Dynamo®
Lonza NZ Limited

Chemwatch Hazard Alert Code: **3**

Chemwatch: **5458-45**
Version No: **4.1.1.1**
Safety Data Sheet according to the Health and Safety at Work (Hazardous Substances) Regulations 2017

Issue Date: **19/03/2021**
Print Date: **31/03/2021**
L.GHS.NZL.EN

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

Product Identifier

Product name	Dynamo®
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	CORROSIVE LIQUID, N.O.S. (contains flumetsulam and bentazone)
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.

Details of the supplier of the safety data sheet

Registered company name	**Lonza 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.lonza.co.nz
Email	office-newplymouth@lonza.com

Emergency telephone number

Association / Organisation	**Lonza NZ Limited**
Emergency telephone numbers	**0800 243 622**
Other emergency telephone numbers	**+64 4 917 9888 (International)**

SECTION 2 Hazards identification

Classification of the substance or mixture

Classification [1]	Corrosive to Metals Category 1, Acute Toxicity (Oral) Category 4, Acute Toxicity (Dermal) Category 4, Skin Corrosion/Irritation Category 1C, Skin Sensitizer Category 1, Serious Eye Damage/Eye Irritation Category 1, Acute Toxicity (Inhalation) Category 4, Reproductive Toxicity Category 1, Specific target organ toxicity - single exposure Category 1, Chronic Aquatic Hazard Category 1, Acute Terrestrial Hazard Category 3, Acute Vertebrate Hazard Category 2
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*
Determined by Chemwatch using GHS/HSNO criteria	8.1A, 6.1D (dermal), 6.1D (inhalation), 6.1D (oral), 8.2C, 8.3A, 6.5B (contact), 6.8A, 6.9A, 9.1A, 9.2C, 9.3B

Label elements

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

Hazard statement(s)

H290	May be corrosive to metals.
H302	Harmful if swallowed.
H312	Harmful in contact with skin.
H314	Causes severe skin burns and eye damage.
H317	May cause an allergic skin reaction.
H332	Harmful if inhaled.
H360	May damage fertility or the unborn child.

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H370	Causes damage to organs.
H410	Very toxic to aquatic life with long lasting effects.
H423	Harmful to the soil environment
H432	Toxic to terrestrial vertebrates.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P234	Keep only in original packaging.
P270	Do not eat, drink or smoke when using this product.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P311	IF exposed or concerned: Call a POISON CENTER/doctor/physician/first aider.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P391	Collect spillage.
P302+P352	IF ON SKIN: Wash with plenty of water.
P363	Wash contaminated clothing before reuse.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P390	Absorb spillage to prevent material damage.
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.

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
25057-89-0	30-60	bentazone
68-12-2	10-20	N,N-dimethylformamide
9007-33-4	10-20	ethanolamine
98967-40-9	<3	flumetsulam
Not Available	balance	Ingredients determined not to be hazardous

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.

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Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay. ▸ Inhalation of vapours or aerosols (mists, fumes) may cause lung oedema. ▸ Corrosive substances may cause lung damage (e.g. lung oedema, fluid in the lungs). ▸ As this reaction may be delayed up to 24 hours after exposure, affected individuals need complete rest (preferably in semi-recumbent posture) and must be kept under medical observation even if no symptoms are (yet) manifested. ▸ Before any such manifestation, the administration of a spray containing a dexamethasone derivative or beclomethasone derivative may be considered. **This must definitely be left to a doctor or person authorised by him/her.** (ICSC13719)
Ingestion	▸ For advice, contact a Poisons Information Centre or a doctor at once. ▸ Urgent hospital treatment is likely to be needed. ▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ 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 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]
Depending on the degree of exposure to dimethylformamide, preplacement and periodic medical examination is desirable, especially evaluating the liver and kidney functions, and possible skin effects.

In cases of recent sulfonamide overdose the stomach should be emptied by aspiration and lavage. If kidney function is adequate, a saline purgative, such as sodium sulfate, 30 g in 250 ml water, may be given to promote peristalsis and elimination of sulfonamide in the urine may be assisted by giving alkalies, such as sodium bicarbonate and increasing fluid intake. Severe crystalluria may require ureteric catheterisation and irrigation with warm 2.5% sodium bicarbonate solution. Treatment should be continued until it can be assumed that the sulfonamide has been eliminated. The majority of sulfonamides are metabolised to acetylated derivatives which retain the toxicity of the parent compound and thus may indicate more active removal when adverse effects are very severe. Active measures may include forced diuresis, peritoneal dialysis and charcoal haemoperfusion.

[*Martindale: The Extra Pharmacopoeia, 28th Ed.*]

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

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

Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Use fire fighting procedures suitable for surrounding area. ▸ **Do not** approach containers suspected to be hot.

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	▸ 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 (CO_2) nitrogen oxides (NOx) sulfur oxides (SOx) 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	Environmental hazard - contain spillage. ▸ 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	Environmental hazard - contain spillage. ▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by all means available, spillage from entering drains or water courses. ▸ Consider evacuation (or protect in place). ▸ No smoking, naked lights or ignition sources. ▸ Increase ventilation. ▸ Stop leak if safe to do so. ▸ Water spray or fog may be used to disperse / absorb vapour. ▸ Contain or absorb spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ 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	Alkanolamines and iron may produced unstable complexes. Monoethanolamine (MEA) and iron form a trisethanolamino-iron complex. This material may spontaneously decompose at temperatures between 130 and 160 degrees C. and is suspected of causing a fire in a nearly empty storage tank containing a "heel" of MEA in contact with carbon steel coils. If steam coil heating is used, low pressure steam in stainless steel coils should be considered. Drum heating should also be reviewed and, where possible, temperatures should be maintained below 130 degrees C. ▸ **DO NOT** allow clothing wet with material to stay in contact with skin ▸ Avoid all personal 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	▸ **DO NOT** store near acids, or oxidising agents ▸ No smoking, naked lights, heat or ignition sources. ▸ Store in original containers. ▸ Keep containers securely sealed. ▸ No smoking, naked lights or ignition sources. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers.

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	▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	HDPE Jerry Can. ▸ Glass container is suitable for laboratory quantities 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. ▸ Polyethylene or polypropylene container. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ Avoid reaction with oxidising agents, bases and strong reducing agents. ▸ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

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X — Must not be stored together
0 — May be stored together with specific preventions
+ — May be stored together

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)	N,N-dimethylformamide	Dimethylformamide	10 ppm / 30 mg/m3	Not Available	Not Available	skin-Skin absorption
New Zealand Workplace Exposure Standards (WES)	ethanolamine	Ethanolamine (2-Aminoethanol)	3 ppm / 7.5 mg/m3	15 mg/m3 / 6 ppm	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
N,N-dimethylformamide	Not Available	Not Available	Not Available
ethanolamine	6 ppm	170 ppm	1,000 ppm

Ingredient	Original IDLH	Revised IDLH
bentazone	Not Available	Not Available
N,N-dimethylformamide	500 ppm	Not Available
ethanolamine	30 ppm	Not Available
flumetsulam	Not Available	Not Available

Occupational Exposure Banding

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

MATERIAL DATA

Exposure controls

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

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	▸ Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area. ▸ Work should be undertaken in an isolated system such as a "glove-box" . Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system. ▸ Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within. ▸ Open-vessel systems are prohibited. ▸ Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation. ▸ Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system. ▸ For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▸ Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas). ▸ Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air. ▸ Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed.
Personal protection	
Eye and face protection	▸ Safety glasses with side shields. ▸ Chemical goggles. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	▸ 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. ▸ Aprotic solvents may greatly promote the toxic properties of solutes because of their unique ability to penetrate synthetic rubber protective gloves and the skin (butyl rubber gloves are reported to be more satisfactory than others
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.

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Recommended material(s)

GLOVE SELECTION INDEX

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

Material	CPI
BUTYL	A
BUTYL/NEOPRENE	A
CPE	C
HYPALON	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
SARANEX-23	C
SARANEX-23 2-PLY	C
TEFLON	C
VITON	C
VITON/CHLOROBUTYL	C

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

Respiratory protection

Type 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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Hazy brown liquid solution concentrate with a characteristic odour; mixes with water.

Physical state	Liquid	**Relative density (Agua= 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)	6.5-7.5	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	>105	**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

SECTION 10 Stability and reactivity

Reactivity	See section 7

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation. Inhalation and/or ingestion, of dimethylformamide (DMF), can lead to headache, dizziness, nausea, vomiting, anorexia and abdominal spasm. The use of alcoholic beverages accentuates the toxic effects and intolerance to alcohol can occur up to 4 days after exposure. Case reports in workers acutely exposed to DMF confirm that the liver is the target organ, with hepatic effects and associated disorders of the digestive system being reported. Symptoms include abdominal pain, anorexia, incoordination, and jaundice, as well as nausea, vomiting, and diarrhoea; nasal and skin irritation have also been reported Single exposures of 6 hours duration to air saturated with DMF vapour (approximately 5000 ppm) at room temperature killed rats and produced injury to lungs, liver and kidneys. At 2500 ppm rats were killed after five 6-hour exposures. At 20 ppm dogs survived more than 100 exposure periods of 6 hours and showed no gross pathology but did show a functional drop in systolic blood pressure. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact.
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. The material can produce chemical burns within the oral cavity and gastrointestinal tract following ingestion. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing. Sulfonamides and their derivatives may precipitate in kidney tubules causing extensive damage. Haemolytic anaemia may also result from use or exposure. Overdose may cause acidosis or hypoglycaemia with confusion and coma resulting. Hypersensitivity reactions may occur in predisposed individuals including those who have been sensitised by topical application. Deaths associated with therapies based on sulfonamide appear to be a result of hypersensitivity reaction, agranulocytosis, aplastic anaemia, other blood dyscrasias and renal and hepatic failure. Doses of 2 to 5 gms have produced toxicity and fatalities. Pathological findings include crystalluria, and necrotic or inflammatory lesions of the heart, liver, kidneys, bone marrow or other organs. Sulfonamides may damage the stem cell which acts as the precursor to components of the blood. Loss of the stem cell may result in pancytopenia (a reduction in the number of red and white blood cells and platelets) with a latency period corresponding to the lifetime of the individual blood cells. Granulocytopenia (a reduction in granular leukocytes) develops within days and thrombocytopenia (a disorder involving platelets), within 1-2 weeks, whilst loss of erythrocytes (red blood cells) need months to become clinically manifest. Aplastic anaemia develops due to complete destruction of the stem cells. Sulfonamides cross the placental barrier, are excreted in the breast milk and may produce adverse effects in the foetus/ embryo and newborn including agranulocytosis, haemolytic anaemia, jaundice and kernicterus. 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).
Skin Contact	Skin contact with the material may be harmful; systemic effects may result following absorption. The material can produce chemical burns following direct contact with the skin. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact. DMF is extensively absorbed through the skin, its metabolism and kinetics are well known, and urinary metabolites exist that can be accurately measured. As a result, biological monitoring has been extensively used in the assessment of the absorbed amounts in occupationally exposed populations. The metabolite most often analysed is *N*-methylformamide (NMF) In one case of accidental exposure, to dimethylformamide. the patient showed itching, hyperaemia, abdominal pain, vomiting and increased blood pressure. There are inconclusive reports of human sensitisation Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
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. Ophthalmic solutions containing sulfonamides are reported to produce local irritation, reactive hyperaemia, burning and transient stinging, blurred vision and temporary impairment of depth perception. Hypersensitivity reactions may occur in predisposed individuals. Possible eye changes produced by phototoxic agents such as the sulfonamides include kerato-conjunctivitis or corneal and lens opacities.
Chronic	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. On the basis, primarily, of animal experiments, the material may be regarded as carcinogenic to humans. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in cancer on the basis of: - appropriate long-term animal studies - other relevant information 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.

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There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of:
- clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.
Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

There is some evidence to provide a presumption that human exposure to the material may result in impaired fertility on the basis of: some evidence in animal studies 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 is not a secondary non-specific consequence of other toxic effects.
Sulfonylureas, imidazolinones, sulfonoanilides and triazolo-pyrimidines, as herbicides, are used extensively because of their wide-spectrum effects on weeds and their low toxicity to mammals. The effects of these herbicides on plants, micro-algae and bacteria are due to the inhibition of acetolactate synthase (ALS) involved in the synthesis of acetolactic and butyric acids, which are the precursors of the branched-chain amino acids: isoleucine, leucine and valine.
Mammals also produce these precursor amino-acids using ALS so the potential for toxic effects is apparent though not evident from many studies.

Repeated ingestion of sulfonamides used for therapeutic purposes has caused nausea, vomiting, abdominal pain, diarrhoea, anorexia, stomatitis, impaired folic acid absorption, exacerbation of porphyria, acidosis, liver injury with jaundice and hypoprothrombinemia, and pancreatitis. Hepatitis has been reported and may be fatal. Renal effects are often prominent and may include crystalluria, haematuria, proteinuria, pain and frequent urination, necrosis of the tubules, nephritic syndrome, and toxic necrosis with oliguria or anuria with azotemia. Neurologic effects include headache, drowsiness, insomnia, vertigo, tinnitus, hearing loss, mental depression, hallucinations, ataxia, muscular paralysis, peripheral neuropathy, transient lesions of the posterior spinal column, transverse myelitis, convulsions and unconsciousness. Haematological effects include eosinophilia, thrombocytopenia, leukopenia, neutropenia, agranulocytosis, pancytopenia, megoblastic anaemia, Heinz body anaemia and aplastic anaemia; petechiae and purpura may result. Acute haemolytic anaemia may also result (possibly as a result of hypersensitivity reactions) with people of African descent apparently more susceptible than Europeans - glucose-6-phosphate deficiency also appears to be a factor. Methaemoglobinaemia, sulfhaemoglobinaemia and cyanosis may also occur. Ocular effects may include acute transient myopia, keratitis and conjunctivitis with inflammation and chemosis accompanied by swelling of the lids and in more severe cases, photophobia. Cross-sensitivity amongst the sulfonamides is common and allergic reaction may occur following systemic use or topical application. Sensitisation may produce generalised skin eruptions, urticaria and pruritus. Stevens-Johnson syndrome; a severe form of erythema multiforme associated with wide-spread lesions of the skin, mucous membranes and which may be fatal in about 25% of cases, has occurred in patients treated with sulfonamides. This syndrome may produce conjunctival and corneal scarring, serum sickness, periorbital oedema, angioedema, arthritis, arthralgia, allergic myocarditis, decreased pulmonary function and eosinophilic pneumonia. Other effects of long-term therapy include fever, chills, alopecia, vasculitis, lupus erythematosus, oligospermia, infertility, hypothyroidism and on occasion, goiter and diuresis.

More severe responses to treatment include irreversible neuromuscular and central nervous system changes and fibrosing alveolitis. During sulfonamide treatment, direct exposure to sunlight should be avoided as photosensitisation dermatitis may develop. This form of phototoxic dermatitis may be contrasted to photoallergic dermatitis produced by specific sensitising agents through immunological intervention. Phototoxic reactions have been described following contact, ingestion or injection of causal agents. The chemical may reach the skin by the circulatory system following ingestion or following parenteral administration. The actual skin changes vary with the agent and circumstances of the exposure. Swelling and redness (erythema) frequently occur, and blistering may also result; increased skin temperature and pruritus may follow. This is analagous to irritant contact dermatitis and occurs immediately following contact.

Hyperpigmentation may also follow the reaction. Photodermatitis of this type requires activation of a chemical substance on the skin surface by UV radiation (290 to 490 nm wavelength) for its clinical expression. In all cases, inflammation develops on the body surfaces normally exposed to sunlight (dorsal hands, arms, neck, face), provided that the responsible photosensitiser also contacts the anatomic areas. Covered skin, the eyelids, submental chin and upper ears covered by hair, are characteristically spared. Phototoxic reactions, analogous to irritant contact dermatitis, are typically accompanied by immediate burning, stinging or "smarting" of the skin shortly following sun exposure, and clinical inflammation appears more like an acute sunburn than an eczematous dermatitis. Photoallergic dermatitis may result from contact with the material; this is characterised by an increased reactivity of the skin to ultra- violet (UV) and/or visible radiation produced by a chemical agent on an immunological basis and occurs after a latent period of days or months. This type of response can be elicited only in individuals who have been previously allergically sensitised to the chemical agent and appropriate radiation.

Photoallergic dermatitis is relatively rare (certainly more so than phototoxic dermatitis produced by non-immunological principals) and presents, clinically, as an eczematous dermatitis in sun-exposed areas (distinguishing it from phototoxic dermatitis which is analogous to contact irritant dermatitis and produces swelling, redness and even blistering); photoallergic dermatitis may eventually spread to areas covered by clothes. Lichenification (thickening with increased skin markings) and chronic pigmentary changes may also develop. Photoallergic reactions may sometimes be followed by a persistent state of light reactivity (persistent light reactor) where clinical dermatitis recurs following exposure to sunlight alone, in the absence of the original initiating chemical. Studies in rats have shown that long-term administration of sulfonamides may produce thyroid malignancies; rats, however, appear to be more susceptible to the goiterogenic effects of sulfonamides than do other animal species. Sulfonamides may cause kernicterus in the neonate and their use is not recommended during pregnancy. Studies in rats and mice given high oral doses have shown that certain sulfonamides cause a significant incidence of cleft palate and other bony abnormalities in the foetus.

Prolonged or chronic exposure to alkanolamines may result in liver, kidney or nervous system injury. Repeated inhalation may aggravate asthma and inflammatory or fibrotic pulmonary disease.

Results of repeated exposure tests with diethanolamine (DEA) in laboratory animals include anaemia (rats) and effects on the kidneys (rats and mice) and liver (mice). DEA produces nervous system injury in dogs and rats. Heart and salivary gland lesions have also been seen in mice treated cutaneously with DEA and in mice receiving DEA in drinking water. Rats given high doses of DEA developed anaemia and testicular lesions.

Exaggerated doses of DEA produced heart and nervous system effects in other animals. Changes in other organs were judged to be secondary due to the poor health of animals subjected to extremely high doses of DEA. Rats, rabbits and guinea pigs exposed to high vapour concentrations of volatile monoethanolamine (MEA) (up to 1250 ppm) for periods of up to 5 weeks developed pulmonary, hepatic and renal lesions. Dogs, rats and guinea pigs exposed to 100 ppm MEA for 30 days, became apathetic and developed poor appetites. Animal tests also indicate that inhalation exposure to MEA may result in nervous system injury. All species exposed to airborne MEA experienced dermal effects, varying from ulceration to hair loss probably resulting from contact with the cage.

An increased incidence of skeletal variations, suggestive of a slight developmental delay was seen in the foetuses of rats given 1500 mg/kg/day DEA cutaneously; this also produced significant maternal toxicity. No foetal malformations, however, were seen in rats nor in rabbits receiving identical treatment. The foetus of rats given high doses of MEA by gavage, showed an increased rate of embryofoetal death, growth retardation, and some malformations including hydronephrosis and hydroureter. The high doses required to produce these effects bring into question the relevance of this finding to humans. There is some evidence that embryofoetotoxicity and teratogenicity does not occur in rats when MEA is administered by dermal application to the mother.

The National Toxicology Program (NTP) concluded that there is clear evidence of liver tumours and some evidence of kidney tumours in mice exposed dermally to DEA over their lifetime. Chronic skin painting studies in mice of both sexes produced liver tumours and an increased incidence of kidney tumours in male mice. The significance of these findings to humans is unclear as DEA is neither genotoxic, mutagenic nor

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clastogenic, and did not induce tumours in rats or transgenic mice similarly treated. Alkanolamines (especially those containing a secondary amine moiety) may react with nitrites or other nitrosating agents to form carcinogenic N-nitrosamines. Alkanolamines are metabolised by biosynthetic routes to ethanolamine and choline and incorporated into phospholipids. They are excreted predominantly unchanged with a half-life of approximately one week. In the absence of sodium nitrite, no conversion to carcinogenic N-nitrosamines was observed.

Diethanolamine competitively inhibits the cellular uptake of choline, in vitro, and hepatic changes in choline homeostasis, consistent with choline deficiency, are observed in vivo.

Many amines are potent skin and respiratory sensitisers and certain individuals especially those described as "atopic" (i.e. those predisposed to asthma and other allergic responses) may show allergic reactions when chronically exposed to alkanolamines.

In a study with coconut diethanolamide, the National Toxicology Program (Technical Report Series 479), showed clear evidence of carcinogenic activity in male B6C3F1 mice based on increased incidences of hepatic and renal tubule neoplasms and in female B6C3F1 mice based on increased incidences of hepatic neoplasms. There was equivocal evidence of carcinogenic activity in female F344/N rats based on a marginal increase in the incidence of renal tube neoplasms. These increases were associated with the concentration of free diethanolamine present as a contaminant in the diethanolamine condensate. Exposure to rats to coconut oil diethanolamine condensate by dermal application in ethanol for 2 years resulted in epidermal hyperplasia, sebaceous gland hyperplasia, hyperkeratosis and parakeratosis in males and females and ulcer in females at the site of application. There were increases in the incidences of chronic inflammation, epithelial hyperplasia, and epithelial ulcer in the forestomach of female rats. The severity of nephropathy in dosed female rats were increased. Exposure of mice to coconut oil diethanolamine condensate by dermal application for 2 years resulted in increased incidences of eosinophilic foci of the liver in males. Increased incidences of epidermal hyperplasia, sebaceous gland hyperplasia, and hyperkeratosis in males and females, ulcer in males, and parakeratosis and inflammation in females at the site of application and of follicular cell hyperplasia in the thyroid gland of males and females, were chemical related.
Liver injury has been produced in cats after prolonged inhalation of 100 ppm dimethylformamide (DMF) (but not rats).
Leather tannery and airframe repair shop workers exposed to DMF and a number of other chemicals showed an increased number of testicular cancers above the number of expected cases. The available data however does not conclusively establish DMF as a carcinogen.
The literature contains citations indicating DMF increases the rate of embryonic mortalities in animals at high maternal doses.

Dynamo®	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

bentazone	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: 1100 mg/kg[2]	Eye (rabbit): moderate *
	Inhalation(Rat) LC50; 5.1 mg/L4[2]	Skin (rabbit): moderate *
	Oral(Dog) LD50; 450 mg/kg[2]	

N,N-dimethylformamide	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >500 mg/kg[2]	Eye (rabbit): 20 mg (open)
	Inhalation(Rat) LC50; >5.85 mg/l4[1]	Eye (rabbit):100mg(rinsed)-SEVERE
	Oral(Mouse) LD50; >5000 mg/kg[2]	Skin (human): 100%/24h - mild
		Skin (rabbit): 10 mg/24h (open)

ethanolamine	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >=2.412<=2.775 mg/kg[1]	Eye (rabbit): 0.76 mg - SEVERE
	Inhalation(Guinea) LC50; ~0.145 mg/l4[2]	Skin (rabbit):505 mg open-moderate
	Oral(Rat) LD50; 1.049 mg/kg[1]	

flumetsulam	**TOXICITY**	**IRRITATION**
	#LC50_Inhal >1.2 mg/l4[2]	Eye (rabbit): slight *
	Dermal (rabbit) LD50: >2000 mg/kg[1]	
	Oral(Rat) LD50; >5000 mg/kg[1]	

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*

BENTAZONE

The following information refers to contact allergens as a group and may not be specific to this product.
Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
for bentazone:
NOEL (1 y) for dogs 13.1 mg/kg: (2 y) for rats 10 mg/kg:
(90 d) for rats 25, dogs 10 mg/kg: (78 weeks) for mice 12 mg/kg
ADI 0.1 mg/kg *
Toxicity Class WHO III; EPA III *
Metabolism studies in rats showed that bentazone is poorly metabolised. The parent compound was the predominant metabolite, with 6-hydroxy-bentazone identified as a minor metabolite and 8-hydroxy-bentazone found in trace amounts.
Metabolism studies in lactating goats and hens showed that the major residue component in meat, milk and eggs was the parent bentazone with small amounts of 6- or 8-hydroxy-bentazone and their glucuronide and sulfate conjugates.
Bentazone is more acutely toxic to rats than are its two hydroxylated metabolites when given by the oral route. The acute oral LD50 of technical-grade bentazone was estimated to be 1800 mg/kg of body weight in males and 1500 mg/kg of body weight in females. The acute oral LD50 value for 6- and 8-hydroxybentazone was 5000 mg/kg of body weight. WHO has classified bentazone as slightly hazardous.

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	The two studies described below indicate that 8-hydroxybentazone does not have the anticoagulant and diuretic effects of bentazone at the doses tested and has less systemic toxicity than the parent compound under the test conditions. No data were available on the short-term toxicity of 6-hydroxybentazone. Rats received technical-grade bentazone in the diet at concentrations of 0, 400, 1200 or 3600 mg/kg for 13 weeks. The body weights of females were decreased and were statistically significantly different from those of controls at 3600 mg/kg from week 10 onward. Examination of haematological parameters indicated statistically significant increases in prothrombin time and partial thromboplastin time in males at 3600 mg/kg in comparison with controls. Bentazone had a diuretic effect in animals of both sexes, reaching statistical significance at 3600 mg/kg. The NOAEL for systemic toxicity was 1200 mg/kg (equal to 78 mg/kg of body weight per day) on the basis of statistically significant decreased body weights in females throughout the latter part of the treatment, increased prothrombin time and partial thromboplastin time in males, increased output of urine with decreased specific gravity in animals of both sexes, and some degree of kidney hypertrophy in both males and females at 3600 mg/kg, equal to 240 mg/kg of body weight per day. Rats received 8-hydroxybentazone in the diet at concentrations of 0, 400, 1200 or 3600 mg/kg for 3 months. No compound-related effects were observed on body weights, clinical signs, food consumption, haematological, clinical chemical or urinary parameters, clotting time, organ weights or gross or histopathological appearance. The NOAEL was 3600 mg/kg (equal to 260 mg/kg of body weight per day), the highest dose tested. No developmental toxicity was observed in pregnant rats that received 8-hydroxybentazone by gavage at 0, 40, 100 or 250 mg/kg of body weight per day on days 6-15 of gestation. The NOAEL for developmental toxicity was 250 mg/kg of body weight per day, the highest dose tested. Bentazone, 6-hydroxybentazone and 8-hydroxybentazone did not induce reverse mutation in bacteria, and 8-hydroxybentazone did not induce gene mutation in mammalian cells or micronucleus formation in mice in vivo. It was concluded that neither bentazone nor its metabolites are genotoxic. The following two studies of developmental toxicity indicate that bentazone has effects at doses below a maternally toxic dose, whereas 8-hydroxybentazone had no developmental or maternal toxicity at any of the doses tested. Pregnant rats received technical-grade bentazone by gavage at 0, 40, 100 or 250 mg/kg of body weight per day on days 6-15 of gestation. The NOAEL for maternal toxicity was 250 mg/kg of body weight per day, the highest dose tested. The NOAEL for developmental toxicity was 100 mg/kg of body weight per day on the basis of significantly decreased mean fetal weights and delays in tissue ossification, which reached statistical significance on a litter basis at the highest dose. The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. ***[* The Pesticides Manual, Incorporating The Agrochemicals Handbook, 10th Edition, Editor Clive Tomlin, 1994, British Crop Protection Council]***
N,N-DIMETHYLFORMAMIDE	For dimethylformamide (DMF): In humans, DMF is absorbed by inhalation and through the skin. After high exposures (up to 60 ppm) headaches, abdominal pain, nausea, vomiting, dizziness, elevated liver enzymes, and alcohol intolerance (facial flashing and palpitations) were seen. Case reports of testicular cancer in aircraft repair and leather tannery facilities failed to be confirmed in further studies. Reports of DNA and chromosomal damage in peripheral lymphocytes of subjects exposed to DMF either failed to take into account smoking as a confounder or coexposure to other chemicals. **Acute toxicity:** N,N-dimethylformamide (DMF) is of low acute toxicity in mammals: LD50 rat (oral) 3040 mg/kg bw, LC50 rat (inhalative, 4 h) > 5900 mg/m3, LD50 rat (dermal) > 3160 mg/kg bw. Main symptoms following exposure were apathy and staggering (oral) and irregular or intermittent respiration (inhalation). It was irritating to the eyes of rabbits but not irritating to the skin of rabbits and rats. DMF did not show a sensitizing potential when used as a vehicle in a local lymph node assay. **Repeat dose toxicity**: In repeated-dose toxicity studies in rats and mice with chronic exposure over 2 years (rats) or 18 months (mice) and subchronic exposure over 13 weeks by inhalation, or in rats treated by oral administration of DMF (90 day feeding study or administration by gavage for 28 days), the predominant target organ was the liver (NOAEC: chronic inhalation rat: 25 ppm (about 80 mg/m3), LOAEC: chronic inhalation mouse: 25 ppm (about 80 mg/m3); NOAEC: subchronic inhalation rat: 100 ppm, mouse: 400 ppm (about 300 mg/m3 and 1210 mg/m3, respectively); NOAEL: rat, 90 days 200 ppm (about 12 mg/kg bw/day), 28 days about 238 mg/kg bw/day). In a 13-week inhalation study with a limited number of Cynomolgus monkeys no treatment-related effects occurred (NOAEC: 500 ppm (about 1500 mg/m3)). **Genetic toxicity:** DMF does not induce chromosome aberrations or gene mutations in various test systems *in vivo* and *in vitro.* In addition, no increased tumor incidence was found in carcinogenicity studies in rats and mice that were exposed to 25, 100 and 400 ppm DMF (about 80, 300, and 1210 mg/m3) by inhalation for 2 years or 18 months, respectively. **Reproductive and developmental toxicity:** Reproductive toxicity was observed at the presence of some general toxicity in a continuous breeding study in mice, when DMF was administered orally in the drinking water at doses of 1000, 4000 and 7000 ppm (about 219, 820 and 1455 mg/kg bw/day). The maximal tolerated dose for generalised toxicity was 1000 ppm (about 219 mg/kg bw/day) for the F0 and the F1 generation, thus a systemic NOAEL could not be determined. Significant reproductive toxicity (e.g. reduced fertility and fecundity characterized by reduced pregnancy and mating index (the latter one only in the high dose group), reduced number of litters, reduced average litter size and for the F1 parental males by effects on prostate weight and epididymal spermatozoa concentration, the latter finding only in the high dose group) and developmental toxicity (e.g. reduced survival and growth of pups, increase in craniofacial and sternebral malformations) occurred at 4000 ppm and above. At 1000 ppm, reduced pup weights were found in F2 pups. Thus 1000 ppm (about 219 mg/kg bw/day) was the NOAEL for reproductive and developmental toxicity in F0 and F1, and the LOAEL for developmental toxicity in F2. Developmental toxicity and teratogenicity occurred in rats and rabbits in various studies (inhalation, oral- or dermal administration) and in mice (oral administration). In rats embryo/ foetotoxicity and teratogenicity were mostly seen at maternally toxic doses, whereas in mice and in rabbits embryo-/foetotoxicity and teratogenicity occurred also at dose levels without maternal toxicity. However, the rabbit appeared to be the most sensitive species to the developmental toxic effects of DMF. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. **WARNING:** This substance has been classified by the IARC as Group 2A: Probably Carcinogenic to Humans.
ETHANOLAMINE	* 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.

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	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 sensitisers, 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** Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. 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. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
FLUMETSULAM	NOEL (mg/kg)*: mice >1000 mg/kg, female rats 500, male rats 1000, dogs 1000 Non-sensitising to skin (guinea pigs).* Non-teratogenic (dietary) in rats. * Non-mutagenic in the Ames test * * The Pesticide Manual ADI: 1.0 mg/kg/day NOEL: 100 mg/kg/day In laboratory testing, flumetsulam did not cause birth defects or other effects even at doses which caused toxic effects in the mother.In animal studies, this material did not interfere with reproduction. In vitro and animal genetic toxicity studies were negative The triazolopyrimidine herbicides have been comprehensively evaluated in guideline and GLP compliant toxicity studies required for the registration and authorization of pesticides in various geographies throughout the world. In general, they exhibit very low mammalian toxicity as assessed through acute, short-term, long-term (chronic), genotoxicity, reproduction, developmental, and neurotoxicity studies. In repeat-dose toxicity studies, the liver and kidneys have been identified as target organs with effects that were often adaptive in nature generally observed only at excessively high-dose levels. In addition, the triazolopyrimidines were shown to be rapidly absorbed and excreted, have a low potential for bioaccumulation, and in general are not extensively metabolized.
BENTAZONE & ETHANOLAMINE	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
N,N-DIMETHYLFORMAMIDE & ETHANOLAMINE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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

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

Continued...

bentazone	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96	Fish	>0.983mg/L	4
	NOEC(ECx)	4	Algae or other aquatic plants	0.002mg/L	4
	EC50	72	Algae or other aquatic plants	24mg/l	4
N,N-dimethylformamide	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	BCF	1344	Fish	0.3-0.8	7
	LC50	96	Fish	21.23mg/L	4
	NOEC(ECx)	336	Crustacea	0.03mg/L	4
	EC50	48	Crustacea	>100mg/l	1
	EC50	72	Algae or other aquatic plants	>1000mg/l	2
	EC50	96	Algae or other aquatic plants	>500mg/l	1
ethanolamine	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	48	Crustacea	65mg/l	1
	LC50	96	Fish	75mg/l	1
	EC50	72	Algae or other aquatic plants	15mg/l	1
	NOEC(ECx)	72	Algae or other aquatic plants	4mg/l	1
	EC50	96	Algae or other aquatic plants	80mg/l	2
flumetsulam	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50(ECx)	120	Algae or other aquatic plants	<0.001mg/L	4
	LC50	96	Fish	>3.898mg/L	4
	EC50	48	Crustacea	3.313-3.419mg/L	4
	EC50	96	Algae or other aquatic plants	10.685mg/l	4
Legend:	*Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*				

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 bentazone:
Kow 5.84 (pH 5), 0.35 (pH 7), 0.28 (pH 9)
Environmental fate:
Plants: Rapidly hydrolysed to derivatives of anthranilic acid with theprinciple metabolites being 6- and 8-hydroxy derivatives.
Soil and Water: In soil, short lived hydroxy compounds are formed at first. These are rapidly degraded. In sunlight, bentazone undergoes oxidation and dimerisation with loss of SO2.
Has low soil persistence. In freshly collected field soils aerobic DT50 (lab, 20 C) 13.6 d. In lab. degradation studies mostly with BBA-standard soils DT50 (average) 45 d.
DT50 (field, average) 12 d. Corresponding DT90 44d
Koc 13.3-176 ml/g (average 42 ml/g).
BCF : 19
The degradation rate of bentazone is significantly faster in the field than in laboratory studies. However, use of the estimated average field degradation half-time (DT50) of 12 days in simple models rather than the laboratory DT50 of 45 days, while reducing the observed potential leaching from “substantial” to “marginal,” still leaves cause for concern regarding leaching.
Under many field conditions, degradation of bentazone will be complete in the upper soil layers. This is particularly true for dry conditions and will still hold for conditions of non-extreme rainfall. However, compounds with this high water solubility and these low soil adsorption characteristics are liable to leach under conditions of extreme rainfall (such as storms shortly after application). Bentazone will be expected to pass both through the soil profile and via cracks to the underlying aquifer. Once outside the zone of biological action, there is no abiotic mechanism for its degradation. Some contamination of the groundwater would be expected to occur under these circumstances. This potential has been confirmed by some reports of bentazone in groundwater.
Ecotoxicity:
Birds: Acute oral LD50 for bobwhite quail 1140 mg/kg
Dietary LC50 for rainbow trout and bluegill sunfish >100 mg/l
Fish LC50 (48 h): 65 mg/L
Bees: non-toxic: LD50 (oral) >100 ug/bee
Daphnia EC50 (48 h) 125 mg/l
Other aquatic spp. EC50 (72 h) for green algae (Ankistrodesmus) 62 mg/l
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
N,N-dimethylformamide	LOW	LOW
ethanolamine	LOW	LOW
flumetsulam	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
N,N-dimethylformamide	LOW (BCF = 1.2)
ethanolamine	LOW (LogKOW = -1.31)
flumetsulam	LOW (LogKOW = 2.9441)

Continued...

Mobility in soil

Ingredient	Mobility
N,N-dimethylformamide	MEDIUM (KOC = 2.411)
ethanolamine	HIGH (KOC = 1)
flumetsulam	LOW (KOC = 28690)

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)

UN number	1760
UN proper shipping name	CORROSIVE LIQUID, N.O.S. (contains flumetsulam and bentazone)
Transport hazard class(es)	Class: 8 Subrisk: Not Applicable
Packing group	III
Environmental hazard	Environmentally hazardous

Continued...

Special precautions for user	Special provisions	223; 274
	Limited quantity	5 L

Air transport (ICAO-IATA / DGR)

UN number	1760	
UN proper shipping name	Corrosive liquid, n.o.s. * (contains flumetsulam and bentazone)	
Transport hazard class(es)	ICAO/IATA Class	8
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	8L
Packing group	III	
Environmental hazard	Environmentally hazardous	
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)

UN number	1760	
UN proper shipping name	CORROSIVE LIQUID, N.O.S. (contains flumetsulam and bentazone)	
Transport hazard class(es)	IMDG Class	8
	IMDG Subrisk	Not Applicable
Packing group	III	
Environmental hazard	Marine Pollutant	
Special precautions for user	EMS Number	F-A , S-B
	Special provisions	223 274
	Limited Quantities	5 L

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

Not Applicable

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

Product name	Group
bentazone	Not Available
N,N-dimethylformamide	Not Available
ethanolamine	Not Available
flumetsulam	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
bentazone	Not Available
N,N-dimethylformamide	Not Available
ethanolamine	Not Available
flumetsulam	Not Available

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
HSR100817	Not Available

bentazone 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)

N,N-dimethylformamide is found on the following regulatory lists

Continued...

Chemical Footprint Project - Chemicals of High Concern List
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 2A: Probably carcinogenic to humans
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)

ethanolamine 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)

flumetsulam 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)

Hazardous Substance Location

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

Hazard Class	Quantities
Not Applicable	Not Applicable

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
6.5A or 6.5B	120	1	3	
8.2C	120	1	3	

Tracking Requirements

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (bentazone; flumetsulam)
Canada - DSL	No (flumetsulam)
Canada - NDSL	No (bentazone; N,N-dimethylformamide; ethanolamine; flumetsulam)
China - IECSC	No (flumetsulam)
Europe - EINEC / ELINCS / NLP	No (flumetsulam)
Japan - ENCS	No (bentazone; flumetsulam)
Korea - KECI	No (flumetsulam)
New Zealand - NZIoC	Yes
Philippines - PICCS	No (flumetsulam)
USA - TSCA	No (flumetsulam)
Taiwan - TCSI	Yes
Mexico - INSQ	No (flumetsulam)
Vietnam - NCI	Yes
Russia - FBEPH	No (flumetsulam)
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 Other information

Revision Date	19/03/2021
Initial Date	10/03/2021

SDS Version Summary

Version	Issue Date	Sections Updated
3.1.1.1	16/03/2021	Acute Health (swallowed), Classification, Ingredients

Continued...

Version	Issue Date	Sections Updated
4.1.1.1	19/03/2021	Classification

Other information

Ingredients with multiple cas numbers

Name	CAS No
N,N-dimethylformamide	68-12-2, 15175-63-0, 15175-77-6, 33513-42-7, 114057-15-7
ethanolamine	141-43-5, 2122854-11-7, 9007-33-4

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

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

Definitions and abbreviations

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