Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

PRODUCT NAME
2-Dimethylaminoethanol

STATEMENT OF HAZARDOUS NATURE

SUPPLIER
Santa Cruz Biotechnology, Inc.
2145 Delaware Avenue
Santa Cruz, California 95060
800.457.3801 or 831.457.3800

EMERGENCY
ChemWatch
Within the US & Canada: 877-715-9305
Outside the US & Canada: +800 2436 2255
(1-800-CHEMCALL) or call +613 9573 3112

SYNONYMS

Section 2 - HAZARDS IDENTIFICATION

CHEMWATCH HAZARD RATINGS

<table>
<thead>
<tr>
<th>Hazard</th>
<th>Min</th>
<th>Max</th>
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</thead>
<tbody>
<tr>
<td>Flammability</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Toxicity</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Body Contact</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Reactivity</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Chronic</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

CANADIAN WHMIS SYMBOLS

Flammability: Low=0, Moderate=1, High=2, Extreme=3
Toxicity: Low=0, Moderate=1, High=2, Extreme=3
Body Contact: Low=0, Moderate=1, High=2, Extreme=3
Reactivity: Low=0, Moderate=1, High=2, Extreme=3
Chronic: Low=0, Moderate=1, High=2, Extreme=3
EMERGENCY OVERVIEW

RISK
Causes burns.
Risk of serious damage to eyes.
Harmful by inhalation, in contact with skin and if swallowed.
Flammable.

POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

SWALLOWED
■ The material can produce chemical burns within the oral cavity and gastrointestinal tract following ingestion.
■ Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.
■ Ingestion of alkaline corrosives may produce burns around the mouth, ulcerations and swellings of the mucous membranes, profuse saliva production, with an inability to speak or swallow. Both the esophagus and stomach may experience burning pain; vomiting and diarrhea may follow.

EYE
■ The material can produce chemical burns to the eye following direct contact. Vapors or mists may be extremely irritating.
■ If applied to the eyes, this material causes severe eye damage.
■ Direct eye contact with corrosive bases can cause pain and burns. There may be swelling, epithelium destruction, clouding of the cornea and inflammation of the iris.

SKIN
■ The material can produce chemical burns following direct contact with the skin.
■ Skin contact with the material may be harmful; systemic effects may result following absorption.
■ The material can produce severe chemical burns following direct contact with the skin.
■ Skin contact with alkaline corrosives may produce severe pain and burns; brownish stains may develop. The corroded area may be soft, gelatinous and necrotic; tissue destruction may be deep.
■ Open cuts, abraded or irritated skin should not be exposed to this material.
■ 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 can cause respiratory irritation in some persons.
The body's response to such irritation can cause further lung damage.
■ Inhaling corrosive bases may irritate the respiratory tract.
Symptoms include cough, choking, pain and damage to the mucous membrane.
■ Inhalation hazard is increased at higher temperatures.
■ Inhalation of amine vapors may cause irritation of the mucous membrane of the nose and throat, and lung irritation with respiratory distress and cough. Swelling and inflammation of the respiratory tract is seen in serious cases; with headache, nausea, faintness and anxiety. There may also be wheezing.
■ Inhalation of quantities of liquid mist may be extremely hazardous, even lethal due to spasm, extreme irritation of larynx and bronchi, chemical pneumonitis and pulmonary edema.

CHRONIC HEALTH EFFECTS
■ 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.
■ Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.

There has been some concern that this material can cause cancer or mutations but there is not enough data to make an assessment.
Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

There is limited evidence that, skin contact with this product is more likely to cause a sensitization reaction in some persons compared to the general population.
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 foetuses of rats given high doses of MEA by gavage, showed an increased rate of embryofetal death, growth retardation, and some malformations including hydronephrosis and hydroureret. 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 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.

Secondary amines may react with nitrites to form potentially carcinogenic N-nitrosamines.

### Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

<table>
<thead>
<tr>
<th>NAME</th>
<th>CAS RN</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>dimethylethanolamine</td>
<td>108-01-0</td>
<td>&gt;98</td>
</tr>
</tbody>
</table>

### Section 4 - FIRST AID MEASURES

#### SWALLOWED
- For advice, contact a Poisons Information Center or a doctor at once. · Urgent hospital treatment is likely to be needed.

#### EYE
  - 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.

#### SKIN
  - 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.

#### INHALED
  - If fumes or combustion products are inhaled remove from contaminated area. · Lay patient down. Keep warm and rested. Inhalation of vapors or aerosols (mists, fumes) may cause lung edema. Corrosive substances may cause lung damage (e.g.

#### NOTES TO PHYSICIAN
  - 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.

### Section 5 - FIRE FIGHTING MEASURES

<table>
<thead>
<tr>
<th>Vapor Pressure (mmHg)</th>
<th>3.99 @ 20 C.</th>
</tr>
</thead>
</table>
**Upper Explosive Limit (%):** 11.9
**Specific Gravity (water=1):** 0.89
**Lower Explosive Limit (%):** 1.6

**EXTINGUISHING MEDIA**
- Water spray or fog.
- Alcohol stable foam.
- Dry chemical powder.
- Carbon dioxide.

**FIRE FIGHTING**
- Alert Emergency Responders and tell them location and nature of hazard.
- May be violently or explosively reactive.

When any large container (including road and rail tankers) is involved in a fire, consider evacuation by 1000 metres in all directions.

**GENERAL FIRE HAZARDS/HAZARDOUS COMBUSTIBLE PRODUCTS**
- Liquid and vapor are flammable.
- Moderate fire hazard when exposed to heat or flame.
- Combustion products include: carbon dioxide (CO2), carbon monoxide (CO), nitrogen oxides (NOx), other pyrolysis products typical of burning organic material.
- May emit corrosive fumes.

**FIRE INCOMPATIBILITY**
- Avoid contamination with oxidizing agents i.e. nitrates, oxidizing acids, chlorine bleaches, pool chlorine etc. as ignition may result.

**PERSONAL PROTECTION**
* Glasses:
  - Chemical goggles.
  - Full face- shield.
* Gloves:
  1. BUTYL
  2. NATURAL RUBBER
* Respirator:
  - Type AK-P Filter of sufficient capacity

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**Section 6 - ACCIDENTAL RELEASE MEASURES**

**MINOR SPILLS**
- Remove all ignition sources.
- Clean up all spills immediately.
- 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.

**MAJOR SPILLS**
- Clear area of personnel and move upwind.
- Alert Emergency Responders and tell them location and nature of hazard.

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**Section 7 - HANDLING AND STORAGE**

**PROCEDURE FOR HANDLING**
- DO NOT USE brass or copper containers / stirrers.
- DO NOT allow clothing wet with material to stay in contact with skin.
- 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.
- Avoid all personal contact, including inhalation.
- Wear protective clothing when risk of exposure occurs.
- Containers, even those that have been emptied, may contain explosive vapours.
- Do NOT cut, drill, grind, weld or perform similar operations on or near containers.

**RECOMMENDED STORAGE METHODS**
- DO NOT use aluminium, galvanised or tin-plated containers.
- Lined metal can, Lined metal pall/drum
- Plastic pail.
- 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.
In its anhydrous state, DMAE is compatible with aluminum, but in an aqueous mixture, it is highly corrosive to aluminum, copper alloys,
STORAGE REQUIREMENTS
- Store in approved flammable liquid storage area.
- No smoking, naked lights/ignition sources.
- Keep containers securely sealed.
- Store away from incompatible materials in a cool, dry, well-ventilated area.
- Protect containers against physical damage and check regularly for leaks.
- Storage areas should be clearly identified, well illuminated, clear of obstruction and accessible only to trained and authorised personnel.
- Adequate security must be provided so that unauthorised personnel do not have access.
- Store in grounded, properly designed and approved vessels and away from incompatible materials.
- Store according to applicable regulations for flammable materials for storage tanks, containers, piping, buildings, rooms, cabinets, allowable quantities and minimum storage distances.
- Use non-sparking ventilation systems, approved explosion proof equipment and intrinsically safe electrical systems.
- Have appropriate extinguishing capability in storage area (e.g. portable fire extinguishers - dry chemical, foam or carbon dioxide) and flammable gas detectors.
- Keep adsorbents for leaks and spills readily available.
- For bulk storages, consider use of floating roof or nitrogen blanketed vessels; where venting to atmosphere is possible, equip storage tank vents with flame arrestors; inspect tank vents during winter conditions for vapour/ice build-up; storage tanks should be above ground and diked to hold entire contents.
- Observe manufacturer’s storing and handling recommendations.
- Do NOT store near acids, or oxidizing agents.
- Material is hygroscopic, i.e. absorbs moisture from the air. Keep containers well sealed in storage.

Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

EXPOSURE CONTROLS

<table>
<thead>
<tr>
<th>Source</th>
<th>Material</th>
<th>TWA ppm</th>
<th>TWA mg/m³</th>
<th>STEL ppm</th>
<th>STEL mg/m³</th>
<th>Peak ppm</th>
<th>Peak mg/m³</th>
<th>TWA F/CC</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Canada - Ontario Occupational Exposure Limits</td>
<td>dimethylethanolamine (N, N-Dimethyl-ethanolamine / Diméthyléthanolamine)</td>
<td>3</td>
<td>11</td>
<td>6</td>
<td>22</td>
<td></td>
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ENDOELTABLE

PERSONAL PROTECTION

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

EYE
- Chemical goggles.
- Full face shield.

HANDS/FEET
- Elbow length PVC gloves.
- When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots.

NOTE: The material may produce skin sensitization in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.

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.
- Contaminated gloves should be replaced. 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.

OTHER
- Overalls.
- PVC Apron.
- Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity.
- For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets), non sparking safety footwear.

ENGINEERING CONTROLS
■ Local exhaust ventilation usually required. If risk of overexposure exists, wear an approved respirator.

Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

PHYSICAL PROPERTIES
Liquid.
Mixes with water.
Corrosive.
Alkaline.

<table>
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<tr>
<th>Property</th>
<th>Value</th>
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<tbody>
<tr>
<td>Molecular Weight</td>
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<tr>
<td>Melting Range (°F)</td>
<td>-74</td>
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<tr>
<td>Boiling Range (°F)</td>
<td>270.5-273</td>
</tr>
<tr>
<td>Flash Point (°F)</td>
<td>102</td>
</tr>
<tr>
<td>Decomposition Temp (°F)</td>
<td>Not Available</td>
</tr>
<tr>
<td>Autoignition Temp (°F)</td>
<td>428</td>
</tr>
<tr>
<td>Upper Explosive Limit (%)</td>
<td>11.9</td>
</tr>
<tr>
<td>Lower Explosive Limit (%)</td>
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</tr>
<tr>
<td>Volatile Component (%vol)</td>
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</tr>
<tr>
<td>VOC(regulatory)</td>
<td>lb/gall</td>
</tr>
<tr>
<td>VOC(actual)</td>
<td>lb/gall</td>
</tr>
</tbody>
</table>

APPEARANCE
■ Material is hygroscopic, absorbs moisture from surrounding air. Flammable liquid; soluble in water. Amine odour. Mixes with water, acetone, ether and benzene. Refractive Index: 1.4300.Ka 8.88+/-0.2

Section 10 - CHEMICAL STABILITY

CONDITIONS CONTRIBUTING TO INSTABILITY
- Presence of incompatible materials.
- Product is considered stable.

STORAGE INCOMPATIBILITY
■ Avoid strong acids.
- Avoid contact with copper, aluminium and their alloys.
Avoid reaction with oxidizing agents.
Dimethylethanolamine
- is a strong organic base
- reacts violently with oxidisers, acids
- ignites spontaneously with cellulose nitrate, and possibly other nitrogen compounds
- may react explosively with silver, cobalt or chromium compounds
- in contact with nitromethane forms a heat-, friction-, and shock- sensitive compound
- is incompatible with acrylates, aldehydes, alcohols, alkylene oxides, caprolactam solution, cresols, organic anhydrides, substituted allyls, epichlorohydrin, glycols, halogenated compounds, isocyanates, ketones, mercury, phenols, vinyl acetate
- attacks copper, zinc and their alloys and galvanised steel.

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

Section 11 - TOXICOLOGICAL INFORMATION

dimethylethanolamine
In one occupational study in the manufacture of polyurethane foam insulation for refrigerators, adverse effects included disorders of the upper respiratory tract and nervous system, along with significant changes in the immune status of workers exposed to a mixture of dimethylaminoethanol (DMAE) and other Ephedra spp. Ephedra alkaloids cause similar cardiovascular and neurological effects reported for DMAE.

DMAE, thought to be a precursor for acetylcholine, has been tested for its efficacy in treating a variety of diseases possibly related to deficiencies of acetylcholine, including tardive dyskinesia, Alzheimer's disease, amnesic disorders, age-related cognitive impairment, and Tourette's syndrome, with mixed results. Treatment with DMAE for tardive dyskinesia, a side effect of neuroleptic medications, was associated with serious cholinergic side effects: nasal and oral secretions, dyspnea, and respiratory failure. DMAE was used in the treatment of one patient for a low-frequency action tremor. This treatment was successful for ten years, until side effects of increasing neck pain and orofacial and respiratory dyskinesia occurred. Treatment was discontinued, and it was concluded that the dyskinesia could be attributed to the effects of DMAE.

A meta-analysis of randomized controlled trials indicated that DMAE was no more effective than placebo in the treatment of tardive dyskinesia. Rather, there was a significantly increased risk of adverse events associated with the DMAE treatment. DMAE treatment increases in the concentration of choline in both the plasma and the brain of treated rats; the mechanism for this phenomenon was unknown. Since it was known that DMAE inhibits the influx of choline to the brain across the blood-brain barrier, it is possible that DMAE also inhibited the efflux of choline from the brain, resulting in an accumulation in the brain.

Differential penetration of the blood-brain barrier by several DMAE derivatives has been noted. Radiolabeled DMAE-p-chlorophenoxyacetate was found in higher concentrations in the brain than radiolabeled DMAE after intravenous treatment of mice. Higher levels of DMAE were found in the brain after dosing with centrophenoxine than with DMAE, possibly due to improved penetration of the blood-brain barrier by the esterified form of DMAE. Similarly radiolabeled cyroprodenate maleate (the cyclohexylpropionic acid ester of DMAE) was more rapidly absorbed and accumulated to a large extent in the brain.

Choline, or trimethylaminoethanol, may be formed by methylation of DMAE. Choline is an essential nutrient. Although small amounts may be synthesised, choline must be supplemented through the diet to maintain adequate physiological concentrations for optimal health. Choline is a precursor for the neurotransmitter, acetylcholine. As a possible precursor of choline, DMAE has also been studied as a potential modulator of many biological processes requiring choline; these include the production of structural components of cell membranes (the phospholipids, especially phosphatidylcholine and sphingomyelin), the synthesis of intracellular signalling molecules (diacylglycerol and ceramide), platelet activating factor and sphingophosphorylcholine. Phosphatidylcholine is a required component of very low-density lipoproteins (VLDL) particles, necessary for the transportation of cholesterol and fat from the liver to other sites in the body. Betaine, a metabolite of choline, participates in methyl-group transfer.

In one occupational study in the manufacture of polyurethane foam insulation for refrigerators, adverse effects included disorders of the upper respiratory tract and nervous system, along with significant changes in the immune status of workers exposed to a mixture of...
DMAE, ethylenediamine, propylene oxide, and 4,4'-methylenebisphenyl disocyanate. A spray painter developed severe respiratory symptoms, which seemed to be related to occupational exposure to a specific type of spray paint containing DMAE. Follow-on skin tests with DMAE (undiluted, and 1:10 and 1:100 dilutions in saline) in three human volunteers produced wheal and flare responses at the high dose. This was interpreted as an irritant response, and not a sign of immunotoxicity. Despite one clear case for occupational asthma form DMAE exposure, it fails to meet the current criteria for classification as a respiratory sensitizer.

Neurotoxicity: Using a method to classify the risks associated with occupational exposures to neurotoxic chemicals obtained from national computer-based registers, DMAE produces a small increase in the risk of damaging the nervous system under normal work conditions.

DMAE (as centrophenoxyne, an ester of DMAE) was tested for its effects on spinal reflexes in mice. 50 mg/kg (0.170 mmol/kg) demonstrated a considerable change in spinal reflexes, specifically in the inhibition of polysynaptic reflexes. Higher doses (400 to 600 mg/kg [1.40 to 2.04 mmol/kg]) intraperitoneally resulted in ataxia, reduced mobility, inhibition, and mortality in some treated mice. Similar doses in rats resulted in limited mobility and an inhibited state.

Intravenous administration of DMAE (175 to 350 mg/kg; 1.95 to 3.90 mmol/kg) resulted in dose-dependant psychoanalytic effects (as demonstrated by spontaneous running and an increase on conditioned reflexes) in rats. DMAE appears to exert a central vasmotor stimulant effect. Intracerebroventricular (ICV) administration of DMAE (0.1 to 2.0 mg; 1.0 to 20 umol) resulted in potentiation of the carotid occlusion response (all doses) resulting in an increase in blood pressure in dogs (higher doses). This effect was not abolished by atropine sulfate (ICV).

With meclofenoxate (centrophenoxine hydrochloride) treatment (10 to 40 mg/kg body weight; 0.040 to 0.16 mmol/kg), a significant dose-dependent reduction in both blood pressure (up to 49.7+/-.0.39 mmHg reduction) and heart rate (up to 71 +/-.5.5% reduction) was observed in the old rats at the 40 mg/kg (0.16 mmol/kg) dose level.

Reproductive toxicity: No histopathological changes in the gonads were observed after repeated exposure to DMAE in a 90-day intrahemato study in rats.

DMAE via inhalation induced maternal toxicity in rats at all tested exposure levels (10, 30, and 100 ppm; 40, 110, and 370 mg/m3; 0.41, 1.20, and 4.10 mmol/m3), as demonstrated by changes in body weight gain in the mid- and high-dose groups and ocular changes in the mid- and low-dose. Sporadic, inconsistent alterations in gestational parameters including significant decreases in viable implants per litter, percentage live foetuses/litter, and litter size in rats exposed to 10 ppm (40 mg/m3; 41 mmol/m3) and a significant decrease in the percentage of male foetuses in rats exposed to 30 ppm (110 mmg/m3; 1.20 mmol/m3). Skeletal variations in foetuses included decreased incidences of poorly ossified cervical centrum, bilobed thoracic centrum, bilobed sternebre, unossified proximal phalanges of the forefoot, and increased incidences of split cervical centrum and bilobed thoracic centrum. However, a consistent pattern was lacking, resulting in a NOAEL for embryofetal toxicity and teratogenicity of 100 ppm (370 mg/m3; 4.10 mmol/m3) or greater. A NOAEL for maternal toxicity was estimated at 10 ppm (40 mg/m3; 0.41 mmol/m3).

A five-generation study was conducted; each generation of rats or only the first and fifth generations were exposed in utero to centrophenoxine on gestation days 11 to 14 (during embryogenesis). Treating Wistar dams with meclofenoxate prenatally resulted in significant increases in weight of the offspring. The increase in embryo weights did not continue into postnatal life. Continuous treatment through several generations increased fertility and an overall increase in the number of offspring.

Carcinogenicity: A five-generation study was conducted; each generation of rats or only the first and fifth generations were exposed in utero to centrophenoxine on gestation days 11 to 14 (during embryogenesis). Treating Wistar dams with meclofenoxate prenatally resulted in significant increases in weight of the offspring. The increase in embryo weights did not continue into postnatal life. Continuous treatment through several generations increased fertility and an overall increase in the number of offspring.

Carcinogenicity: There was no statistically significant and an increase, or morphological differences, in the incidence of neoplasms in any organ in female C3H/HeN mice given drinking water with 10 mM (900 ug/mL) DMAE for 105 weeks, or in female C3H/HeN mice given 15 mM (1300 ug/mL) DMAE for 123 weeks. No changes in the structure, appearance, or microscopic morphology of various organs were observed. Treatment with DMAE did not affect survival, initial body weight gain, or mature body weight of either strain of mouse.

Di- and triaminoethanols, which are structurally related to DMAE and are found in cutting fluids, pesticides, and cosmetics, can give rise to N-nitrosodiethanolamine (NDELA) via nitrosation resulting from reaction with nitrite or nitrous oxide. The authors also noted that NDELA has been shown to be a potent carcinogen, producing mainly hepatocellular carcinomas in rats and epithelial neoplasms of the nasal cavity and trachea in hamsters.

Genotoxicity: Salmonella typhimurium assay. Tester strains TA98, TA100, TA1535, TA1537, and TA1538 were all tested, both in the presence and absence of a metabolic activation system. DMAE, ranging from 0.37 to 995 umol (0.033 to 89.5 mg)/plate failed to demonstrate any mutagenic response.

DMAE also failed to induce any sex-linked recessive lethal mutations in the Drosophila melanogaster (7200 or 8100 ppm; 80.10 or 90.10 mmol/L).

The genotoxicity of DMAE was investigated in several mammalian systems, both in vitro and in vivo. In vitro assays included sister chromatid exchange and hypoxanthine-guanine phosphoribosyl transferase forward gene mutation test (HGPT), both in Chinese hamster ovary cells. All of the in vitro assays failed to demonstrate genotoxicity within the dose range.

Immunotoxicity: DMAE was unable to covalently derivatise protein in an in vitro assay. It is thought that the ability to covalently derivatise protein enables some low-molecular-weight chemicals (LMWC) to induce allergic antibody-mediated responses that may cause asthma in people occupationally exposed to LMWC. The ability of DMAE to act as a skin sensitizer was tested in the murine local lymph node assay at 0, 3, 10, and 30% w/v (0, 33, 110, and 330 mmol/L). The test resulted in test/control ratios of 0, 1.93, 2.13, and 14.50 respectively. Typically, ratios greater than 3 are indicative of potential sensitisers; therefore, based on this test, DMAE was classified as a potential skin sensitizer. Human experiences with DMAE under normal handling precautions have not supported this result. Similarly, DMAE, evaluated in the guinea pig maximisation procedure, was without any clear evidence of skin sensitization.

Metabolism: DMAE is absorbed (either from the small intestine after oral dosing or from the bloodstream after injections), and rapidly transported to the liver where much of it is metabolised. DMAE is metabolised through the phospholipid cycle to produce phosphodymethylthanolamine and glycerophosphodiacylglycerol. Pigs and rats dosed with cyprodenate maleate, the cyclohexylypropionic acid ester of DMAE, was found to be was well absorbed from the digestive tract and distributed to tissues and organs. Similarly, centrophenoxyne (an ester of DMAE) was well absorbed after oral administration. After transport to the liver, a portion of centrophenoxyne is converted to its constituent moieties, DMAE and p-chlorophenoxyacetic acid (PCPA), while the unmethylated form was transported throughout the body by the circulatory system.

In humans, 33% of an injected 1 g (10 mmol) dose of DMAE was excreted unchanged. It was suggested that the remaining dose may have been demethylated to ethanolamine and entered into normal metabolic pathways.

CARCINOGEN
This material and its container must be disposed of as hazardous waste.

**Section 14 - TRANSPORTATION INFORMATION**

DOT:
Symbols: None Hazard class or Division: 8
Identification Numbers: UN2051 PG: II
Label Codes: 8, 3 Special provisions: B2, IB2, T7, TP2
Packaging: Exceptions: 154 Packaging: Non-bulk: 202
Packaging: Exceptions: 154 Quantity limitations: 1 L
Passenger aircraft/rail:
Quantity Limitations: Cargo 30 L Vessel stowage: Location: A aircraft only:
Vessel stowage: Other: None
Hazardous materials descriptions and proper shipping names:
2-Dimethylaminoethanol
**Air Transport IATA:**
UN/ID Number: 2051 Packing Group: II
Special provisions: None
Cargo Only

9 of 10
Packing Instructions: 855 Maximum Qty/Pack: 30 L
Passenger and Cargo Passenger and Cargo
Packing Instructions: Y840 Maximum Qty/Pack: 1 L
Passenger and Cargo Limited Quantity Passenger and Cargo Limited Quantity
Packing Instructions: 851 Maximum Qty/Pack: 0.5 L
Shipping Name: 2-DIMETHYLAMINOETHANOL

Maritime Transport IMDG:
IMDG Class: 8 IMDG Subrisk: 3
UN Number: 2051 Packing Group: II
EMS Number: F-E,S-C Special provisions: None
Limited Quantities: 1 L
Shipping Name: 2-DIMETHYLAMINOETHANOL

Section 15 - REGULATORY INFORMATION

dimethylethanolamine (CAS: 108-01-0) is found on the following regulatory lists;

Section 16 - OTHER INFORMATION

LIMITED EVIDENCE
■ Cumulative effects may result following exposure*.
■ Limited evidence of a carcinogenic effect*.
■ Possible skin sensitizer*.
* (limited evidence).

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■ 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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