1,2-Dipalmitoyl-sn-glycero-3-phospho-(N,N-dimethyl)-ethanolamine

sc-208740

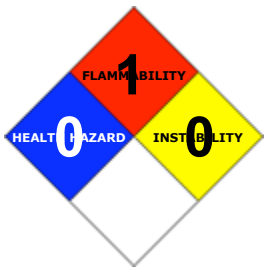
Material Safety Data Sheet

Hazard Alert Code Key:	EXTREME	HIGH	MODERATE	LOW

Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

PRODUCT NAME
1,2-Dipalmitoyl-sn-glycero-3-phospho-(N,N-dimethyl)-ethanolamine

STATEMENT OF HAZARDOUS NATURE
CONSIDERED A HAZARDOUS SUBSTANCE ACCORDING TO OSHA 29 CFR 1910.1200.

NFPA


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
C39-H78-N-O8-P, "for L-form", "1, 2-dipalmitoyl-sn-glycero-3-phospho-N, N-dimethylethanolamine", "L-beta, gamma-dipalmitoyl-N, N-dimethyl-alpha-cephalin", "3-sn-phosphatidyl-N, N-dimethylethanolamine, 1, 2-dipalmitoyl", "for DL-form", "rac-1, 2-dipalmitoyl-glycero-3-phospho-N, N-dimethylethanolamine", "DL-beta, gamma-dipalmitoyl-N, N-dimethyl-alpha-cephalin", "rac-phosphatidyl-N, N-dimethylethanolamine, 1, 2-dipalmitoyl", "cationic surfactant"

Section 2 - HAZARDS IDENTIFICATION

CHEMWATCH HAZARD RATINGS

		Min	Max
Flammability:	1		
Toxicity:	0		
Body Contact:	0		
Reactivity:	1		
Chronic:	2		

Min/Nil=0
Low=1
Moderate=2
High=3
Extreme=4

CANADIAN WHMIS SYMBOLS

EMERGENCY OVERVIEW

RISK

POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

SWALLOWED

■ The material has NOT been classified as "harmful by ingestion".
This is because of the lack of corroborating animal or human evidence.

EYE

■ Although the material is not thought to be an irritant, direct contact with the eye may cause transient discomfort characterized by tearing or conjunctival redness (as with windburn).
Slight abrasive damage may also result.

SKIN

■ The material is not thought to produce adverse health effects or skin irritation following contact (as classified 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.
■ 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 is not thought to produce adverse health effects or irritation of the respiratory tract (as classified 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.
■ Persons with impaired respiratory function, airway diseases and conditions such as emphysema or chronic bronchitis, may incur further disability if excessive concentrations of particulate are inhaled.
■ Fine mists generated from plant/ vegetable (or more rarely from animal) oils may be hazardous.
Extreme heating for prolonged periods, at high temperatures, may generate breakdown products which include acrolein and acrolein-like substances.

CHRONIC HEALTH EFFECTS

■ There has been some concern that this material can cause cancer or mutations but there is not enough data to make an assessment.
Long term exposure to high dust concentrations may cause changes in lung function i.e. pneumoconiosis; caused by particles less than 0.5 micron penetrating and remaining in the lung.

Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

NAME	CAS RN	%
beta,gamma-dipalmitoyl-N,N-dimethyl-alpha-cephalin	3922-61-0	>98

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 Center or a doctor.

EYE

■ If this product comes in contact with eyes: · Wash out immediately with water. · If irritation continues, seek medical attention.

SKIN

■ If skin or hair contact occurs: · Flush skin and hair with running water (and soap if available). · Seek medical attention in event of irritation.

INHALED

· If dust is inhaled, remove from contaminated area. · Encourage patient to blow nose to ensure clear passage of breathing. · If irritation or discomfort persists seek medical attention.

NOTES TO PHYSICIAN

■ Treat symptomatically.

Section 5 - FIRE FIGHTING MEASURES

Vapour Pressure (mmHG):	Negligible
Upper Explosive Limit (%):	Not available.
Specific Gravity (water=1):	Not available
Lower Explosive Limit (%):	Not available

EXTINGUISHING MEDIA
· Water spray or fog.
· Foam.

FIRE FIGHTING
· Alert Emergency Responders and tell them location and nature of hazard.
· Wear breathing apparatus plus protective gloves.

GENERAL FIRE HAZARDS/HAZARDOUS COMBUSTIBLE PRODUCTS
· Combustible solid which burns but propagates flame with difficulty.
· Avoid generating dust, particularly clouds of dust in a confined or unventilated space as dusts may form an explosive mixture with air, and any source of ignition, i.e. flame or spark, will cause fire or explosion. Dust clouds generated by the fine grinding of the solid are a particular hazard; accumulations of fine dust may burn rapidly and fiercely if ignited.
Combustion products include: carbon monoxide (CO), carbon dioxide (CO2), nitrogen oxides (NOx), phosphorus oxides (POx), acrolein, other pyrolysis products typical of burning organic material.
May emit poisonous 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.
Gloves:
Respirator:
Particulate

Section 6 - ACCIDENTAL RELEASE MEASURES

MINOR SPILLS
· Clean up all spills immediately.
· Avoid breathing dust and contact with skin and eyes.
MAJOR SPILLS
■ Moderate hazard.
· CAUTION: Advise personnel in area.
· Alert Emergency Responders and tell them location and nature of hazard.

Section 7 - HANDLING AND STORAGE

PROCEDURE FOR HANDLING
· Avoid all personal contact, including inhalation.
· Wear protective clothing when risk of exposure occurs.
Empty containers may contain residual dust which has the potential to accumulate following settling. Such dusts may explode in the presence of an appropriate ignition source.
· Do NOT cut, drill, grind or weld such containers.
· In addition ensure such activity is not performed near full, partially empty or empty containers without appropriate workplace safety authorisation or permit.

RECOMMENDED STORAGE METHODS
· Polyethylene or polypropylene container.
· Check all containers are clearly labelled and free from leaks.

STORAGE REQUIREMENTS
· Store in original containers.
· Keep containers securely sealed.

Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

EXPOSURE CONTROLS

Source	Material	TWA ppm	TWA mg/m³	STEL ppm	STEL mg/m³	Peak ppm	Peak mg/m³	TWA F/CC	Notes
Canada - Ontario Occupational Exposure Limits	beta,gamma-dipalmitoyl-N,N-dimethyl-alpha-cephalin (Particles (Insoluble or Poorly Soluble) Not Otherwise)		10 (I)						
Canada - British Columbia Occupational Exposure Limits	beta,gamma-dipalmitoyl-N,N-dimethyl-alpha-cephalin (Particles (Insoluble or Poorly Soluble) Not Otherwise Classified (PNOC))		10 (N)						
Canada - Ontario Occupational Exposure Limits	beta,gamma-dipalmitoyl-N,N-dimethyl-alpha-cephalin (Specified (PNOS) / Particules (insolubles ou peu solubles) non précisées par ailleurs)		3 (R)						
US - Tennessee Occupational Exposure Limits - Limits For Air Contaminants	beta,gamma-dipalmitoyl-N,N-dimethyl-alpha-cephalin (Particulates not otherwise regulated Respirable fraction)		5						
US - California Permissible Exposure Limits for Chemical Contaminants	beta,gamma-dipalmitoyl-N,N-dimethyl-alpha-cephalin (Particulates not otherwise regulated Respirable fraction)		5						(n)
US - Oregon Permissible Exposure Limits (Z-1)	beta,gamma-dipalmitoyl-N,N-dimethyl-alpha-cephalin (Particulates not otherwise regulated (PNOR) (f) Total Dust)	-	10						Bold print identifies substances for which the Oregon Permissible Exposure Limits (PELs) are different than the federal Limits. PNOR means “particles not otherwise regulated.”

US - Michigan Exposure Limits for Air Contaminants	beta,gamma-dipalmitoyl-N,N-dimethyl-alpha-cephalin (Particulates not otherwise regulated, Respirable dust)		5		
US - Oregon Permissible Exposure Limits (Z-1)	beta,gamma-dipalmitoyl-N,N-dimethyl-alpha-cephalin (Particulates not otherwise regulated (PNOR) (f) Respirable Fraction)	-	5		Bold print identifies substances for which the Oregon Permissible Exposure Limits (PELs) are different than the federal Limits. PNOR means “particles not otherwise regulated.”
US - Wyoming Toxic and Hazardous Substances Table Z1 Limits for Air Contaminants	beta,gamma-dipalmitoyl-N,N-dimethyl-alpha-cephalin (Particulates not otherwise regulated (PNOR)(f)-Respirable fraction)		5		
Canada - Prince Edward Island Occupational Exposure Limits	beta,gamma-dipalmitoyl-N,N-dimethyl-alpha-cephalin (Particles (Insoluble or Poorly Soluble) [NOS] Inhalable particles)		10		See Appendix B current TLV/BEI Book

ENDOELTABLE

PERSONAL PROTECTION

RESPIRATOR

• particulate.

EYE

· Safety glasses with side shields
· Chemical goggles.

HANDS/FEET

■ Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: such as:
· 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).

· 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) 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) 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.
Experience indicates that the following polymers are suitable as glove materials for protection against undissolved, dry solids, where abrasive particles are not present.
· polychloroprene
· nitrile rubber
· butyl rubber
· fluorocaoutchouc
· polyvinyl chloride
Gloves should be examined for wear and/ or degradation constantly.

OTHER

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

ENGINEERING CONTROLS

· Local exhaust ventilation is required where solids are handled as powders or crystals; even when particulates are relatively large, a certain proportion will be powdered by mutual friction.
· Exhaust ventilation should be designed to prevent accumulation and recirculation of particulates in the workplace.

Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

PHYSICAL PROPERTIES

Solid.
Mixes with water.

State	Divided solid	Molecular Weight	720.03
Melting Range (°F)	Not available	Viscosity	Not Applicable
Boiling Range (°F)	Not available	Solubility in water (g/L)	Miscible
Flash Point (°F)	Not available	pH (1% solution)	Not available
Decomposition Temp (°F)	Not available.	pH (as supplied)	Not applicable
Autoignition Temp (°F)	Not available	Vapour Pressure (mmHG)	Negligible
Upper Explosive Limit (%)	Not available.	Specific Gravity (water=1)	Not available
Lower Explosive Limit (%)	Not available	Relative Vapor Density (air=1)	>1
Volatile Component (%vol)	Negligible	Evaporation Rate	Not applicable

APPEARANCE

White to off-white solid.

Section 10 - CHEMICAL STABILITY

CONDITIONS CONTRIBUTING TO INSTABILITY

· Presence of incompatible materials.
· Product is considered stable.

STORAGE INCOMPATIBILITY

■ Avoid reaction with oxidizing agents.

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

Section 11 - TOXICOLOGICAL INFORMATION

beta,gamma-dipalmitoyl-N,N-dimethyl-alpha-cephalin

TOXICITY AND IRRITATION

BETA,GAMMA-DIPALMITOYL-N,N-DIMETHYL-ALPHA-CEPHALIN:

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

■ For dimethylethanolamine (DMAE) and selected salts and esters:

Toxicology:

Humans: 10 to 20 mg (0.042-0.084 mmol) of DMAE tartrate administered orally to humans, produced mild mental stimulation. At 20 mg/day (0.084 mmol), there was a gradual increase in muscle tone and perhaps an increased frequency of convulsions in susceptible individuals. Larger doses (not specified) produced insomnia, muscle tenseness, and spontaneous muscle twitches.

Doses of DMAE as high as 1200 mg/day (13.46 mmol/day) produced no serious side effects. A single 2500-mg (27.80-mmol) dose taken in a suicide attempt had no adverse effect. A single 2500-mg (27.80-mmol) dose taken in a suicide attempt had no adverse effect.

DMAE supplementation is contraindicated during pregnancy and lactation It is also contraindicated for treatment of people with symptoms of schizophrenia and clonic-tonic seizure disorders The principal contraindication to the use of DMAE was grand mal epilepsy. DMAE also antagonizes the depressant effects of barbiturates.

A large number of adverse health effects are associated with DMAE. These include cardiovascular, neurological, and/or psychological effects. Specific attribution of adverse effects to DMAE is unlikely, as many of these products also contained Ephedra vulgaris alkaloids 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 cyprodenate 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 spingophosphorylcholine. 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'-methylenediphenyl diisocyanate . 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 sensitiser

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

DMAE (as centrophenoxine, 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 psychoanaleptic effects (as demonstrated by spontaneous running in mice) and an influence on conditioned reflexes in rats.

DMAE appears to exert a central vasomotor 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 +/-4.5% 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 inhalation 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 mg/m3; 1.20 mmol/m3). Skeletal variations in foetuses included decreased incidences of poorly ossified cervical centrum , bilobed thoracic centrum , bilobed sternebrae ,unossified proximal phalanges of the forelimb, and increased incidences of split cervical centra ,and bilobed thoracic centrum . However, a consistent pattern was lacking, resulting in a NOAEL for embryofoetal 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: There was no statistically significant increase, or morphological difference, 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/HeJ(+) 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 ranges..
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 sensitiser 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 phosphoryldimethylethanolamine and glycerophosphatidylcholine Pigs and rats dosed with cyprodenate maleate, the cyclohexylpropionic acid ester of DMAE, was found to be was well absorbed from the digestive tract and distributed to tissues and organs. Similarly, centrophenoxine (an ester of DMAE) was well absorbed after oral administration. After transport to the liver, a portion of centrophenoxine is converted to its constituent moieties, DMAE and p-chlorophenoxyacetic acid (PCPA), while the unmetabolised 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.
No significant acute toxicological data identified in literature search.

Section 12 - ECOLOGICAL INFORMATION

No data

Ecotoxicity

Ingredient	Persistence: Water/Soil	Persistence: Air	Bioaccumulation	Mobility
beta,gamma-dipalmitoyl-N,N-dimethyl-alpha-cephalin	No Data Available	No Data Available		

Section 13 - DISPOSAL CONSIDERATIONS

Disposal Instructions
All waste must be handled in accordance with local, state and federal regulations.
¦ 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. 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 equipment to enter drains. Collect all wash water for treatment before disposal.

· Recycle wherever possible.
· Consult manufacturer for recycling options or consult Waste Management Authority for disposal if no suitable treatment or disposal facility can be identified.

Section 14 - TRANSPORTATION INFORMATION

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

Section 15 - REGULATORY INFORMATION

beta,gamma-dipalmitoyl-N,N-dimethyl-alpha-cephalin (CAS: 3922-61-0,1487-55-4) is found on the following regulatory lists;
"Canada - British Columbia Occupational Exposure Limits","Canada - Ontario Occupational Exposure Limits","Canada - Prince Edward Island Occupational Exposure Limits","Canada National Pollutant Release Inventory (NPRI)","US - California Permissible Exposure Limits for Chemical Contaminants","US - Michigan Exposure Limits for Air Contaminants","US - Oregon Permissible Exposure Limits (Z-1)","US - Tennessee Occupational Exposure Limits - Limits For Air Contaminants","US - Wyoming Toxic and Hazardous Substances Table Z1 Limits for Air Contaminants"

Section 16 - OTHER INFORMATION

LIMITED EVIDENCE
■ Limited evidence of a carcinogenic effect*.
* (limited evidence).

Denmark Advisory list for selfclassification of dangerous substances
Substance CAS Suggested codes beta, gamma- dipalmitoyl- N, N- 3922- 61- 0 Mut3; R68 Rep3; dimethyl- alpha- cephalin R63 Xi; R38 beta, gamma- dipalmitoyl- N, N- 1487- 55- 4 Mut3; R68 Rep3; dimethyl- alpha- cephalin R63 Xi; R38

Ingredients with multiple CAS Nos
Ingredient Name CAS beta,gamma-dipalmitoyl-N,N-dimethyl-alpha-cephalin 3922-61-0, 1487-55-4

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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.

Issue Date: Nov-29-2008

Print Date:Jun-8-2011