

Dunlop Abamastic

Chemwatch Material Safety Data Sheet

Issue Date: 25-Jan-2013

A317LP

Hazard Alert Code: MODERATE

CHEMWATCH 15-5659

Version No:2.1.1.1

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Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

PRODUCT NAME

Dunlop Abamastic

PRODUCT USE

Water based premixed acrylic adhesive for fixing ceramic, slate tiles.

Used as per manufacturers directions.

SUPPLIER

Company: Ardex Australia Pty Ltd

Address:

20 Powers Road

Seven Hills

NSW, 2147

Australia

Telephone: 1800 224 070

Emergency Tel:1800 222 841

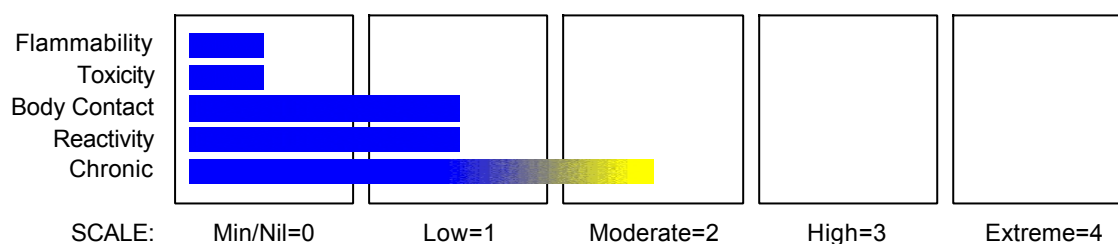
Fax: +61 2 9838 7817

Section 2 - HAZARDS IDENTIFICATION

STATEMENT OF HAZARDOUS NATURE

NON-HAZARDOUS SUBSTANCE. NON-DANGEROUS GOODS. According to NOHSC Criteria, and ADG Code.

CHEMWATCH HAZARD RATINGS



RISK

■ May produce discomfort of the eyes*.

■ Possible skin sensitiser*.

■ May be harmful to the foetus/ embryo*.

* (limited evidence).

SAFETY

• Avoid contact with skin.

• Avoid contact with eyes.

• Wear eye/face protection.

• Avoid exposure - obtain special instructions before use.

• In case of contact with eyes, rinse with plenty of water and contact Doctor or Poisons Information Centre.

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Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

NAME	CAS RN	%
ethylene glycol monobutyl ether	111-76-2	<0.9
biocide, unspecified		<1
non hazardous ingredients, unspecified		>60

Section 4 - FIRST AID MEASURES

SWALLOWED

- Immediately give a glass of water.
- First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

EYE

- If this product comes in contact with the eyes:
- Wash out immediately with fresh running water.
- Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.
- Seek medical attention without delay; if pain persists or recurs seek medical attention.
- Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

SKIN

- If skin contact occurs:
- Immediately remove all contaminated clothing, including footwear.
- Flush skin and hair with running water (and soap if available).
- Seek medical attention in event of irritation.

INHALED

- If fumes, aerosols or combustion products are inhaled remove from contaminated area.
- Other measures are usually unnecessary.

NOTES TO PHYSICIAN

Treat symptomatically.

Section 5 - FIRE FIGHTING MEASURES

EXTINGUISHING MEDIA

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

FIRE FIGHTING

- Alert Fire Brigade and tell them location and nature of hazard.
- Wear breathing apparatus plus protective gloves in the event of a fire.
- Prevent, by any means available, spillage from entering drains or water courses.
- Use fire fighting procedures suitable for surrounding area.
- DO NOT approach containers suspected to be hot.
- Cool fire exposed containers with water spray from a protected location.
- If safe to do so, remove containers from path of fire.
- Equipment should be thoroughly decontaminated after use.

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Section 5 - FIRE FIGHTING MEASURES

FIRE/EXPLOSION HAZARD

- Non combustible.
 - Not considered a significant fire risk, however containers may burn.
- Decomposition may produce toxic fumes of: carbon dioxide (CO₂), other pyrolysis products typical of burning organic material.
- May emit poisonous fumes.
- May emit corrosive fumes.

FIRE INCOMPATIBILITY

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

HAZCHEM

None

Section 6 - ACCIDENTAL RELEASE MEASURES

MINOR SPILLS

- Clean up all spills immediately.
- Avoid contact with skin and eyes.
- Wear impervious gloves and safety goggles.
- Trowel up/scrape up.
- Place spilled material in clean, dry, sealed container.
- Flush spill area with water.
- Clean up all spills immediately.
- Avoid breathing vapours/ aerosols or dusts and avoid contact with skin and eyes.
- Place in a suitable, labelled container for waste disposal.

MAJOR SPILLS

Minor hazard.

- Clear area of personnel.
- Alert Fire Brigade and tell them location and nature of hazard.
- Control personal contact with the substance, by using protective equipment as required.
- Prevent spillage from entering drains or water ways.
- Contain spill with sand, earth or vermiculite.
- Collect recoverable product into labelled containers for recycling.
- Absorb remaining product with sand, earth or vermiculite and place in appropriate containers for disposal.
- Wash area and prevent runoff into drains or waterways.
- If contamination of drains or waterways occurs, advise emergency services.

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

Section 7 - HANDLING AND STORAGE

PROCEDURE FOR HANDLING

- Avoid all personal contact, including inhalation.
- Wear protective clothing when risk of exposure occurs.
- Use in a well-ventilated area.
- Prevent concentration in hollows and sumps.

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Section 7 - HANDLING AND STORAGE

- DO NOT enter confined spaces until atmosphere has been checked.
- DO NOT allow material to contact humans, exposed food or food utensils.
- 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 MSDS.
- Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.

SUITABLE CONTAINER

- 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.

STORAGE REQUIREMENTS

- 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 MSDS.

SAFE STORAGE WITH OTHER CLASSIFIED CHEMICALS



+: May be stored together

O: May be stored together with specific preventions

X: Must not be stored together

Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

EXPOSURE CONTROLS

Source	Material	TWA ppm	TWA mg/m ³	STEL ppm	STEL mg/m ³	Peak ppm	Peak mg/m ³	TWA F/CC	Notes
Australia Exposure Standards	ethylene glycol monobutyl ether (2- Butoxyethanol)	20	96.9	50	242				

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Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

EMERGENCY EXPOSURE LIMITS

Material	Revised IDLH Value (mg/m3)	Revised IDLH Value (ppm)
ethylene glycol monobutyl ether	3384	700 [Unch]

ODOUR SAFETY FACTOR (OSF)

OSF=2E2 (2-BUTOXY ETHANOL)

MATERIAL DATA

DUNLOP ABAMASTIC:

ETHYLENE GLYCOL MONOBUTYL ETHER:

Exposed individuals are reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class A or B.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

Class	OSF	Description
A	550	Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV- TWA for example) is being reached, even when distracted by working activities
B	26- 550	As "A" for 50- 90% of persons being distracted
C	1- 26	As "A" for less than 50% of persons being distracted
D	0.18- 1	10- 50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached
E	<0.18	As "D" for less than 10% of persons aware of being tested

ETHYLENE GLYCOL MONOBUTYL ETHER:

For ethylene glycol monobutyl ether (2-butoxyethanol)

Odour Threshold Value: 0.10 ppm (detection), 0.35 ppm (recognition)

Although rats appear to be more susceptible than other animals anaemia is not uncommon amongst humans following exposure. The TLV reflects the need to maintain exposures below levels found to cause blood changes in experimental animals. It is concluded that this limit will reduce the significant risk of irritation, haematologic effects and other systemic effects observed in humans and animals exposed to higher vapour concentrations. The toxic effects typical of some other glycol ethers (pancytopenia, testis atrophy and teratogenic effects) are not found with this substance.

Odour Safety Factor (OSF)

OSF=2E2 (2-BUTOXYETHANOL).

PERSONAL PROTECTION

continued...



EYE

- 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 lens 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].

HANDS/FEET

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

NOTE:

- The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.

OTHER

- Overalls.
- P.V.C. apron.
- Barrier cream.
- Skin cleansing cream.
- Eye wash unit.

RESPIRATOR

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

The local concentration of material, quantity and conditions of use determine the type of personal protective equipment required. For further information consult site specific CHEMWATCH data (if available), or your Occupational Health and Safety Advisor.

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

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particular process and chemical or contaminant in use.

Employers may need to use multiple types of controls to prevent employee overexposure.

General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in specific circumstances. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas.

Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

APPEARANCE

Thick white paste with a mild acrylic and ammonia odour; mixes with water.

PHYSICAL PROPERTIES

Mixes with water.

State	Non Slump Paste	Molecular Weight	Not Applicable
Melting Range (°C)	Not Available	Viscosity	Not Available
Boiling Range (°C)	100	Solubility in water (g/L)	Miscible
Flash Point (°C)	Not Applicable	pH (1% solution)	Not Available
Decomposition Temp (°C)	Not Available	pH (as supplied)	8.7- 9.2
Autoignition Temp (°C)	Not Applicable	Vapour Pressure (kPa)	Not Available
Upper Explosive Limit (%)	Not Applicable	Specific Gravity (water=1)	1.4 approx.
Lower Explosive Limit (%)	Not Applicable	Relative Vapour Density (air=1)	>1
Volatile Component (%vol)	Not Available	Evaporation Rate	Not Available

Section 10 - STABILITY AND REACTIVITY

CONDITIONS CONTRIBUTING TO INSTABILITY

■ Product is considered stable and hazardous polymerisation will not occur.

For incompatible materials - refer to Section 7 - Handling and Storage.

Section 11 - TOXICOLOGICAL INFORMATION

POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

SWALLOWED

■ The material has NOT been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (eg. liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.

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EYE

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

SKIN

■ The material is not thought to produce adverse health effects or skin irritation following contact (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting.

Entry into the blood-stream, through, for example, cuts, abrasions 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.

INHALED

■ The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.

CHRONIC HEALTH EFFECTS

■ There is limited evidence that, skin contact with this product is more likely to cause a sensitisation reaction in some persons compared to the general population.

There is some evidence from animal testing that exposure to this material may result in toxic effects to the unborn baby.

TOXICITY AND IRRITATION

DUNLOP ABAMASTIC:

■ Not available. Refer to individual constituents.

ETHYLENE GLYCOL MONOBUTYL ETHER:

■ unless otherwise specified data extracted from RTECS - Register of Toxic Effects of Chemical Substances.

TOXICITY

Oral (rat) LD50:470 mg/kg

Dermal (rabbit) LD50:220 mg/kg

Inhalation (human) TCLo:100 ppm

Inhalation (human) TCLo:195 ppm/8h * [Union Carbide]

Inhalation (Rat) LC50:450 ppm *

Oral (Rat) LD50:300 mg/kg **

Dermal (Guinea pig) LD50:210 mg/kg **

Inhalation (Rat) LC50:2210 mg/m³ **

■ The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.

For ethylene glycol monoalkyl ethers and their acetates (EGMAEs):

Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates.

EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers.

Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m³) for EGHE, LC50 > 400ppm (2620 mg/m³) for EGBEA to LC50

IRRITATION

Skin (rabbit):500 mg, open; Mild

Eye (rabbit):100 mg/24h- Moderate

Eye (rabbit):100 mg SEVERE

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> 2132 ppm (9061 mg/m³) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD₅₀ values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE in vitro than those of rats.

Repeat dose toxicity: The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA in vitro and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA in vitro.

Mutagenicity: In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in *S. typhimurium* strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. In vitro cytogenicity and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and in vivo micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.

Carcinogenicity: In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity.

Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes).

Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m³ and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m³), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m³), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m³) indicate that the members of the category are not teratogenic.

The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m³ (rabbit-EGPE), 100 ppm or 425 mg/m³ (rat-EGPE), 50 ppm or 241 mg/m³ (rat EGBE) and 100 ppm or 483 mg/m³ (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m³ (rat and rabbit-EGHE).

Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetotoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species.

At least one researcher has stated that the reproductive effects were less than that of other monoalkyl ethers of ethylene glycol.

Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility. Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions (1). The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of

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erythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration -dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma.

1: NTP Toxicology Program Technical report Series 484, March 2000.

For ethylene glycol:

Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO₂, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO₂, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

Respiratory Effects. Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning. The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning. Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases).

Cardiovascular Effects. Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12- 24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol.

Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases. Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.

Gastrointestinal Effects. Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition.

Musculoskeletal Effects. Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tenderness and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia.

Hepatic Effects. Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol.

Renal Effects. Adverse renal effects after ethylene glycol ingestion in humans can be observed during the

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Section 11 - TOXICOLOGICAL INFORMATION

third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, anuria, and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near normal renal function can return with adequate supportive therapy.

Metabolic Effects. One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate).

Neurological Effects: Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion.

Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months.

Reproductive Effects: Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration.

Developmental Effects: The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embryotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight.

Cancer: No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol.

Genotoxic Effects: Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available in vivo and in vitro laboratory studies provide consistently negative genotoxicity results for ethylene glycol.

NOTE: Changes in kidney, liver, spleen and lungs are observed in animals exposed to high concentrations of this substance by all routes.

** ASCC (NZ) SDS

CARCINOGEN

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Section 11 - TOXICOLOGICAL INFORMATION

ethylene glycol monobutyl ether	International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs	Group 3	Not classifiable as to its carcinogenicity to humans
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SKIN

ethylene glycol monobutyl ether	Australia Exposure Standards - Skin	Notes	Sk
ethylene glycol monobutyl ether	GESAMP/EHS Composite List - GESAMP Hazard Profiles	D1: skin irritation/corrosion	1

Section 12 - ECOLOGICAL INFORMATION

ETHYLENE GLYCOL MONOBUTYL ETHER:

Fish LC50 (96hr.) (mg/l):	1490
BCF<100:	0.4
log Kow (Prager 1995):	0.83
log Kow (Sangster 1997):	0.8
Half- life Soil - High (hours):	672
Half- life Soil - Low (hours):	168
Half- life Air - High (hours):	32.8
Half- life Air - Low (hours):	3.28
Half- life Surface water - High (hours):	672
Half- life Surface water - Low (hours):	168
Half- life Ground water - High (hours):	1344
Half- life Ground water - Low (hours):	336
Aqueous biodegradation - Aerobic - High (hours):	672
Aqueous biodegradation - Aerobic - Low (hours):	168
Aqueous biodegradation - Anaerobic - High (hours):	2688
Aqueous biodegradation - Anaerobic - Low (hours):	672
Photooxidation half- life air - High (hours):	32.8
Photooxidation half- life air - Low (hours):	3.28
Fish LC50 (96hr.) (mg/l):	1250- 1650
Daphnia magna EC50 (48hr.) (mg/l):	600- 1000

For Ethelene Glycol Monoalkyl Ethers and their Acetates:

log BCF: 0.463 to 0.732;

LC50 : 94 to > 5000 mg/L. (aquatic species).

Members of this category include ethylene glycol propyl ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE).

Environmental Fate: Aquatic Fate - The ethers possess no functional groups that are readily subject to hydrolysis in the presence of waters. The acetates possess an ester group that hydrolyses in neutral ambient water under abiotic conditions. Will partition predominately to water and, to a lesser extent, to air and soil. Soil - Highly mobile in soil.

Ecotoxicity: Ethelene glycol monoalkyl ethers and their acetates are readily biodegradable. The physical chemistry and environmental fate properties indicate that category members will not persist or bioconcentrate in the environment. Glycol ether acetates do not hydrolyze rapidly into their corresponding glycol ethers in water under environmental conditions. Glycol ether acetates are not acutely toxic to fish, specifically, zebra fish, rainbow trout and water fleas. Population changes were noted in freshwater and green algae species.

For Glycol Ethers:

continued...

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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.

DO NOT discharge into sewer or waterways.

log Kow: 0.76-0.83

Koc: 67

Half-life (hr) air: 17

Henry's atm m³ /mol: 2.08E-08

BOD 5 if unstated: 0.71

COD: 2.2

Log BCF: 0.4

Fish LC50 (24 h): 983-1650 mg/L

Fish LC50 (96 h): fathead minnow 1700 mg/L **

Invertebrate toxicity:

cell mult. inhib.91-900mg/L

(Daphnia) 48h LC50: >1000 mg/L **

Bioaccumulation: not sig

Effects on algae and plankton: cell mult. inhib.35-900mg/L

Degradation Biological: rapid

processes Abiotic: no hydrol&photol,RxnOH*

** [Union Carbide]

Ecotoxicity

Ingredient	Persistence: Water/Soil	Persistence: Air	Bioaccumulation	Mobility
ethylene glycol monobutyl ether	LOW	LOW	LOW	HIGH

Section 13 - DISPOSAL CONSIDERATIONS

- 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 MSDS and observe all notices pertaining to the product.

Section 14 - TRANSPORTATION INFORMATION

HAZCHEM:

None (ADG7)

continued...

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Section 14 - TRANSPORTATION INFORMATION

NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS: ADG7, IATA, IMDG

Section 15 - REGULATORY INFORMATION

POISONS SCHEDULE

None

REGULATIONS

Regulations for ingredients

ethylene glycol monobutyl ether (CAS: 111-76-2) is found on the following regulatory lists;

"Acros Transport Information", "Australia Exposure Standards", "Australia FAISD Handbook - First Aid Instructions, Warning Statements, and General Safety Precautions", "Australia Hazardous Substances", "Australia High Volume Industrial Chemical List (HVICL)", "Australia Inventory of Chemical Substances (AICS)", "Australia National Pollutant Inventory", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix F (Part 3)", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix I", "Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6", "GESAMP/EHS Composite List - GESAMP Hazard Profiles", "IMO IBC Code Chapter 17: Summary of minimum requirements", "IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances", "IMO Provisional Categorization of Liquid Substances - List 3: (Trade-named) mixtures containing at least 99% by weight of components already assessed by IMO, presenting safety hazards", "International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs", "International Fragrance Association (IFRA) Survey: Transparency List", "OECD List of High Production Volume (HPV) Chemicals", "OSPAR National List of Candidates for Substitution – Norway", "Sigma-Aldrich Transport Information"

No data for Dunlop Abamastic (CW: 15-5659)

Section 16 - OTHER INFORMATION

REPRODUCTIVE HEALTH GUIDELINES

■ Established occupational exposure limits frequently do not take into consideration reproductive end points that are clearly below the thresholds for other toxic effects. Occupational reproductive guidelines (ORGs) have been suggested as an additional standard. These have been established after a literature search for reproductive no-observed-adverse effect-level (NOAEL) and the lowest-observed-adverse-effect-level (LOAEL). In addition the US EPA's procedures for risk assessment for hazard identification and dose-response assessment as applied by NIOSH were used in the creation of such limits. Uncertainty factors (UFs) have also been incorporated.

Ingredient	ORG	UF	Endpoint	CR	Adeq TLV
ethylene glycol monobutyl ether	3.6 mg/m ³	100	D	NA	-

■ These exposure guidelines have been derived from a screening level of risk assessment and should not be construed as unequivocally safe limits. ORGS represent an 8-hour time-weighted average unless specified otherwise.

CR = Cancer Risk/10000; UF = Uncertainty factor:

TLV believed to be adequate to protect reproductive health:

LOD: Limit of detection

Toxic endpoints have also been identified as:

D = Developmental; R = Reproductive; TC = Transplacental carcinogen

Jankovic J., Drake F.: A Screening Method for Occupational Reproductive

continued...

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American Industrial Hygiene Association Journal 57: 641-649 (1996).

■ 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.

A list of reference resources used to assist the committee may be found at:
www.chemwatch.net/references.

■ The (M)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.

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Issue Date: 25-Jan-2013

Print Date: 5-Jun-2013

This is the end of the MSDS.