test method: OECD 301F; ISO 9408; 92/69/EEC, C.4-D (aerobic), activated sludge, domestic
Degree of elimination: 90 - 100 % (28 d)
Evaluation: Readily biodegradable (according to OECD criteria).
This material will biodegrade relatively rapidly in both soil and water, and will not persist in the environment.

Monoethanolamine is biodegraded or transformed into other compounds under both aerobic and anaerobic conditions even at concentrations greater than 1500 mg/kg.
Ammonium, acetate, and nitrogen gas are the dominant by-products . The generation of nitrogen gas suggests that simultaneous nitrification and denitrification occurs because of the existence of anoxic zones resulting from diffusion limited oxygen transport into the soils.Low temperatures (5 C) reduced the biodegradation rates significantly compared to rates at room temperature.

Bioaccumulation: Because of the n-octanol/water distribution coefficient (log Pow) accumulation in organisms is not to be expected.
Bioconcentration potential is low (BCF less than 100 or log Kow less than 3).
Bioaccumulation: Because of this material's high solubility and rapid biodegradability, it is unlikely that bioaccumulation will occur in aquatic or terrestrial systems.

Biochemical oxygen demand (BOD): Incubation period 5 d: 800 mg/g
Due to the pH-value of the product, neutralization is generally required before discharging sewage into treatment plants.

Ecotoxicity:
This material is highly soluble in water. Laboratory toxicity tests indicate that is not significantly toxic to fish and aquatic invertebrates, although amphibians may be more sensitive. Wildlife species may be more susceptible since mammals and birds do not readily metabolise this material. The odor and flavor of this material may attract some wildlife and cause them to consume spilled material
BASF date:

Fish LC50 (96 h): goldfish 170 mg/l (APHA 1971 static)
Daphnia magna EC50 (48 h): 65 mg/l (Directive 84/449/EEC); (24 h): 120-140 mg/l
Aquatic plants EC50 (72 h): green algae 22 mg/l (Guideline 92/69/EEC)
Algae NOEC (192 h): 0.75-0.97 mg/l
Activated sludge EC50 (0.5 h): >1000 mg/l (DIV/EN/ISO 8192-OECD 209-88/302/EEC)
Bacteria EC50 (17 h): 100 mg/l
Dow Chemicals data

The material is practically non-toxic to aquatic organisms on an acute basis (LC50/ EC50 >100 mg/l in most sensitive species).
Daphnia LC50 (-) 114 mg/l *
Fish LC50 (-): Oncorhynchus mykiss 150 mg/l, gold fish 170 mg/l, bluegill 300-1000 mg/l, fathead minnow 635 mg/l, mosquito fish 337.5, golden orfe 224-525 mg/l
* (Dow Chemical)

BOD5: 60%; BOD19: 75%; BOD20: 100% **
Toxicity to microorganisms: IC50 700 mg/l
Daphnia LC50 (48 h): 33 mg/l; 93 mg/l **
Fish LC50 (96 h): fathead minnow 125 mg/l, 206 mg/l **
ThOD: 1.54 mg/mg (measured); 1.31 mg/mg (calculated) **
Monoethanolamine may be toxic to aquatic life at relatively low concentrations in water.

Editors note: there is clear contradiction between the conclusion reached by Dow Chemical and other manufacturers relating to aquatic toxicity. Under present EC Directives the material is not toxic to aquatic life.
** (Dow Chemical)

MCPA and its formulations are rapidly degraded by soil microorganisms and it has low persistence, with a reported field half-life of 14 days to 1 month, depending on soil moisture and soil organic matter. Decreased soil moisture and microbial activity, as well as increased soil organic matter, will prolong the field half-life for MCPA.
Water – MCPA is relatively stable to light breakdown, but can be rapidly broken down by microorganisms. In sterilized water, it takes about five weeks for half of the compound to degrade due to the action of sunlight. In rice paddy water, however, MCPA is almost totally degraded by aquatic microorganisms in under two weeks.

Transport
MCPA readily leaches in most soils, but its mobility decreases with increasing organic matter. MCPA and its formulations show little affinity for soil.

Metabolism
Mode of Action – MCPA works by concentrating in the actively growing regions of a plant (meristematic tissue) where it interferes with protein synthesis, cell division and ultimately the growth of the plant .
Plant Metabolism – MCPA is absorbed, translocated, and actively broken down by vegetation. The metabolite found in plants is 2-methyl-4-chlorophenol.
Prevent, by any means available, spillage from entering drains or water courses.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
MCPA	HIGH (Half-life = 180 days)	LOW (Half-life = 2.19 days)
diethylene glycol monobutyl ether	LOW	LOW
fluroxypyr methylheptyl ester	HIGH	HIGH
monoethanolamine	LOW	LOW
water	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
MCPA	LOW (BCF = 13)

Ingredient	Bioaccumulation
diethylene glycol monobutyl ether	LOW (BCF = 0.46)
hydrocarbons, C10, aromatics, >1% naphthalene	LOW (BCF = 159)
fluroxypyr methylheptyl ester	HIGH (LogKOW = 4.53)
monoethanolamine	LOW (LogKOW = -1.31)
water	LOW (LogKOW = -1.38)

Mobility in soil

Ingredient	Mobility
MCPA	LOW (Log KOC = 29.41)
diethylene glycol monobutyl ether	LOW (Log KOC = 10)
fluroxypyr methylheptyl ester	LOW (Log KOC = 13.72)
monoethanolamine	HIGH (Log KOC = 1)

SECTION 13 Disposal considerations**Waste treatment methods**

Product / Packaging disposal	- Containers may still present a chemical hazard/ danger when empty. - Return to supplier for reuse/ recycling if possible. Otherwise: - If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. - Where possible retain label warnings and SDS and observe all notices pertaining to the product. 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. - Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. - Treat and neutralise at an approved treatment plant. - Treatment should involve: Neutralisation with suitable dilute acid followed 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.

Ensure that the hazardous substance is disposed in accordance with the Hazardous Substances (Disposal) Notice 2017

Disposal Requirements

Packages that have been in direct contact with the hazardous substance must be only disposed if the hazardous substance was appropriately removed and cleaned out from the package. The package must be disposed according to the manufacturer's directions taking into account the material it is made of. Packages which hazardous content have been appropriately treated and removed may be recycled.

The hazardous substance must only be disposed if it has been treated by a method that changed the characteristics or composition of the substance and it is no longer hazardous.

Only dispose to the environment if a tolerable exposure limit has been set for the substance.

Only deposit the hazardous substance into or onto a landfill or sewage facility or incinerator, where the hazardous substance can be handled and treated appropriately.

SECTION 14 Transport information**Labels Required**

	
Marine Pollutant	
HAZCHEM	2X

Land transport (UN)

14.1 UN number or ID number	1760
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14.2 UN proper shipping name	CORROSIVE LIQUID, N.O.S. (contains MCPA and mecoprop-P-potassium)	
14.3 Transport hazard class(es)	Class	8
	Subsidiary Hazard	Not Applicable
14.4. Packing group	III	
14.5. Environmental hazard	Environmentally hazardous	
14.6 Special precautions for user	Special provisions	223; 274
	Limited quantity	5 L

Air transport (ICAO-IATA / DGR)

14.1. UN number	1760	
14.2 UN proper shipping name	Corrosive liquid, n.o.s. * (contains MCPA and mecoprop-P-potassium)	
14.3 Transport hazard class(es)	ICAO/IATA Class	8
	ICAO / IATA Subsidiary Hazard	Not Applicable
	ERG Code	8L
14.4. Packing group	III	
14.5. Environmental hazard	Environmentally hazardous	
14.6 Special precautions for user	Special provisions	A3 A803
	Cargo Only Packing Instructions	856
	Cargo Only Maximum Qty / Pack	60 L
	Passenger and Cargo Packing Instructions	852
	Passenger and Cargo Maximum Qty / Pack	5 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y841
	Passenger and Cargo Limited Maximum Qty / Pack	1 L

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	1760	
14.2 UN proper shipping name	CORROSIVE LIQUID, N.O.S. (contains MCPA and mecoprop-P-potassium)	
14.3 Transport hazard class(es)	IMDG Class	8
	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-B
	Special provisions	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

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

Product name	Group
mecoprop-P-potassium	Not Applicable
MCPA	Not Applicable
diethylene glycol monobutyl ether	Not Applicable
hydrocarbons, C10, aromatics, >1% naphthalene	Not Applicable
fluroxypyr methylheptyl ester	Not Applicable
monoethanolamine	Not Applicable
nonylphenol, ethoxylated, propoxylated	Not Applicable
potassium hydroxide	Not Applicable
potassium acetate	Not Applicable
water	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
mecoprop-P-potassium	Not Applicable
MCPA	Not Applicable
diethylene glycol monobutyl ether	Not Applicable
hydrocarbons, C10, aromatics, >1% naphthalene	Not Applicable
fluroxypyr methylheptyl ester	Not Applicable
monoethanolamine	Not Applicable
nonylphenol, ethoxylated, propoxylated	Not Applicable
potassium hydroxide	Not Applicable
potassium acetate	Not Applicable
water	Not Applicable

SECTION 15 Regulatory information

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

This substance is to be managed using the conditions specified in an applicable Group Standard

HSR Number	Group Standard
HSR100501	Not Available

Please refer to Section 8 of the SDS for any applicable tolerable exposure limit or Section 12 for environmental exposure limit.

mecoprop-P-potassium is found on the following regulatory lists

- New Zealand Approved Hazardous Substances with controls
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
- New Zealand Inventory of Chemicals (NZIoC)
- New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 1 Quantity limits for dangerous goods

MCPA is found on the following regulatory lists

- International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
- International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
- International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
- New Zealand Approved Hazardous Substances with controls
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
- New Zealand Inventory of Chemicals (NZIoC)
- New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 1 Quantity limits for dangerous goods
- New Zealand Workplace Exposure Standards (WES)

diethylene glycol monobutyl ether is found on the following regulatory lists

- New Zealand Approved Hazardous Substances with controls
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
- New Zealand Inventory of Chemicals (NZIoC)

hydrocarbons, C10, aromatics, >1% naphthalene is found on the following regulatory lists

- International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
- New Zealand Inventory of Chemicals (NZIoC)
- New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 1 Quantity limits for dangerous goods

fluroxypyr methylheptyl ester is found on the following regulatory lists

- New Zealand Approved Hazardous Substances with controls
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
- New Zealand Inventory of Chemicals (NZIoC)
- New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 1 Quantity limits for dangerous goods

monoethanolamine is found on the following regulatory lists

- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
- New Zealand Inventory of Chemicals (NZIoC)
- New Zealand Workplace Exposure Standards (WES)

nonylphenol, ethoxylated, propoxylated is found on the following regulatory lists

- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
- New Zealand Inventory of Chemicals (NZIoC)

potassium hydroxide is found on the following regulatory lists

- New Zealand Approved Hazardous Substances with controls
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
- New Zealand Inventory of Chemicals (NZIoC)
- New Zealand Workplace Exposure Standards (WES)

potassium acetate is found on the following regulatory lists

- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
- New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
- New Zealand Inventory of Chemicals (NZIoC)

water is found on the following regulatory lists

- New Zealand Inventory of Chemicals (NZIoC)

Additional Regulatory Information

Not Applicable

Hazardous Substance Location

Subject to the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Quantity (Compliance Certificate)	Quantity (Compliance Certificate - Farms >4 ha)
8.2B	250 kg or 250 L	3500 kg or 3500 L

Certified Handler

Subject to Part 4 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Class of substance	Quantities
Not Applicable	Not Applicable

Refer Group Standards for further information

Maximum quantities of certain hazardous substances permitted on passenger service vehicles

Subject to Regulation 13.14 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Gas (aggregate water capacity in mL)	Liquid (L)	Solid (kg)	Maximum quantity per package for each classification
8.2B	120	1	3	

Tracking Requirements

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIC / Australia Non-Industrial Use	No (fluroxypr methylheptyl ester)
Canada - DSL	No (mecoprop-P-potassium; MCPA; fluroxypr methylheptyl ester)
Canada - NDSL	No (mecoprop-P-potassium; diethylene glycol monobutyl ether; hydrocarbons, C10, aromatics, >1% naphthalene; fluroxypr methylheptyl ester; monoethanolamine; nonylphenol, ethoxylated, propoxylated; potassium hydroxide; potassium acetate; water)
China - IECSC	No (mecoprop-P-potassium; fluroxypr methylheptyl ester)
Europe - EINEC / ELINCS / NLP	No (nonylphenol, ethoxylated, propoxylated)
Japan - ENCS	No (fluroxypr methylheptyl ester; nonylphenol, ethoxylated, propoxylated)
Korea - KECI	No (mecoprop-P-potassium)
New Zealand - NZIoC	Yes
Philippines - PICCS	No (mecoprop-P-potassium; fluroxypr methylheptyl ester)
USA - TSCA	TSCA Inventory 'Active' substance(s) (MCPA; diethylene glycol monobutyl ether; hydrocarbons, C10, aromatics, >1% naphthalene; monoethanolamine; nonylphenol, ethoxylated, propoxylated; potassium hydroxide; potassium acetate; water); No (mecoprop-P-potassium; fluroxypr methylheptyl ester)
Taiwan - TCSI	No (mecoprop-P-potassium)
Mexico - INSQ	No (mecoprop-P-potassium; nonylphenol, ethoxylated, propoxylated)
Vietnam - NCI	Yes
Russia - FBEPH	No (mecoprop-P-potassium; fluroxypr methylheptyl ester; nonylphenol, ethoxylated, propoxylated)
UAE - Control List (Banned/Restricted Substances)	No (mecoprop-P-potassium; MCPA; diethylene glycol monobutyl ether; hydrocarbons, C10, aromatics, >1% naphthalene; fluroxypr methylheptyl ester; monoethanolamine; nonylphenol, ethoxylated, propoxylated; potassium hydroxide; potassium acetate; water)
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	03/09/2026
Initial Date	25/09/2019

SDS Version Summary

Version	Date of Update	Sections Updated
13.1	18/12/2025	Hazards identification - Classification
14.1	03/09/2026	Toxicological information - Acute Health (eye), Toxicological information - Acute Health (inhaled), Toxicological information - Acute Health (skin), Toxicological information - Acute Health (swallowed), First Aid measures - Advice to Doctor, Toxicological information - Chronic Health, Hazards identification - Classification, Disposal considerations - Disposal, Exposure controls / personal protection - Engineering Control, Ecological Information - Environmental, Firefighting measures - Fire Fighter (extinguishing media), Firefighting measures - Fire Fighter (fire/explosion hazard), Firefighting measures - Fire Fighter (fire fighting), Firefighting measures - Fire Fighter (fire incompatibility), First Aid measures - First Aid (eye), First Aid measures - First Aid (inhaled), First Aid measures - First Aid (skin), First Aid measures - First Aid (swallowed), Handling and storage - Handling Procedure, Composition / information on ingredients - Ingredients, Stability and reactivity - Instability Condition, Exposure controls / personal protection - Personal Protection (other), Exposure controls / personal protection - Personal Protection (Respirator), Exposure controls / personal protection - Personal Protection (eye), Exposure controls / personal protection - Personal Protection (hands/feet), Accidental release measures - Spills (major), Accidental release measures - Spills (minor), Handling and storage - Storage (storage incompatibility), Handling and storage - Storage (storage requirement), Handling and storage - Storage (suitable container), Transport information - Transport, Transport Information

Other information

Ingredients with multiple cas numbers

Name	CAS No
mecoprop-P-potassium	66423-05-0, 1929-86-8
fluroxypyr methylheptyl ester	81406-37-3, 69377-81-7, 154486-27-8, 94133-62-7, 35622-80-1
monoethanolamine	141-43-5, 2122854-11-7, 9007-33-4
nonylphenol, ethoxylated, propoxylated	37251-69-7, 68855-07-2
potassium acetate	127-08-2, 134092-62-9, 325477-95-0, 662141-29-9

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

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

Definitions and abbreviations

- ▶ PC—TWA: Permissible Concentration-Time Weighted Average
- ▶ PC—STEL: Permissible Concentration-Short Term Exposure Limit
- ▶ IARC: International Agency for Research on Cancer
- ▶ ACGIH: American Conference of Governmental Industrial Hygienists
- ▶ STEL: Short Term Exposure Limit
- ▶ TEEL: Temporary Emergency Exposure Limit
- ▶ IDLH: Immediately Dangerous to Life or Health Concentrations
- ▶ ES: Exposure Standard
- ▶ OSF: Odour Safety Factor
- ▶ NOAEL: No Observed Adverse Effect Level
- ▶ LOAEL: Lowest Observed Adverse Effect Level
- ▶ TLV: Threshold Limit Value
- ▶ LOD: Limit Of Detection
- ▶ OTV: Odour Threshold Value
- ▶ BCF: BioConcentration Factors
- ▶ BEI: Biological Exposure Index
- ▶ 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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