



# GEA FOAM MARK PINK AEROSOL FIL ( a part of GEA Technologies)

Chemwatch Hazard Alert Code: 4

Version No: 10.34.1.1

Safety Data Sheet according to the Health and Safety at Work (Hazardous Substances) Regulations 2017

Issue Date: 21/04/2021

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L.GHS.NZL.EN

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

### Product Identifier

Product name	GEA FOAM MARK PINK AEROSOL
Chemical Name	Not Applicable
Synonyms	CNR3206
Proper shipping name	AEROSOLS
Other means of identification	Not Available

### Relevant identified uses of the substance or mixture and uses advised against

Relevant identified uses	Coloured foam marker for use as a temporary animal marker to identify animals which are to be drafted out
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### Details of the supplier of the safety data sheet

Registered company name	FIL ( a part of GEA Technologies)
Address	72 Portside Drive, Mt Manganui Tauranga 3116 New Zealand
Telephone	+647 575 2162
Fax	+64 7 575 2161
Website	<a href="http://www.fil.co.nz">www.fil.co.nz</a>
Email	office.fil@gea.com

### Emergency telephone number

Association / Organisation	CHEMCALL
Emergency telephone numbers	NZ-0800 243 622 AU -1800127406
Other emergency telephone numbers	+64 4 9179888(global)

## SECTION 2 Hazards identification

### Classification of the substance or mixture

**Considered a Hazardous Substance according to the criteria of the New Zealand Hazardous Substances New Organisms legislation. Classified as Dangerous Goods for transport purposes.**

#### ChemWatch Hazard Ratings

	Min	Max	
Flammability	4		
Toxicity	0		0 = Minimum
Body Contact	2		1 = Low
Reactivity	0		2 = Moderate
Chronic	2		3 = High
			4 = Extreme

Classification [1]	Flammable Aerosols Category 1, Specific target organ toxicity - repeated exposure Category 2, Eye Irritation Category 2, Reproductive Toxicity Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI
Determined by Chemwatch using GHS/HSNO criteria	2.1.2A, 6.4A, 6.8B, 6.9B

### Label elements

Hazard pictogram(s)	
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## GEA FOAM MARK PINK AEROSOL

Signal word **Danger**

## Hazard statement(s)

H222	Extremely flammable aerosol.
H373	May cause damage to organs through prolonged or repeated exposure.
H319	Causes serious eye irritation.
H361	Suspected of damaging fertility or the unborn child.

## Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P211	Do not spray on an open flame or other ignition source.
P251	Do not pierce or burn, even after use.
P260	Do not breathe fumemist/vapours/spray
P280	Wear protective gloves/protective clothing/eye protection/face protection.

## Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P314	Get medical advice/attention if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.

## Precautionary statement(s) Storage

P405	Store locked up.
P410+P412	Protect from sunlight. Do not expose to temperatures exceeding 50 °C/122 °F.

## Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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## SECTION 3 Composition / information on ingredients

## Substances

See section below for composition of Mixtures

## Mixtures

CAS No	%[weight]	Name
102-71-6	8-15	<u>triethanolamine</u>
111-42-2	1-5	<u>diethanolamine</u>
67701-08-0	3-12	<u>fatty acids, C16-18 and C18-unsaturated</u>
12179-04-3	<0.5	<u>sodium borate, pentahydrate</u>
106-97-8.	3-12	<u>butane</u>
74-98-6	<1	<u>propane</u>
Not Available	Balance	Ingredients not contributing to classification

**Legend:** 1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; \* EU IOELVs available

## SECTION 4 First aid measures

## Description of first aid measures

<b>Eye Contact</b>	<p>If aerosols come in contact with the eyes:</p> <ul style="list-style-type: none"> <li>▶ Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh running water.</li> <li>▶ 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.</li> <li>▶ Transport to hospital or doctor without delay.</li> <li>▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> <li>▶ Generally not applicable.</li> </ul>
<b>Skin Contact</b>	<p>If solids or aerosol mists are deposited upon the skin:</p> <ul style="list-style-type: none"> <li>▶ Flush skin and hair with running water (and soap if available).</li> <li>▶ Remove any adhering solids with industrial skin cleansing cream.</li> <li>▶ <b>DO NOT use solvents.</b></li> <li>▶ Seek medical attention in the event of irritation.</li> <li>▶ Generally not applicable.</li> </ul>
<b>Inhalation</b>	<p>If aerosols, fumes or combustion products are inhaled:</p> <ul style="list-style-type: none"> <li>▶ Remove to fresh air.</li> <li>▶ Lay patient down. Keep warm and rested.</li> <li>▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.</li> </ul>

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	<ul style="list-style-type: none"> <li>▶ If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.</li> <li>▶ Transport to hospital, or doctor.</li> <li>▶ Generally not applicable.</li> </ul>
<b>Ingestion</b>	<ul style="list-style-type: none"> <li>▶ Not considered a normal route of entry.</li> <li>▶ Generally not applicable.</li> </ul>

### Indication of any immediate medical attention and special treatment needed

For petroleum distillates

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

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Treat symptomatically.

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

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

Alkalis continue to cause damage after exposure.

INGESTION:

- ▶ Milk and water are the preferred diluents

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

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

\* Catharsis and emesis are absolutely contra-indicated.

\* Activated charcoal does not absorb alkali.

\* Gastric lavage should not be used.

Supportive care involves the following:

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

SKIN AND EYE:

- ▶ Injury should be irrigated for 20-30 minutes.

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

## SECTION 5 Firefighting measures

### Extinguishing media

**SMALL FIRE:**

- ▶ Water spray, dry chemical or CO<sub>2</sub>

**LARGE FIRE:**

- ▶ Water spray or fog.

### Special hazards arising from the substrate or mixture

<b>Fire Incompatibility</b>	▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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### Advice for firefighters

<b>Fire Fighting</b>	<ul style="list-style-type: none"> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ May be violently or explosively reactive.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ If safe, switch off electrical equipment until vapour fire hazard removed.</li> <li>▶ Use water delivered as a fine spray to control fire and cool adjacent area.</li> <li>▶ <b>DO NOT</b> approach containers suspected to be hot.</li> <li>▶ Cool fire exposed containers with water spray from a protected location.</li> <li>▶ If safe to do so, remove containers from path of fire.</li> <li>▶ Equipment should be thoroughly decontaminated after use.</li> </ul> <p>Slight hazard when exposed to heat, flame and oxidisers.</p>
<b>Fire/Explosion Hazard</b>	<ul style="list-style-type: none"> <li>▶ Liquid and vapour are highly flammable.</li> <li>▶ Severe fire hazard when exposed to heat or flame.</li> <li>▶ Vapour forms an explosive mixture with air.</li> <li>▶ Severe explosion hazard, in the form of vapour, when exposed to flame or spark.</li> <li>▶ Vapour may travel a considerable distance to source of ignition.</li> <li>▶ Heating may cause expansion or decomposition with violent container rupture.</li> <li>▶ Aerosol cans may explode on exposure to naked flames.</li> <li>▶ Rupturing containers may rocket and scatter burning materials.</li> <li>▶ Hazards may not be restricted to pressure effects.</li> <li>▶ May emit acrid, poisonous or corrosive fumes.</li> <li>▶ On combustion, may emit toxic fumes of carbon monoxide (CO).</li> </ul>

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Combustion products include:  
 carbon monoxide (CO)  
 carbon dioxide (CO<sub>2</sub>)  
 acrolein  
 nitrogen oxides (NO<sub>x</sub>)  
 other pyrolysis products typical of burning organic material.  
 Articles and manufactured articles may constitute a fire hazard where polymers form their outer layers or where combustible packaging remains in place.  
 Certain substances, found throughout their construction, may degrade or become volatile when heated to high temperatures. This may create a secondary hazard.

### SECTION 6 Accidental release measures

#### Personal precautions, protective equipment and emergency procedures

See section 8

#### Environmental precautions

See section 12

#### Methods and material for containment and cleaning up

<b>Minor Spills</b>	<ul style="list-style-type: none"> <li>▶ Clean up all spills immediately.</li> <li>▶ Avoid breathing vapours and contact with skin and eyes.</li> <li>▶ Wear protective clothing, impervious gloves and safety glasses.</li> <li>▶ Shut off all possible sources of ignition and increase ventilation.</li> <li>▶ Wipe up.</li> <li>▶ If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated.</li> <li>▶ Undamaged cans should be gathered and stowed safely.</li> </ul>
<b>Major Spills</b>	<ul style="list-style-type: none"> <li>▶ Clear area of all unprotected personnel and move upwind.</li> <li>▶ Alert Emergency Authority and advise them of the location and nature of hazard.</li> <li>▶ May be violently or explosively reactive.</li> <li>▶ Wear full body clothing with breathing apparatus.</li> <li>▶ Prevent by any means available, spillage from entering drains and water-courses.</li> <li>▶ Consider evacuation.</li> <li>▶ Shut off all possible sources of ignition and increase ventilation.</li> <li>▶ No smoking or naked lights within area.</li> <li>▶ Use extreme caution to prevent violent reaction.</li> <li>▶ Stop leak only if safe to do so.</li> <li>▶ Water spray or fog may be used to disperse vapour.</li> <li>▶ <b>DO NOT enter confined space where gas may have collected.</b></li> <li>▶ Keep area clear until gas has dispersed.</li> <li>▶ Remove leaking cylinders to a safe place.</li> <li>▶ Fit vent pipes. Release pressure under safe, controlled conditions</li> <li>▶ Burn issuing gas at vent pipes.</li> <li>▶ <b>DO NOT exert excessive pressure on valve; DO NOT attempt to operate damaged valve.</b></li> <li>▶ Clear area of personnel and move upwind.</li> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ May be violently or explosively reactive.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water courses</li> <li>▶ No smoking, naked lights or ignition sources.</li> <li>▶ Increase ventilation.</li> <li>▶ Stop leak if safe to do so.</li> <li>▶ Water spray or fog may be used to disperse / absorb vapour.</li> <li>▶ Absorb or cover spill with sand, earth, inert materials or vermiculite.</li> <li>▶ If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated.</li> <li>▶ Undamaged cans should be gathered and stowed safely.</li> <li>▶ Collect residues and seal in labelled drums for disposal.</li> <li>▶ Clean up all spills immediately.</li> <li>▶ Wear protective clothing, safety glasses, dust mask, gloves.</li> <li>▶ Secure load if safe to do so. Bundle/collect recoverable product.</li> <li>▶ Use dry clean up procedures and avoid generating dust.</li> <li>▶ Vacuum up (consider explosion-proof machines designed to be grounded during storage and use).</li> <li>▶ Water may be used to prevent dusting.</li> <li>▶ Collect remaining material in containers with covers for disposal.</li> <li>▶ Flush spill area with water.</li> </ul>

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

### SECTION 7 Handling and storage

#### Precautions for safe handling

<b>Safe handling</b>	<p>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.</p>
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	<p>Natural gases contain a contaminant, radon-222, a naturally occurring radioactive gas. During subsequent processing, radon tends to concentrate in liquefied petroleum streams and in product streams having similar boiling points. Industry experience indicates that the commercial product may contain small amounts of radon-222 and its radioactive decay products (radon daughters). The actual concentration of radon-222 and radioactive daughters in process equipment (IE lines, filters, pumps and reactor units) may reach significant levels and produce potentially damaging levels of gamma radiation. A potential external radiation hazard exists at or near any pipe, valve or vessel containing a radon enriched stream or containing internal deposits of radioactive material. Field studies, however, have not shown that conditions exist that expose the worker to cumulative exposures in excess of general population limits. Equipment containing gamma-emitting decay products should be presumed to be internally contaminated with alpha-emitting decay products which may be hazardous if inhaled or ingested. During maintenance operations that require the opening of contaminated process equipment, the flow of gas should be stopped and a four hour delay enforced to allow gamma-radiation to drop to background levels. Protective equipment (including high efficiency particulate respirators (P3) suitable for radionucleotides or supplied air) should be worn by personnel entering a vessel or working on contaminated process equipment to prevent skin contamination or inhalation of any residue containing alpha-radiation. Airborne contamination may be minimised by handling scale and/or contaminated materials in a wet state. [TEXACO]</p> <ul style="list-style-type: none"> <li>▶ Avoid all personal contact, including inhalation.</li> <li>▶ Wear protective clothing when risk of exposure occurs.</li> <li>▶ Use in a well-ventilated area.</li> <li>▶ Prevent concentration in hollows and sumps.</li> <li>▶ <b>DO NOT enter confined spaces until atmosphere has been checked.</b></li> <li>▶ Avoid smoking, naked lights or ignition sources.</li> <li>▶ Avoid contact with incompatible materials.</li> <li>▶ <b>When handling, DO NOT eat, drink or smoke.</b></li> <li>▶ <b>DO NOT incinerate or puncture aerosol cans.</b></li> <li>▶ <b>DO NOT spray directly on humans, exposed food or food utensils.</b></li> <li>▶ Avoid physical damage to containers.</li> <li>▶ Always wash hands with soap and water after handling.</li> <li>▶ Work clothes should be laundered separately.</li> <li>▶ Use good occupational work practice.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.</li> </ul>
Other information	<ul style="list-style-type: none"> <li>▶ Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can</li> <li>▶ Store in original containers in approved flammable liquid storage area.</li> <li>▶ <b>DO NOT store in pits, depressions, basements or areas where vapours may be trapped.</b></li> <li>▶ No smoking, naked lights, heat or ignition sources.</li> <li>▶ Keep containers securely sealed. Contents under pressure.</li> <li>▶ Store away from incompatible materials.</li> <li>▶ Store in a cool, dry, well ventilated area.</li> <li>▶ Avoid storage at temperatures higher than 40 deg C.</li> <li>▶ Store in an upright position.</li> <li>▶ Protect containers against physical damage.</li> <li>▶ Check regularly for spills and leaks.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ Store away from incompatible materials.</li> </ul>

## Conditions for safe storage, including any incompatibilities

Suitable container	<p>Generally packaging as originally supplied with the article or manufactured item is sufficient to protect against physical hazards. If repackaging is required ensure the article is intact and does not show signs of wear. As far as is practicably possible, reuse the original packaging or something providing a similar level of protection to both the article and the handler.</p> <ul style="list-style-type: none"> <li>▶ Aerosol dispenser.</li> <li>▶ Check that containers are clearly labelled.</li> </ul>
Storage incompatibility	<p>Triethanolamine:</p> <ul style="list-style-type: none"> <li>· is an organic base</li> <li>· reacts violently with strong oxidisers, permanganates, peroxides, ammonium persulfate, bromine dioxide, sulfuric acid, nitric acid</li> <li>· is incompatible with organic anhydrides, acrylates, alcohols, aldehydes, alkylene oxides, substituted allyls, cellulose nitrate, cresols, caprolactam solution, epichlorohydrin, ethylene dichloride, isocyanates, ketones, glycols, mercury, nitrates, phenols, vinyl acetate</li> <li>· decomposes exothermically with maleic anhydride</li> <li>· increase the explosive sensitivity of nitromethane</li> <li>· corrodes, aluminium, copper, its alloys, tin, zinc</li> </ul> <p>Butane/ isobutane</p> <ul style="list-style-type: none"> <li>▶ reacts violently with strong oxidisers</li> <li>▶ reacts with acetylene, halogens and nitrous oxides</li> <li>▶ is incompatible with chlorine dioxide, conc. nitric acid and some plastics</li> <li>▶ may generate electrostatic charges, due to low conductivity, in flow or when agitated - these may ignite the vapour.</li> </ul> <p>Segregate from nickel carbonyl in the presence of oxygen, heat (20-40 C)</p> <p>Diethanolamine:</p> <ul style="list-style-type: none"> <li>▶ reacts vigorously with strong oxidisers</li> <li>▶ reacts with aldehydes, ketones, acrylates, formates, oxalates, nitrites, non-oxidising mineral acids, strong acids, organic acids, organic anhydrides, isocyanates, vinyl acetate, acrylates, substituted allyls, alkylene oxides, epichlorohydrin,</li> <li>▶ may undergo self-sustaining thermal decomposition at temperatures above 250 C</li> <li>▶ attacks aluminium, copper, zinc and their alloys, and galvanised iron</li> </ul> <p>Monoethanolamine</p> <ul style="list-style-type: none"> <li>▶ is a strong organic base</li> <li>▶ reacts violently with strong oxidisers, strong acids (with spattering)</li> <li>▶ is incompatible with acetic acid, acetic anhydride, acrolein, acrylates, acrylic acid, acrylonitrile, alcohols, aldehydes, alkali metals, alkylene oxides, substituted allyls, caprolactam solution, cellulose nitrate, chlorosulfonic acid, cresols, epichlorohydrin, glycols, halogenated hydrocarbons, isocyanates, ketones, mesityl oxide, oleum, organic anhydrides, phenols, beta-propiolactone, vinyl acetate</li> <li>▶ forms explosive mixture with sodium perchlorate</li> <li>▶ reacts with iron forming tris-ethanolamineiron</li> <li>▶ may undergo a self-sustaining thermal decomposition when heated in excess of 250 degrees C</li> <li>▶ attacks aluminium, copper, lead, tin, zinc, and their alloys</li> <li>▶ attacks plastics, coatings an rubber</li> </ul> <ul style="list-style-type: none"> <li>▶ Avoid strong acids, bases.</li> </ul>

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- ▶ Avoid contact with copper, aluminium and their alloys.
- ▶ Avoid reaction with oxidising agents
- ▶ Compressed gases may contain a large amount of kinetic energy over and above that potentially available from the energy of reaction produced by the gas in chemical reaction with other substances

## SECTION 8 Exposure controls / personal protection

## Control parameters

## Occupational Exposure Limits (OEL)

## INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
New Zealand Workplace Exposure Standards (WES)	triethanolamine	Triethanolamine	5 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	diethanolamine	Diethanolamine	3 ppm / 13 mg/m3	Not Available	Not Available	skin-Skin absorption
New Zealand Workplace Exposure Standards (WES)	sodium borate, pentahydrate	Borates, tetra, sodium salts: Anhydrous	1 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	sodium borate, pentahydrate	Borates, tetra, sodium salts: Pentahydrate	1 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	sodium borate, pentahydrate	Borates, tetra, sodium salts: Decahydrate	5 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	butane	Butane	800 ppm / 1900 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	propane	Propane	Not Available	Not Available	Not Available	Simple asphyxiant - may present an explosion hazard

## Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
triethanolamine	15 mg/m3	240 mg/m3	1,500 mg/m3
diethanolamine	3 mg/m3	28 mg/m3	130 mg/m3
sodium borate, pentahydrate	6 mg/m3	190 mg/m3	1,100 mg/m3
sodium borate, pentahydrate	6 mg/m3	88 mg/m3	530 mg/m3
butane	Not Available	Not Available	Not Available
propane	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
triethanolamine	Not Available	Not Available
diethanolamine	Not Available	Not Available
fatty acids, C16-18 and C18-unsaturated	Not Available	Not Available
sodium borate, pentahydrate	Not Available	Not Available
butane	Not Available	1,600 ppm
propane	2,100 ppm	Not Available

## MATERIAL DATA

For butane:

Odour Threshold Value: 2591 ppm (recognition)

Butane in common with other homologues in the straight chain saturated aliphatic hydrocarbon series is not characterised by its toxicity but by its narcosis-inducing effects at high concentrations. The TLV is based on analogy with pentane by comparing their lower explosive limits in air. It is concluded that this limit will protect workers against the significant risk of drowsiness and other narcotic effects.

Odour Safety Factor(OSF)

OSF=0.22 (n-BUTANE)

for triethanolamine:

Exposure at or below the TLV-TWA is thought to minimise the potential for skin and eye irritation, and acute effects (including liver, kidney and nerve damage) and chronic effects (including cancer and allergic contact dermatitis).

Odour Safety Factor (OSF)

OSF=0.77 (triethanolamine)

for diethanolamine:

Odour Threshold: 2.6 ppm

The TLV-TWA is thought to be protective against the significant risk of eye damage and skin irritation.

Odour Safety Factor (OSF)

OSF=1.7 (DIETHANOLAMINE)

For propane

Odour Safety Factor(OSF)

OSF=0.16 (PROPANE)


## Exposure controls

## Appropriate engineering controls

Articles or manufactured items, in their original condition, generally don't require engineering controls during handling or in normal use. Exceptions may arise following extensive use and subsequent wear, during recycling or disposal operations where substances, found in the article, may be released to the environment.

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## GEA FOAM MARK PINK AEROSOL

	<p>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:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection.</p> <p>Provide adequate ventilation in warehouse or closed storage areas.</p> <p>Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p> <table border="1" data-bbox="389 521 1437 622"> <tr> <td>Type of Contaminant:</td> <td>Speed:</td> </tr> <tr> <td>aerosols, (released at low velocity into zone of active generation)</td> <td>0.5-1 m/s</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="389 678 1090 846"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	Type of Contaminant:	Speed:	aerosols, (released at low velocity into zone of active generation)	0.5-1 m/s	direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use	4: Large hood or large air mass in motion	4: Small hood-local control only
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<p><b>Eye and face protection</b></p>	<ul style="list-style-type: none"> <li>▶ Safety glasses with side shields.</li> <li>▶ Chemical goggles.</li> <li>▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]</li> <li>▶ Close fitting gas tight goggles</li> </ul> <p><b>DO NOT wear contact lenses.</b></p> <ul style="list-style-type: none"> <li>▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]</li> </ul> <p>No special equipment required due to the physical form of the product.</p>																
<p><b>Skin protection</b></p>	<p>See Hand protection below</p>																
<p><b>Hands/feet protection</b></p>	<ul style="list-style-type: none"> <li>▶ No special equipment needed when handling small quantities.</li> <li>▶ <b>OTHERWISE:</b></li> <li>▶ For potentially moderate exposures:</li> <li>▶ Wear general protective gloves, eg. light weight rubber gloves.</li> <li>▶ For potentially heavy exposures:</li> <li>▶ Wear chemical protective gloves, eg. PVC. and safety footwear.</li> </ul> <p>No special equipment required due to the physical form of the product.</p>																
<p><b>Body protection</b></p>	<p>See Other protection below</p>																
<p><b>Other protection</b></p>	<ul style="list-style-type: none"> <li>▶ The clothing worn by process operators insulated from earth may develop static charges far higher (up to 100 times) than the minimum ignition energies for various flammable gas-air mixtures. This holds true for a wide range of clothing materials including cotton.</li> <li>▶ Avoid dangerous levels of charge by ensuring a low resistivity of the surface material worn outermost.</li> </ul> <p>BREThERICK: Handbook of Reactive Chemical Hazards.</p> <p>No special equipment needed when handling small quantities.</p> <p><b>OTHERWISE:</b></p> <ul style="list-style-type: none"> <li>▶ Overalls.</li> <li>▶ Skin cleansing cream.</li> <li>▶ Eyewash unit.</li> <li>▶ Do not spray on hot surfaces.</li> </ul> <p>No special equipment required due to the physical form of the product.</p>																



## GEA FOAM MARK PINK AEROSOL

## Recommended material(s)

## GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:

GEA FOAM MARK PINK AEROSOL

Material	CPI
BUTYL	A
NATURAL RUBBER	A
NATURAL+NEOPRENE	A
NEOPRENE	A
NITRILE	A
PVC	A
NEOPRENE/NATURAL	C
PVA	C
TEFLON	C
VITON	C

\* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

**NOTE:** As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

\* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

## Respiratory protection

Respiratory protection not normally required due to the physical form of the product.

► Generally not applicable.

Aerosols, in common with most vapours/ mists, should never be used in confined spaces without adequate ventilation. Aerosols, containing agents designed to enhance or mask smell, have triggered allergic reactions in predisposed individuals.

## SECTION 9 Physical and chemical properties

## Information on basic physical and chemical properties

Appearance	SOFT PINK AEROSOL FOAM WITH A MILD ODOUR		
Physical state	article	Relative density (Water= 1)	0.934
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	431
pH (as supplied)	9	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Available
Flash point (°C)	-81	Taste	Not Available
Evaporation rate	Not Available BuAC = 1	Explosive properties	Not Available
Flammability	HIGHLY FLAMMABLE.	Oxidising properties	Not Available
Upper Explosive Limit (%)	10	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	1.5	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

## SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> <li>► Elevated temperatures.</li> <li>► Presence of open flame.</li> <li>► Product is considered stable.</li> <li>► Hazardous polymerisation will not occur.</li> </ul>
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7



## GEA FOAM MARK PINK AEROSOL

## Hazardous decomposition products

See section 5

## SECTION 11 Toxicological information

## Information on toxicological effects

Inhaled	<p>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.</p> <p>High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations. No health effects were seen in humans exposed at 1,000 ppm isobutane for up to 8 hours or 500 ppm for 8 hours/day for 10 days. Isobutane can have anaesthetic and asphyxiant effects at high concentrations, well above the lower explosion limit of 1.8% (18,000 ppm). Butane is a simple asphyxiant and is mildly anaesthetic at high concentrations (20-25%). 10000 ppm for 10 minutes causes drowsiness. Narcotic effects may be accompanied by exhilaration, dizziness, headache, nausea, confusion, incoordination and unconsciousness in severe cases</p> <p>The paraffin gases C1-4 are practically nontoxic below the lower flammability limit, 18,000 to 50,000 ppm; above this, low to moderate incidental effects such as CNS depression and irritation occur, but are completely reversible upon cessation of the exposure.</p> <p>The vapour is discomforting</p> <p><b>WARNING: Intentional misuse by concentrating/inhaling contents may be lethal.</b></p> <p>When rats (both sexes) were exposed to statically generated triethanolamine (25 deg. C) for six hours, there were no major signs nor was there any gross pathology (Kill rate 0/6).</p>
Ingestion	<p>Not normally a hazard due to physical form of product.</p> <p>Considered an unlikely route of entry in commercial/industrial environments</p> <p>Ingestion of triethanolamine may cause gastro-intestinal irritation with haemorrhage and congestion of intestines. May be fatal if swallowed.</p> <p>Calculated median lethal dose in 70 kg man is 560 gms. [ICI]</p> <p>Triethanolamine may cause burning or painful sensations in the mouth, throat, chest and abdomen, vomiting and diarrhoea.</p> <p>In rats where triethanolamine was administered orally, major toxic signs included sluggishness, lachrymation, piloerection, unsteady gait, diarrhoea and red or brown discharge on perinasal and perigenital hair. Gross pathological investigations revealed discoloured lungs, stomach, intestines and a dark red liquid in the stomach and intestine</p>
Skin Contact	<p>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.</p> <p>Spray mist may produce discomfort</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p> <p>Brief contact with triethanolamine may cause slight irritation with itching and local redness. Prolonged contact may produce more severe irritation with discomfort, or pain, localised redness and swelling (oedema) and possible tissue destruction. Skin contact may produce sensitisation in a small proportion of individuals. Covered patch testing resulted in a small percentage of subjects who displayed signs of allergic contact dermatitis (BIBRA Toxicology Profile, 1990). Triethanolamine has also been identified as the cause of erythematous vesicular lesions, eczema, non-allergic contact dermatitis and irritation amongst workers.</p> <p>Rabbits exposed percutaneously to toxic levels of triethanolamine showed sluggishness, unsteady gait and emaciation. Gross pathology consisted of discoloured lungs, thymus, spleen, kidneys, stomach and, gas and/ or liquid-filled intestines.</p> <p>Guinea pigs exposed dermally to triethanolamine (8 g/kg/day, 5 days/week applied to shaved and subsequently bandaged skin), died between the second and seventeenth application. Necrosis of the epithelium was observed. Kidneys and liver showed cloudy swelling and congestion; fatty changes were seen in the central ascini of the liver, and lung and adrenal congestion were observed.</p>
Eye	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals.</p> <p>Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p> <p>Direct contact with the eye may not cause irritation because of the extreme volatility of the gas; however concentrated atmospheres may produce irritation after brief exposures..</p>
Chronic	<p>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.</p> <p>Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.</p> <p>Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.</p>

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## GEA FOAM MARK PINK AEROSOL

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

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

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

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

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

Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses. Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.

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

#### Animal studies:

No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) was observed in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar

naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of a alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human. Principal route of occupational exposure to the gas is by inhalation.

On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

Reversible liver and kidney damage has been demonstrated in animals from chronic exposure to triethanolamine.

Although the product is not, in itself, carcinogenic, reaction under strong acid conditions, with nitrites and nitrous acids results in the formation of a potent carcinogen, N-nitrosodiethanolamine.

This situation might be encountered in certain metal-treatment operations, for example.

A cohort study, in which cancer morbidity and mortality were investigated in workers exposed to cutting fluids with nitrates and amines (amongst

## GEA FOAM MARK PINK AEROSOL

	them triethanolamine), had negative results. The effects on workers industrially exposed to metal-working coolants containing sodium nitrite and triethanolamine solutions were investigated in a Russian study. Observed vascular effects were attributed to sodium nitrite; no effects were attributed to triethanolamine. Screening studies in mice suggest that the material does not effect foetal development. In the European Union, trialkylamines, trialkanolamines, and their salts (ingredients containing TEA) may only be used up to 2.5%, must be at least 99% pure, are not to be used with nitrosating systems, must have a maximum secondary amine content of 0.5%, must have a maximum nitrosamine content of 50 ug/kg, and must be kept in nitrite-free containers	
GEA FOAM MARK PINK AEROSOL	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
triethanolamine	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >11 mg/kg <sup>[2]</sup>	Eye (rabbit): 0.1 ml -
	Oral(Rat) LD50; 4.354 mg/kg <sup>[2]</sup>	Eye (rabbit): 10 mg - mild
		Eye (rabbit): 5.62 mg - SEVERE
		minor conjunctival irritation
		no irritation *
		Skin (human): 15 mg/3d (int)-mild
		Skin (rabbit): 4 h occluded
	Skin (rabbit): 560 mg/24 hr- mild	
diethanolamine	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 12200 mg/kg <sup>[2]</sup>	Eye (rabbit): 5500 mg - SEVERE
	Oral(Rat) LD50; 0.568 mg/kg <sup>[1]</sup>	Eye (rabbit):0.75 mg/24 hr SEVERE
		Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin (rabbit): 50 mg (open)-mild
		Skin (rabbit): 500 mg/24 hr-mild
	Skin: adverse effect observed (irritating) <sup>[1]</sup>	
fatty acids, C16-18 and C18-unsaturated	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
sodium borate, pentahydrate	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral(Rat) LD50; 2660 mg/kg <sup>[2]</sup>	Eye (rabbit) 100 mg - SEVERE
		Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
butane	<b>TOXICITY</b>	<b>IRRITATION</b>
	Inhalation(Rat) LC50; 658 mg/4h <sup>[2]</sup>	Not Available
propane	<b>TOXICITY</b>	<b>IRRITATION</b>
	Inhalation(Rat) LC50; >13023 ppm4h <sup>[1]</sup>	Not Available
<b>Legend:</b>	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. * Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

<b>TRIETHANOLAMINE</b>	<p>Lachrymation, diarrhoea, convulsions, urinary tract changes, changes in bladder weight, changes in testicular weight, changes in thymus weight, changes in liver weight, dermatitis after systemic exposure, kidney, ureter, bladder tumours recorded. Equivocal tumourigen by RTECS criteria. Dermal rabbit value quoted above is for occluded patch in male or female animals * Union Carbide</p> <p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p> <p>For triethanolamine (and its salts):</p> <p><b>Acute toxicity:</b> Triethanolamine is of low toxicity by the oral, dermal and inhalation routes of exposure. Oral LD50 values have been shown to range from approximately 5-10 g/kg. The dermal LD50 is greater than 2 g/kg. The inhalation LC50 is greater than a saturated atmosphere</p> <p><b>Repeat Dose Toxicity:</b> The studies to determine toxicity of triethanolamine from repeated exposure were conducted for a duration of 91 days or 2 years. In both studies the NOAEL was at least 1000 mg/kg. There was no evidence of gross or histopathological change that could be attributed to treatment. Also, triethanolamine was shown to be non-carcinogenic.</p> <p><b>Genetic Toxicity:</b> Mutation (bacterial); This endpoint has been satisfied by two studies using 4 strains (TA 98, TA 100, TA 1535 and TA 1537) of <i>Salmonella typhimurium</i>. Triethanolamine was not mutagenic in any of the tester strains.</p> <p>Chromosomal aberration (mammalian, <i>in vitro</i>) – This endpoint was satisfied by a cytogenetic assay using Chinese hamster lung cells. Triethanolamine did not induce chromosome aberrations in this test system.</p> <p><b>Reproductive Toxicity:</b> No studies have been conducted to specifically evaluate the effect of triethanolamine on reproductive performance. However, based on consideration of the repeat dose toxicity studies of at least 90 days duration, there were no abnormalities noted in the histopathological examination of reproductive organs. This fact, and the lack of effects on foetal development, allow the conclusion that triethanolamine would not be expected to produce adverse effects to reproductive performance and fertility.</p> <p><b>Developmental Toxicity:</b> This endpoint was satisfied using a developmental toxicity screening study according to the Chernoff-Kavlock method.</p>
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	<p>Based on the results from this test, triethanolamine does not impair development of the fetus.</p> <p>A Cosmetic Ingredient Review (CIR) expert panel conducted a review of triethanolamine-containing personal care products. The panel was concerned with the levels of free diethanolamine that could be present as an impurity in TEA or TEA-containing ingredients. The panel stated that the amount of free diethanolamine available must be limited to the present practices of use and concentration of diethanolamine.</p> <p>The Panel concluded that TEA and 31 related TEA-containing ingredients, are safe when formulated to be nonirritating and when the levels of free diethanolamine do not exceed the prescribed levels. These ingredients should not be used in cosmetic products in which N-nitroso compounds can be formed.</p> <p>Dermal carcinogenicity studies performed by the NTP on TEA reported equivocal evidence of carcinogenic activity in male mice based on the occurrence of liver hemangiosarcoma, some evidence of carcinogenic activity in female mice based on increased incidences of hepatocellular adenoma, and equivocal evidence of carcinogenic activity in male rats based on a marginal increase in the incidence of renal tubule cell adenoma. It has been hypothesized that TEA may cause liver tumours in mice via a choline-depletion mode of action. Humans are much less sensitive to this deficiency, and these hepatic findings are considered to have little relevance to humans regarding the safety of the use of TEA in personal care products.</p> <p>The panel was concerned that the potential exists for dermal irritation with the use of products formulated using TEA or TEA-related ingredients. The panel specified that products containing these ingredients must be formulated to be nonirritating.</p> <p>Tertiary alkyl amines such as TEA do not react with N-nitrosating agents to directly form nitrosamines. However, tertiary amines can act as precursors in nitrosamine formation by undergoing nitrosative cleavage. The resultant secondary amine (ie, diethanolamine) can then be N-nitrosated to products that may be carcinogenic. Because of the potential for this process to occur, TEA and TEA-containing ingredients should not be used in cosmetic products in which N-nitroso compounds can be formed.</p> <p>Safety Assessment of Triethanolamine and Triethanolamine-Containing Ingredients as Used in Cosmetics: International Journal of Toxicology (supplement 1) 59S-83S. 2013  <a href="http://citeseerx.ist.psu.edu/viewdoc/download?doi=10.1.1.901.4174&amp;rep=rep1&amp;type=pdf">http://citeseerx.ist.psu.edu/viewdoc/download?doi=10.1.1.901.4174&amp;rep=rep1&amp;type=pdf</a></p> <p>The substance is classified by IARC as Group 3:  <b>NOT</b> classifiable as to its carcinogenicity to humans.  Evidence of carcinogenicity may be inadequate or limited in animal testing.  <b>NOTE:</b> Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.</p>
DIETHANOLAMINE	<p>for diethanolamine (DEA):</p> <p>In animal studies, DEA has low acute toxicity via the oral and dermal routes with moderate skin irritation and severe eye irritation. In subchronic toxicity testing conducted via the oral route in rats and mice, the main effects observed were increased organ weights and histopathology of the kidney and/or liver, with the majority of other tissue effects noted only at relatively high dosages. In subchronic studies conducted via the dermal route, skin irritation was noted as well as systemic effects similar to those observed in the oral studies. DEA has not been shown to be mutagenic or carcinogenic in rats; however, there is evidence of its carcinogenicity in mice.</p> <p><b>Subchronic toxicity:</b> The subchronic toxicity of DEA has been studied in F344 rats and B6C3F1 mice by exposure through drinking water or dermal administration, in 2 week and 13 week studies.</p> <p>Target organs for toxicity included blood, kidney, brain and spinal cord, seminiferous tubules and dermal application site in rats and liver, kidney, heart, salivary gland and dermal application site in mice. Effects on seminiferous tubules were accompanied by reductions in sperm count and reduced sperm motility. Hematological evaluations indicated normochromic, microcytic anemia in the dermal study in male rats (NOEL =32 mg/g) and females (LOEL = 32 mg/kg). Anemia was also observed in rats in the drinking water study with a LOEL of 14 mg/kg/d in females and a LOEL of 48 mg/kg/d in males for altered hematological parameters. These findings were similar to those observed in the 2 week studies, but the magnitude of the changes was greater in the 13 week studies. Hematological parameters were normal in controls. No associated histopathological changes were noted in femoral bone marrow. Haematological parameters were not evaluated in mice.</p> <p><b>Developmental toxicity:</b> In a developmental toxicity study conducted via the oral route, effects of concern were observed only in the presence of maternal toxicity. In a developmental toxicity study conducted via the dermal route using two species of mammals, developmental toxicity was observed only in one species and only at doses causing significant maternal toxicity. Metabolically, DEA is excreted largely unchanged in the urine.</p> <p><b>Carcinogenicity:</b> A two-year dermal cancer study bioassay results on DEA and three fatty acid condensates of DEA indicated that liver tumours occurred in male and female mice exposed to DEA and two of the condensates. In addition kidney tumours occurred in male mice exposed to DEA and one of the condensates. Compelling evidence suggested that the toxicity observed in mice and rats treated with the DEA condensates was associated with free DEA and not with other components of the condensates. A weight of evidence analysis of data relevant to the assessment of the liver and kidney tumours in mice resulted in the conclusion that these tumours are not relevant to humans under the expected conditions of exposure and that liver and kidney toxicity should be evaluated on a threshold basis. This conclusion is based on the following:</p> <ul style="list-style-type: none"> <li>▶ DEA is not genotoxic</li> <li>▶ tumour development occurred at doses also associated with chronic hyperplasia</li> <li>▶ there was no dose-related increase in malignancy, multiplicity of tumours or decrease in latency period</li> <li>▶ tumours occurred late in life</li> <li>▶ tumour response was species-specific (only mice were affected, not rats)</li> <li>▶ tumour response was sex-specific (only male mice were affected, not females)</li> <li>▶ tumour development was site-specific, with only liver and kidney affected, both sites of DEA accumulation;</li> <li>▶ there was no tumour response in skin, despite evidence of chronic dermal toxicity</li> <li>▶ there is a plausible mechanism, supported by various data, to explain the renal toxicity of DEA</li> <li>▶ data support threshold mechanisms of renal carcinogenesis for a number of non-genotoxic chemicals</li> <li>▶ the exposure regime used in the mouse study (<i>i.e.</i>, lifetime continuous exposure to DEA in ethanol vehicle at doses causing chronic dermal toxicity) is not relevant to human exposure (exposure through cosmetic vehicles with daily removal, under non-irritating conditions).</li> </ul> <p>In considering the aggregate data on a DEA basis from the four studies using DEA and related condensates, the NOEL for kidney toxicity was 19 mg/kg/d, which resulted from a dose of 100 mg/kg/d of cocamide DEA containing 19% free DEA.</p> <p><b>Anaemia:</b> Rats exposed to DEA condensates developed anaemia. This was considered to be of to be relevant for humans since anaemia in rodents and humans share common etiologies. The proposed mechanism by which DEA could cause anaemia involves disruption of phospholipid metabolism leading to membrane perturbation and functional change to erythrocytes. Some doubt about the relevance of the findings arises because ethanol was used as the vehicle in the dermal studies, and ethanol is known to cause anaemia in rodents through a mechanism involving membrane disruption. The possibility of a synergistic or additive role for DEA and ethanol in combination cannot be ruled out.</p> <p>In considering the aggregate data on a DEA basis from the four 13-week dermal studies using DEA and related condensates, the NOEL for microcytic anemia was 9.5 mg/kg/d, which resulted from a dose of 50 mg/kg/d of cocamide DEA containing 19% free DEA.</p> <p>The NOELs for mice and rats derived in this hazard assessment were as follows:  Anaemia in rats: 9.5 mg/kg/d (based on microcytic anemia)  Organ toxicity in mice: 2.2 mg/kg/d (based on liver toxicity)</p> <p>In extrapolating among species for the purposes of risk assessment, the prime consideration with respect to dermally applied DEA was differential dermal absorption. Evidence indicates that dermal penetration of DEA is greatest in mice and lower in rats and humans. Interspecies extrapolation was accomplished in this assessment by converting applied doses to bioavailable doses (<i>i.e.</i>, internal doses) using dermal bioavailability determined in studies with rats and mice <i>in vivo</i>, so as to be able to compare these with internal doses expected to be experienced by humans through use of personal care products.</p> <p>Based on measured bioavailability in mice and rats, the bioavailable NOELs corresponding to the foregoing were:  Anaemia in rats: 0.8 mg/kg/d (based on microcytic anemia)  Organ toxicity in mice: 0.55 mg/kg/d (based on liver toxicity)</p> <p><b>Kidney toxicity:</b> Effects on the kidney were observed in rats treated with DEA in drinking water or by dermal exposure after as little as 2 weeks of exposure. Effects included renal tubule hyperplasia, renal tubular epithelial necrosis, renal tubule mineralization and increased relative organ weight. Similar changes were observed after 13 weeks of exposure of rats to DEA in drinking water and by dermal administration. The NOEL in</p>

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	<p>male rats was 250 mg/kg/d in the dermal study, while in female rats renal tubule mineralisation was observed at the lowest dose of 32 mg/kg/d. After 2 years of dermal exposure there were no histopathological changes in the kidneys of male rats given doses of up to 64 mg/kg/d. In females, there were no significant increases in the incidences of renal tubule epithelial necrosis, hyperplasia or mineralisation as was observed after 13 weeks of exposure, however, there was an increase in the severity and incidence of nephropathy. This was the result of a treatment-related exacerbation of a previously existing lesion, since the incidence in controls was 80%, increasing to 94-96% in treated groups. There was no significant increase in the incidence of kidney tumours in rats treated with DEA or any of the condensates in 2-year dermal studies.</p> <p><b>Liver toxicity:</b> Effects on liver, including increases in relative organ weight and histopathological changes were observed in male and female mice in the 2 week drinking water study with DEA. Increases in liver weight were observed in the two week dermal study, but were not associated with histopathological changes. After 13 weeks of exposure, relative liver weights were increased compared to controls in male and female rats, with no associated histopathology. There is some doubt about whether these changes in liver weights were of toxicological significance, since there was no associated histopathology, the dose-response was not consistent and there were no effects on liver in the 2 year study in rats. In the study with coconut diethanolamide (CDEA) (100 and 200 mg/kg/d) in which 19% of the applied dose was DEA, there were no liver effects in rats after 13 weeks or 2 years of dermal exposure. No liver toxicity in rats was observed in the 2 year dermal studies of lauramide or oleamide DEA</p> <p><b>WARNING:</b> This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.</p>
<p><b>FATTY ACIDS, C16-18 AND C18-UNSATURATED</b></p>	<p>Fatty acid salts are of low acute toxicity. Their skin and eye irritation potential is chain length dependent and decreases with increasing chain length - they are poorly absorbed through the skin nor are they skin sensitisers. The available repeated dose toxicity data demonstrate the low toxicity of the fatty acids and their salts. Also, they are not considered to be mutagenic, genotoxic or carcinogenic, and are not reproductive or developmental toxicants. Accidental ingestion of fatty acid salt containing detergent products is not expected to result in any significant adverse health effects. This assessment is based on toxicological data demonstrating the low acute oral toxicity of fatty acid salts and the fact that not a single fatality has been reported in the UK following accidental ingestion of detergents containing fatty acid salts. Also in a report published by the German Federal Institute for Health Protection of Consumers and Veterinary Medicine, detergent products were not mentioned as dangerous products with a high incidence of poisoning. The estimated total human exposure to fatty acid salts, from the different exposure scenarios for the handling and use of detergent products containing fatty acid salts, showed a margin of exposure (MOE) of 258,620. This extremely large MOE is large enough to be reassuring with regard to the relatively small variability of the hazard data on which it is based. Also, in the UK, the recommended dietary fatty acid intake by the Department of Health is about 100 g of fatty acids per day or 1.7 g (1700 mg) of fatty acids per kilogram body weight per day. This exposure is several orders of magnitude above that resulting from exposure to fatty acid salts in household cleaning products. Based on the available data, the use of fatty acid salts in household detergent and cleaning products does not raise any safety concerns with regard to consumer</p>
<p><b>SODIUM BORATE, PENTAHYDRATE</b></p>	<p>for sodium borate, decahydrate. Reproductive effector in rats Mutagenic towards bacteria</p>
<p><b>TRIETHANOLAMINE &amp; DIETHANOLAMINE &amp; SODIUM BORATE, PENTAHYDRATE</b></p>	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.</p>
<p><b>TRIETHANOLAMINE &amp; DIETHANOLAMINE</b></p>	<p>While it is difficult to generalise about the full range of potential health effects posed by exposure to the many different amine compounds, characterised by those used in the manufacture of polyurethane and polyisocyanurate foams, it is agreed that overexposure to the majority of these materials may cause adverse health effects.</p> <ul style="list-style-type: none"> <li>▶ Many amine-based compounds can induce histamine liberation, which, in turn, can trigger allergic and other physiological effects, including bronchoconstriction or bronchial asthma and rhinitis.</li> <li>▶ Systemic symptoms include headache, nausea, faintness, anxiety, a decrease in blood pressure, tachycardia (rapid heartbeat), itching, erythema (reddening of the skin), urticaria (hives), and facial edema (swelling). Systemic effects (those affecting the body) that are related to the pharmacological action of amines are usually transient.</li> </ul> <p>Typically, there are four routes of possible or potential exposure: inhalation, skin contact, eye contact, and ingestion.</p> <p><b>Inhalation:</b></p> <p>Inhalation of vapors may, depending upon the physical and chemical properties of the specific product and the degree and length of exposure, result in moderate to severe irritation of the tissues of the nose and throat and can irritate the lungs. Products with higher vapour pressures have a greater potential for higher airborne concentrations. This increases the probability of worker exposure. Higher concentrations of certain amines can produce severe respiratory irritation, characterised by nasal discharge, coughing, difficulty in breathing, and chest pains. Chronic exposure via inhalation may cause headache, nausea, vomiting, drowsiness, sore throat, bronchopneumonia, and possible lung damage. Also, repeated and/or prolonged exposure to some amines may result in liver disorders, jaundice, and liver enlargement. Some amines have been shown to cause kidney, blood, and central nervous system disorders in laboratory animal studies.</p> <p>While most polyurethane amine catalysts are not sensitizers, some certain individuals may also become sensitized to amines and may experience respiratory distress, including asthma-like attacks, whenever they are subsequently exposed to even very small amounts of vapor. Once sensitized, these individuals must avoid any further exposure to amines. Although chronic or repeated inhalation of vapor concentrations below hazardous or recommended exposure limits should not ordinarily affect healthy individuals, chronic overexposure may lead to permanent pulmonary injury, including a reduction in lung function, breathlessness, chronic bronchitis, and immunologic lung disease. Inhalation hazards are increased when exposure to amine catalysts occurs in situations that produce aerosols, mists, or heated vapors. Such situations include leaks in fitting or transfer lines. Medical conditions generally aggravated by inhalation exposure include asthma, bronchitis, and emphysema.</p> <p><b>Skin Contact:</b></p> <p>Skin contact with amine catalysts poses a number of concerns. Direct skin contact can cause moderate to severe irritation and injury-i.e., from simple redness and swelling to painful blistering, ulceration, and chemical burns. Repeated or prolonged exposure may also result in severe cumulative dermatitis.</p> <p>Skin contact with some amines may result in allergic sensitization. Sensitized persons should avoid all contact with amine catalysts. Systemic effects resulting from the absorption of the amines through skin exposure may include headaches, nausea, faintness, anxiety, decrease in blood pressure, reddening of the skin, hives, and facial swelling. These symptoms may be related to the pharmacological action of the amines, and they are usually transient.</p> <p><b>Eye Contact:</b></p> <p>Amine catalysts are alkaline in nature and their vapours are irritating to the eyes, even at low concentrations. Direct contact with the liquid amine may cause severe irritation and tissue injury, and the "burning" may lead to blindness. (Contact with solid products may result in mechanical irritation, pain, and corneal injury.) Exposed persons may experience excessive tearing, burning, conjunctivitis, and corneal swelling.</p>



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	<p>The corneal swelling may manifest itself in visual disturbances such as blurred or "foggy" vision with a blue tint ("blue haze") and sometimes a halo phenomenon around lights. These symptoms are transient and usually disappear when exposure ceases.</p> <p>Some individuals may experience this effect even when exposed to concentrations below doses that ordinarily cause respiratory irritation.</p> <p><b>Ingestion:</b></p> <p>The oral toxicity of amine catalysts varies from moderately to very toxic.</p> <p>Some amines can cause severe irritation, ulceration, or burns of the mouth, throat, esophagus, and gastrointestinal tract.</p> <p>Material aspirated (due to vomiting) can damage the bronchial tubes and the lungs.</p> <p>Affected persons also may experience pain in the chest or abdomen, nausea, bleeding of the throat and the gastrointestinal tract, diarrhea, dizziness, drowsiness, thirst, circulatory collapse, coma, and even death.</p> <p><b>Polyurethane Amine Catalysts: Guidelines for Safe Handling and Disposal; Technical Bulletin June 2000</b></p> <p><b>Alliance for Polyurethanes Industry</b></p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>
<b>TRIETHANOLAMINE &amp; SODIUM BORATE, PENTAHYDRATE</b>	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
<b>FATTY ACIDS, C16-18 AND C18-UNSATURATED &amp; PROPANE</b>	No significant acute toxicological data identified in literature search.

<b>Acute Toxicity</b>	✗	<b>Carcinogenicity</b>	✗
<b>Skin Irritation/Corrosion</b>	✗	<b>Reproductivity</b>	✓
<b>Serious Eye Damage/Irritation</b>	✓	<b>STOT - Single Exposure</b>	✗
<b>Respiratory or Skin sensitisation</b>	✗	<b>STOT - Repeated Exposure</b>	✓
<b>Mutagenicity</b>	✗	<b>Aspiration Hazard</b>	✗

**Legend:** ✗ – Data either not available or does not fill the criteria for classification  
 ✓ – Data available to make classification

## SECTION 12 Ecological information

## Toxicity

GEA FOAM MARK PINK AEROSOL	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
triethanolamine	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48	Crustacea	565.2-658.3mg/l	4
	LC50	96	Fish	11800mg/l	2
	BCF	1008	Fish	<0.4	7
	EC10(ECx)	96	Algae or other aquatic plants	7.1 mg/l	1
	EC50	72	Algae or other aquatic plants	>107<260mg/l	2
diethanolamine	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48	Crustacea	28.8mg/l	1
	LC50	96	Fish	>2.322mg/L	4
	EC50	72	Algae or other aquatic plants	2.7mg/l	2
	NOEC(ECx)	72	Algae or other aquatic plants	0.6mg/l	2
fatty acids, C16-18 and C18-unsaturated	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
	EC50	48	Crustacea	1332-2135mg/l	4
	EC50(ECx)	48	Crustacea	1332-2135mg/l	4
	LC50	96	Fish	29.627mg/L	4
sodium borate, pentahydrate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72	Algae or other aquatic plants	40.2mg/l	2
	NOEC(ECx)	768	Fish	0.009mg/l	2
	EC50	96	Algae or other aquatic plants	2.6-21.8mg/l	4
	LC50	96	Fish	24.11mg/l	2
	EC50(ECx)	96	Algae or other aquatic plants	7.71mg/l	2
butane	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	96	Algae or other aquatic plants	7.71mg/l	2
	EC50	96	Algae or other aquatic plants	7.71mg/l	2

Continued...

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propane	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	96	Algae or other aquatic plants	7.71mg/l	2
	LC50	96	Fish	24.11mg/l	2
	EC50	96	Algae or other aquatic plants	7.71mg/l	2

**Legend:** *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*

For aliphatic fatty acids and alcohols:

Environmental fate:

Saturated fatty acids are very stable in air, whereas unsaturated (C=C bonds) fatty acids are susceptible to oxidation.

Unsaturation increases the rate of metabolism although the degree of unsaturation and positioning of double bonds is not highly significant.

The available data indicate all fatty acid salt chain lengths up to and including C18 can be metabolised under aerobic conditions and can be considered to be readily biodegradable

All tests showed that fatty acids and lipids are readily biodegradable

The aliphatic acids are of similar very weak acid strength (approximately pKa 5), i.e., partially dissociate in aqueous solution; the salts of the aliphatic acids are highly dissociated in water solution such that the anion is the same for homologous salts and acids.

Slight (although inconsistent) effects on the trend for decreasing vapour pressure are also observed with the mono-, di- and tri-unsaturated substances as compared to the corresponding saturated substances.

Dicarboxylic acids: Compared to their corresponding single acid substances (C8-10 single component, saturated), the dicarboxylic acids exhibit modestly higher melting/ boiling points and water solubility, and lower partition coefficients and vapour pressures. The trends described above for changes in physical chemical properties with increasing carbon chain length apply.

Salts: As expected, the salts differ in physical / chemical properties as compared to their homologous single component substances. However the trends described above for single components with regard to changes in physical chemical properties with increasing carbon chain length apply

Models also indicate that the aliphatic acids will distribute primarily to soil and water, with lesser amounts to air and sediment. With increasing chain length, the percent distributions to soil and sediment generally increase and the percent distributions to water and air generally decrease.

The rate of degradation of fatty acids was investigated in two non-GLP studies.

The total fatty acids residue exhibits low persistence in soil. From the pattern of peaks decline, it was hypothesised a degradation pathway by the sequential elimination of C2 fragments. Consequently, the major soil metabolites of a given fatty acid would be other fatty acids with shorter chains.

Although mineralisation was not measured in these experiments, formation of CO<sub>2</sub> is the expected terminal step of this process. Fatty acids undergo aerobic biodegradation by the process of beta-oxidation. Beta-oxidation of the parent fatty acid forms acetate and a new fatty acid of two less carbon atoms. This process repeats itself until the compound is completely broken down. The hydrocarbon will eventually be degraded to CO<sub>2</sub> and H<sub>2</sub>O. For this reason, the length of the fatty acid chain does not preclude biodegradation, but it may take longer to achieve complete mineralisation. The beta-oxidation sequence does not necessarily require the presence of molecular oxygen, and fatty acid biodegradation may proceed under anaerobic conditions.

Hydrolysis is not an important fate path in the environment due to the fact that the substances lack hydrolysable functional groups. Aliphatic acids are hydrolytically stable in aqueous solution.

Water solubility:

In general, the water solubility of single carbon chain length substances followed a pattern of decreasing solubility as carbon chain length increases, especially at C16 and higher.

In addition, greater solubility is seen for dicarboxylic acids as compared to their homologous single acids:

In reviewing the physical/ chemical properties of the aliphatic acids, two predominant trends are clearly evident with increasing alkyl chain length and include: i) increasing melting point, boiling point, and partition coefficient, and ii) decreasing water solubility and vapour pressure. Within a given carbon chain length, melting point increases with increasing saturation and decreases with increasing unsaturation. The noted general trends with increasing alkyl chain length are observed when an entire single component group (12 saturated, 4 mono-unsaturated, 2 di-unsaturated, and 1 tri-unsaturated substances) is evaluated together; that is the degree of saturation or unsaturation does not alter the properties trend. The effect of mono-unsaturation (C14:1 to C22:1) appears to be a slight increase in water solubility and a slight decrease in the partition coefficient, as compared to the corresponding saturated substances; a similar trend is noted for the C18 di- or tri-unsaturated substances.

Fatty acids (including methyl esters) were stable to hydrolysis in the pH range of 1-14. It is not expected that photolysis would significantly contribute to the degradation of fatty acids in water.

According to modelling, the aliphatic acids are subject to photodegradation in air. Estimated half-lives generally increase with decreasing chain length and range from 0.6 hours to 17.5 hours.

Methyl (and other) esters are estimated to exhibit high mobility and the acids very high mobility. Mobility may be expected to be higher for the salts than for the corresponding acids and methyl esters

Biodegradation studies or model estimations for single and multi-component aliphatic acids generally confirm that the extent of biodegradation observed in 28 days meets the ready biodegradability criterion (>60%). When the 10-day window was not met or less than 60%, biodegradation was observed in 28 days, it is likely that the aliphatic acids tested were not fully in solution.

Biodegradability tests demonstrated that pelargonic acid (C9), potassium salts and methyl octanoate / methyl decanoate are readily biodegradable. It can be assumed that both acids and methyl esters fatty acids C7-C18 are readily biodegradable.

No experimental bioaccumulation data appear to be available but log Kow data from various sources are higher than 4, which indicates that fatty acids and natural lipids have a potential for bioaccumulating in aquatic organisms.

Fatty alcohols up to chain length C18 are biodegradable, with length up to C16 biodegrading within 10 days completely. Chains C16 to C18 were found to biodegrade from 62% to 76% in 10 days. Chains greater than C18 were found to degrade by 37% in 10 days. Field studies at waste-water treatment plants have shown that 99% of fatty alcohols lengths C12-C18 are removed.

A review of soaps (including calcium and magnesium salts) states that the available data indicate all fatty acid salt chain lengths up to and including C18 can be metabolised under aerobic conditions and can be considered to be biodegradable. Biodegradability did not appear to be influenced by even or odd chain length, degree of saturation or unsaturation or branching. For example odd/even chain length C8 and C9 are readily biodegradable; Saturation/unsaturation: C18(saturated) and C18 (di-unsaturated) are biodegradable, while C18 (mono-unsaturated) are readily biodegradable; branching or hydroxylation: the C18 hydroxylated substance was readily biodegradable and the C18 methyl branched substance was biodegradable.

Higher water solubility of the potassium, sodium and ammonium salts make these a lower ranked analogy for the aquatic toxicity endpoints for the (non-salt) aliphatic acids (and vice versa), while lower water solubility of the magnesium and calcium salts make these a lower ranked analogy for all other members of the category

The aliphatic acids also undergo biodegradation under anaerobic conditions.

Estimated bioconcentration factor values are calculated using EPI Suite v4.10. The aliphatic acids have BCF

values less than 100, indicating a low potential for bioaccumulation

Fate prediction using fugacity modeling has shown that fatty alcohols with chain lengths of C10 and greater in water partition into sediment. Lengths C14 and above are predicted to stay in the air upon release. Modeling shows that each type of fatty alcohol will respond independently upon environmental release

Ecotoxicity

Structure-activity relationships based on carbon chain length are evident in the available data on the aquatic ecotoxicity of substances of this category (aquatic toxicity increases with increasing chain length up to a "cutoff" at or near 12 carbons).

The aliphatic acids category members possess properties indicating a hazard for the environment (acute toxicity to fish: between 1-100 mg/L for carbon chain lengths C6 through C12, and multi-component sodium or potassium salts C16-18; acute toxicity to aquatic invertebrates: between 1 and 100 mg/L for carbon chain lengths C6 through C9 (including sodium salts) and less than 1 mg/L for sodium salts single component aliphatic acids C18 and multi component sodium salt aliphatic acids with carbon chain lengths including C14 through C18; and, acute toxicity to aquatic plants: between 1-100 mg/L for carbon chain length C12, including sodium or ammonium salts).

There are a number of acute data for fatty acids and fatty acid salts to aquatic organisms although there is a predominance of data for fatty acid. There are few toxicity values for terrestrial organisms. Data availability / quality covering all the taxonomic groups for specific fatty acid salt chain lengths is poor. The chronic data set is very limited.

For chain lengths >C12, solubility decreases to a degree where an adverse effect would not be expected in the environment due to reduced bioavailability. Data for longer chain lengths have been generated using solvents which makes interpretation more difficult.

The most of few available data indicate low toxicity towards aquatic organisms with EC/LC50 values above 1000 mg/l. However, EC/LC50 values below 100 mg/l are not unusual



## GEA FOAM MARK PINK AEROSOL

either

Fish, invertebrates and algae experience similar levels of toxicity with fatty alcohols although it is dependent on chain length with the shorter chain having greater toxicity potential. Longer chain lengths show no toxicity to aquatic organisms.

The available toxicity data indicated low acute and short-term (for birds only) toxicity to birds and mammals. Given that fatty acids are an essential component of the diet of birds and mammals a low risk is expected. On the basis that fatty acids are readily biodegradable and are an essential component of the diet of birds and mammals, a low reproductive risk is expected.

No toxicity data were available for higher aquatic plants and therefore a risk assessment cannot be performed. As pelargonic acid, fatty acid/salt and C8-C10 methyl esters are used as herbicides and plant growth regulators, a data gap to address the risk to higher aquatic plants was identified

A low risk to natural populations of bees and non-target arthropods was concluded for representative greenhouses uses of potassium salts of fatty acids, fatty acid/salt and C8-C10 methyl esters.

Given that fatty acids are readily biodegradable a low risk to sewage treatment organisms was concluded for all of the representative uses.

For petroleum distillates:

Environmental fate:

When petroleum substances are released into the environment, four major fate processes will take place: dissolution in water, volatilization, biodegradation and adsorption. These processes will cause changes in the composition of these UVCB substances. In the case of spills on land or water surfaces, photodegradation-another fate process-can also be significant.

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

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

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

Biodegradation:

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

- (1) n-alkanes, especially in the C10–C25 range, which are degraded readily;
- (2) isoalkanes;
- (3) alkenes;
- (4) benzene, toluene, ethylbenzene, xylenes (BTEX) (when present in concentrations that are not toxic to microorganisms);
- (5) monoaromatics;
- (6) polynuclear (polycyclic) aromatic hydrocarbons (PAHs); and
- (7) higher molecular weight cycloalkanes (which may degrade very slowly).

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

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

Bioaccumulation:

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

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

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

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

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

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

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

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

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

Ecotoxicity:

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

The tropical mysid *Metamysidopsis insularis* was shown to be very sensitive to diesel fuel, with a 96-hour LC50 value of 0.22 mg/L this species has been shown to be as sensitive as temperate mysids to toxicants. However, this study used nominal concentrations, and therefore was not considered acceptable. In another study involving diesel fuel, the effect on brown or common shrimp (*Crangon crangon*) a 96-hour LC50 of 22 mg/L was determined. A "gas oil" was also tested and a 96-hour LC50 of 12 mg/L was determined. The steady state cell density of marine phytoplankton decreased with increasing concentrations of diesel fuel, with different sensitivities between species. The diatom *Phaeodactylum tricornutum* showed a 20% decrease in cell density in 24 hours following a 3 mg/L exposure with a 24-hour no-observed effect concentration (NOEC) of 2.5 mg/L. The microalga *Isochrysis galbana* was more tolerant to diesel fuel, with a 24-hour lowest-observed-effect concentration (LOEC) of 26 mg/L (14% decrease in cell density), and a NOEC of 25 mg/L.

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

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

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

For butane:

log Kow: 2.89

Koc: 450-900

BCF: 1.9

**Environmental Fate**

**Terrestrial Fate:** An estimated Koc value of 900, determined from a log Kow of 2.89 indicates that n-butane is expected to have low mobility in soil. Volatilisation of n-butane from moist soil surfaces is expected to be an important fate process given an estimated Henry's Law constant of 0.95 atm-cu m/mole, derived from its vapor pressure, 1820 mm Hg and water solubility, 61.2 mg/l. The potential for volatilisation of n-butane from dry soil surfaces may exist based upon its vapor pressure. While volatilisation from soil surfaces is expected to

Continued...

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be the predominant fate process of n-butane released to soil, this compound is also susceptible to biodegradation. In one soil, a biodegradation rate of 1.8 mgC/day/kg dry soil was reported.

**Aquatic fate:** The estimated Koc value indicates that n-butane may adsorb to suspended solids and sediment. Volatilisation from water surfaces is expected based upon an estimated Henry's Law constant. Using this Henry's Law constant volatilisation half-lives for a model river and model lake are estimated to be 2.2 hours and 3 days, respectively. An estimated BCF of 33 derived from the log Kow suggests the potential for bioconcentration in aquatic organisms is moderate. While volatilisation from water surfaces is expected to be the major fate process for n-butane released to water, biodegradation of this compound is also expected to occur. In a screening study, complete biodegradation was reported in 34 days. In a second study using a defined microbial culture, it was reported that n-butane was degraded to 2-butanone and 2-butanol. Photolysis or hydrolysis of n-butane in aquatic systems is not expected to be important.

**Atmospheric fate:** According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere and the vapour pressure, n-butane, is expected to exist solely as a gas in the ambient atmosphere. Gas-phase n-butane is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 6.3 days, calculated from its rate constant of  $2.54 \times 10^{-12}$  cu cm/molecule-sec at 25 deg. Based on data for iso-octane and n-hexane, n-butane is not expected to absorb UV light in the environmentally significant range, >290 nm and probably will not undergo direct photolysis in the atmosphere. Experimental data showed that 7.7% of the n-butane fraction in a dark chamber reacted with nitrogen oxide to form the corresponding alkyl nitrate, suggesting nighttime reactions with radical species and nitrogen oxides may contribute to the atmospheric transformation of n-butane.

for triethanolamine:

Koc : 3

Half-life (hr) air : 4

Henry's atm m<sup>3</sup>/mol: 3.38E-19

BOD 5 if unstated: nil-0.17

COD : 1.5

ThOD : 2.04; 1.61 p/p

ThOD (measured) 1.52 mg/mg (Union Carbide)

ThOD (calculated) 1.61 mg/mg (Union Carbide)

BCF : <1

Biodegradability: 96% DOC reduction (OECD Method 301E)

BOD; Day 5: 8%, Day 10: 9%, Day 20: 66%

Passes Sturm, AFNOR tests for biodegradability.

Reaches more than 70% mineralisation in OECD test for inherent biodegradability (Zahn-Wellens test)

Theoretical oxygen demand ThOD) is calculated at 1.61 p/p. Degradation is expected in the atmospheric environment within minutes to hours.

Log octanol/ water partition coefficient (log Kow) is estimated using the Pomona-Medchem structural fragment to be -1.746.

Potential for the mobility in soil is very high (Koc between 0 and 50).

Henry's Law Constant (H) is estimated to be 3.38E-19 atm.m<sup>3</sup>/mol (25 C)

Log soil organic carbon partition coefficient (log Koc) is estimated to be 0.48.

When released into soil the material is expected to degrade without significant evaporation. A half-life of between 1 to 10 days is expected.

Material has shown a potential to biodegrade. Attains >99% degradation in activated sludge in 24 hours. Attains >99% degradation in soil is 1-14 days.

Bioconcentration potential is low (BCF less than 100 or log Kow less than 3).

When released into water, the material is expected to degrade with a half-life of about 1 to 10 days. Because the material has a log octanol-water coefficient of less than 3 it is not expected to bioaccumulate.

Release to air is expected to produce photolytic degradation resulting in hydroxyl radicals. The material is expected to be removed from the atmosphere by dry and wet deposition (half-life between 1 and 10 days).

### Environmental fate:

**Transport:** Due to the high water solubility and low vapour pressure of triethanolamine, it is likely to partition preferentially into the water phase from which volatilisation to the atmosphere is likely to be only a minor removal process. The low log Kow value indicates that bioaccumulation and adsorption onto soils/sediments is unlikely to occur.

**Water:** If released to water, triethanolamine should biodegrade. The half-life of this compound is expected to range from a few days to a few weeks depending on the degree of acclimation of the system. Bioconcentration in aquatic organisms, adsorption to suspended solids and sediments, and volatilization are not expected to be important fate processes in water. Triethanolamine does not decompose or hydrolyze in contact with water and there is no abiotic degradation.

**Biodegradation:** Triethanolamine is readily biodegradable, possibly after a short acclimation period. The data indicate that triethanolamine is inherently biodegradable. Extensive removal due to biodegradation is to be expected in sewage treatment plants. In the ready biodegradation tests, triethanolamine was readily biodegradable in the AFNOR (97% degradation based on DOC removal), STURM (91% degradation based on CO<sub>2</sub> evolution) and OECD Screening test (96% degradation based on DOC removal, but little degradation was observed in the MITI (14 day test; 2% removal based on BOD and Closed Bottle (0-9% removal based on BOD).

### Ecotoxicity:

Material is practically non-toxic to aquatic organisms on an acute basis (LC<sub>50</sub> >100 mg/l in most sensitive species)

Fish LC<sub>50</sub> (96 h): fathead minnow (Pimephales promelas) 1800-11800 mg/l; fathead minnow 5600 mg/l (Union Carbide); bluegill (Leuciscus idus) 7930 mg/l; goldfish (Carassius auratus) 5000 mg/l

Daphnia magna LC<sub>50</sub> (24 h): 1390 - 2038 mg/l

Daphnia magna LC<sub>50</sub> (48 h): 947 mg/l (Union Carbide)

Algae LC<sub>50</sub> (48 h): 750 mg/l

Brine shrimp LC<sub>50</sub>: (Artemia salina) 5600 mg/l

Maximum acceptable toxicant concentration (MATC): 22 mg/l

Algal growth inhibition (Scenedesmus subspicatus) EC<sub>50</sub>: 470-750 mg/l

Inhibition of bacteria in effluent: 50% inhibition: >10000 mg/l

Inhibitory concentration (IC<sub>50</sub>) is OECD "Activated Sludge, Respiration Inhibition Test" (Guideline 209) is >1000 mg/l.

**DO NOT discharge into sewer or waterways.**

### Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
triethanolamine	LOW	LOW
diethanolamine	LOW (Half-life = 14 days)	LOW (Half-life = 0.3 days)
butane	LOW	LOW
propane	LOW	LOW

### Bioaccumulative potential

Ingredient	Bioaccumulation
triethanolamine	LOW (BCF = 3.9)
diethanolamine	LOW (BCF = 1)
butane	LOW (LogKOW = 2.89)
propane	LOW (LogKOW = 2.36)

### Mobility in soil

Ingredient	Mobility
triethanolamine	LOW (KOC = 10)

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Ingredient	Mobility
diethanolamine	HIGH (KOC = 1)
butane	LOW (KOC = 43.79)
propane	LOW (KOC = 23.74)

## SECTION 13 Disposal considerations

## Waste treatment methods

<b>Product / Packaging disposal</b>	<ul style="list-style-type: none"> <li>· Recycle wherever possible or consult manufacturer for recycling options.</li> <li>· Consult State Land Waste Management Authority for disposal.</li> <li>▶ Consult State Land Waste Management Authority for disposal.</li> <li>▶ Discharge contents of damaged aerosol cans at an approved site.</li> <li>▶ Allow small quantities to evaporate.</li> <li>▶ <b>DO NOT incinerate or puncture aerosol cans.</b></li> <li>▶ Bury residues and emptied aerosol cans at an approved site.</li> </ul>
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Ensure that the hazardous substance is disposed in accordance with the Hazardous Substances (Disposal) Notice 2017

## Disposal Requirements


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

The hazardous substance must only be disposed if it has been treated by a method that changed the characteristics or composition of the substance and it is no longer hazardous. Only dispose to the environment if a tolerable exposure limit has been set for the substance.

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

## SECTION 14 Transport information

## Labels Required

	
<b>Marine Pollutant</b>	NO
<b>HAZCHEM</b>	Not Applicable

## Land transport (UN)

<b>UN number</b>	1950	
<b>UN proper shipping name</b>	AEROSOLS	
<b>Transport hazard class(es)</b>	Class	2.1
	Subrisk	Not Applicable
<b>Packing group</b>	Not Applicable	
<b>Environmental hazard</b>	Not Applicable	
<b>Special precautions for user</b>	Special provisions	63; 190; 277; 327; 344; 381
	Limited quantity	1000ml

## Air transport (ICAO-IATA / DGR)

<b>UN number</b>	1950	
<b>UN proper shipping name</b>	Aerosols, flammable	
<b>Transport hazard class(es)</b>	ICAO/IATA Class	2.1
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	10L
<b>Packing group</b>	Not Applicable	
<b>Environmental hazard</b>	Not Applicable	
<b>Special precautions for user</b>	Special provisions	A145 A167 A802
	Cargo Only Packing Instructions	203
	Cargo Only Maximum Qty / Pack	150 kg
	Passenger and Cargo Packing Instructions	203
	Passenger and Cargo Maximum Qty / Pack	75 kg
	Passenger and Cargo Limited Quantity Packing Instructions	Y203
	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G

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## Sea transport (IMDG-Code / GGVSee)

<b>UN number</b>	1950	
<b>UN proper shipping name</b>	AEROSOLS	
<b>Transport hazard class(es)</b>	IMDG Class	2.1
	IMDG Subrisk	Not Applicable
<b>Packing group</b>	Not Applicable	
<b>Environmental hazard</b>	Not Applicable	
<b>Special precautions for user</b>	EMS Number	F-D , S-U
	Special provisions	63 190 277 327 344 381 959
	Limited Quantities	1000 ml

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

Not Applicable

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

Product name	Group
triethanolamine	Not Available
diethanolamine	Not Available
fatty acids, C16-18 and C18-unsaturated	Not Available
sodium borate, pentahydrate	Not Available
butane	Not Available
propane	Not Available

## Transport in bulk in accordance with the ICG Code

Product name	Ship Type
triethanolamine	Not Available
diethanolamine	Not Available
fatty acids, C16-18 and C18-unsaturated	Not Available
sodium borate, pentahydrate	Not Available
butane	Not Available
propane	Not Available

## SECTION 15 Regulatory information

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

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

HSR Number	Group Standard
HSR002515	Aerosols (Flammable) Group Standard 2017

## triethanolamine is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

New Zealand Approved Hazardous Substances with controls

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

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

New Zealand Inventory of Chemicals (NZIoC)

New Zealand Workplace Exposure Standards (WES)

## diethanolamine is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

New Zealand Approved Hazardous Substances with controls

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

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

New Zealand Inventory of Chemicals (NZIoC)

New Zealand Workplace Exposure Standards (WES)

## fatty acids, C16-18 and C18-unsaturated is found on the following regulatory lists

New Zealand Approved Hazardous Substances with controls

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

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

New Zealand Inventory of Chemicals (NZIoC)

## sodium borate, pentahydrate is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List

New Zealand Approved Hazardous Substances with controls

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

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

New Zealand Inventory of Chemicals (NZIoC)

New Zealand Workplace Exposure Standards (WES)

## butane is found on the following regulatory lists

Continued...

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Chemical Footprint Project - Chemicals of High Concern List  
 New Zealand Approved Hazardous Substances with controls  
 New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

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

**propane is found on the following regulatory lists**

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

New Zealand Inventory of Chemicals (NZIoC)  
 New Zealand Workplace Exposure Standards (WES)

**Hazardous Substance Location**

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

Hazard Class	Quantity (Closed Containers)	Quantity (Open Containers)
2.1.2A	3 000 L (aggregate water capacity)	3 000 L (aggregate water capacity)

**Certified Handler**

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

Class of substance	Quantities
Not Applicable	Not Applicable

Refer Group Standards for further information

**Maximum quantities of certain hazardous substances permitted on passenger service vehicles**

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

Hazard Class	Gas (aggregate water capacity in mL)	Liquid (L)	Solid (kg)	Maximum quantity per package for each classification
2.1.2A				1L (aggregate water capacity)

**Tracking Requirements**

Not Applicable

**National Inventory Status**

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDLS	No (triethanolamine; diethanolamine; fatty acids, C16-18 and C18-unsaturated; sodium borate, pentahydrate; butane; propane)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (fatty acids, C16-18 and C18-unsaturated)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (fatty acids, C16-18 and C18-unsaturated)
Vietnam - NCI	Yes
Russia - FBEPH	No (fatty acids, C16-18 and C18-unsaturated)
<b>Legend:</b>	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing (see specific ingredients in brackets)

**SECTION 16 Other information**

<b>Revision Date</b>	21/04/2021
<b>Initial Date</b>	27/05/2014

**SDS Version Summary**

Version	Date of Update	Sections Updated
9.34.1.1	21/04/2021	Acute Health (inhaled), Acute Health (skin), Acute Health (swallowed), Chronic Health, Classification, Environmental, Exposure Standard, Ingredients

**Other information**

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.

**GEA FOAM MARK PINK AEROSOL**

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

**Definitions and abbreviations**

PC—TWA: Permissible Concentration-Time Weighted Average  
PC—STEL: Permissible Concentration-Short Term Exposure Limit  
IARC: International Agency for Research on Cancer  
ACGIH: American Conference of Governmental Industrial Hygienists  
STEL: Short Term Exposure Limit  
TEEL: Temporary Emergency Exposure Limit.  
IDLH: Immediately Dangerous to Life or Health Concentrations  
ES: Exposure Standard  
OSF: Odour Safety Factor  
NOAEL :No Observed Adverse Effect Level  
LOAEL: Lowest Observed Adverse Effect Level  
TLV: Threshold Limit Value  
LOD: Limit Of Detection  
OTV: Odour Threshold Value  
BCF: BioConcentration Factors  
BEI: Biological Exposure Index  
AII: Australian Inventory of Industrial Chemicals  
DSL: Domestic Substances List  
NDSL: Non-Domestic Substances List  
IECSC: Inventory of Existing Chemical Substance in China  
EINECS: European INventory of Existing Commercial chemical Substances  
ELINCS: European List of Notified Chemical Substances  
NLP: No-Longer Polymers  
ENCS: Existing and New Chemical Substances Inventory  
KECI: Korea Existing Chemicals Inventory  
NZIoC: New Zealand Inventory of Chemicals  
PICCS: Philippine Inventory of Chemicals and Chemical Substances  
TSCA: Toxic Substances Control Act  
TCSI: Taiwan Chemical Substance Inventory  
INSQ: Inventario Nacional de Sustancias Químicas  
NCI: National Chemical Inventory  
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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