

Ethanol Shield

Techtronic Industries Australia Pty Ltd

Chemwatch Hazard Alert Code: **3**

Chemwatch: 5359-80

Issue Date: **30/07/2019**

Version No: 5.1.1.1

Print Date: **01/08/2019**

Safety Data Sheet according to WHS and ADG requirements

L.GHS.AUS.EN

SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	Ethanol Shield
Synonyms	Not Available
Proper shipping name	TOXIC LIQUID, ORGANIC, N.O.S. (contains ethylene glycol monobutyl ether)
Other means of identification	Not Available

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

Relevant identified uses	Fuel system treatment. SDS are intended for use in the workplace. For domestic-use products, refer to consumer labels.
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Details of the supplier of the safety data sheet

Registered company name	Techtronic Industries Australia Pty Ltd
Address	31 Gilby Road Mount Waverley VIC 3149 Australia
Telephone	1300 361 505
Fax	Not Available
Website	http://www.ttigroup.com/
Email	customerservice@ttibrands.com.au

Emergency telephone number

Association / Organisation	Poison Information Centre (Australia)
Emergency telephone numbers	13 11 26 (24 hours a day, seven days a week)
Other emergency telephone numbers	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

COMBUSTIBLE LIQUID, regulated for storage purposes only

Poisons Schedule	S6
Classification ^[1]	Flammable Liquid Category 4, Acute Toxicity (Oral) Category 4, Acute Toxicity (Dermal) Category 4, Acute Toxicity (Inhalation) Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Germ cell mutagenicity Category 2, Carcinogenicity Category 1B, Chronic Aquatic Hazard Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
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Ethanol Shield

SIGNAL WORD **DANGER**

Hazard statement(s)

H227	Combustible liquid.
H302	Harmful if swallowed.
H312	Harmful in contact with skin.
H332	Harmful if inhaled.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H341	Suspected of causing genetic defects.
H350	May cause cancer.
H411	Toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
P271	Use only outdoors or in a well-ventilated area.
P281	Use personal protective equipment as required.
P261	Avoid breathing mist/vapours/spray.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/attention.
P321	Specific treatment (see advice on this label).
P322	Specific measures (see advice on this label).
P362	Take off contaminated clothing and wash before reuse.
P370+P378	In case of fire: Use alcohol resistant foam or fine spray/water fog for extinction.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P330	Rinse mouth.
P332+P313	If skin irritation occurs: Get medical advice/attention.

Precautionary statement(s) Storage

P403+P235	Store in a well-ventilated place. Keep cool.
P405	Store locked up.

Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.
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SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
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Ethanol Shield

111-76-2	>60	<u>ethylene glycol monobutyl ether</u>
73398-61-5	<10	<u>caprylic/ capric triglyceride</u>
128-37-0	<10	<u>2,6-di-tert-butyl-4-methylphenol</u>
95-14-7	<=1	<u>1H-benzotriazole</u>
108-01-0	<=1	<u>dimethylethanolamine</u>
95-63-6	<=1	<u>1,2,4-trimethyl benzene</u>
108-67-8	<=1	<u>1,3,5-trimethyl benzene</u>
1330-20-7	<=1	<u>xylene</u>
98-82-8	<=1	<u>cumene</u>
526-73-8	<=1	<u>1,2,3-trimethyl benzene</u>

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> Wash out immediately with fresh running water. Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. Seek medical attention without delay; if pain persists or recurs seek medical attention. Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> Immediately remove all contaminated clothing, including footwear. Flush skin and hair with running water (and soap if available). Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> If fumes or combustion products are inhaled remove from contaminated area. Lay patient down. Keep warm and rested. Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. Transport to hospital, or doctor.
Ingestion	<ul style="list-style-type: none"> If swallowed do NOT induce vomiting. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. Observe the patient carefully. Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.

Followed acute or short term repeated exposures to ethylene glycol monoalkyl ethers and their acetates:

- Hepatic metabolism produces ethylene glycol as a metabolite.
- Clinical presentation, following severe intoxication, resembles that of ethylene glycol exposures.
- Monitoring the urinary excretion of the alkoxyacetic acid metabolites may be a useful indication of exposure.

[Ellenhorn and Barceloux: Medical Toxicology]

For acute or short term repeated exposures to ethylene glycol:

- Early treatment of ingestion is important. Ensure emesis is satisfactory.
- Test and correct for metabolic acidosis and hypocalcaemia.
- Apply sustained diuresis when possible with hypertonic mannitol.
- Evaluate renal status and begin haemodialysis if indicated. [I.L.O.]
- Rapid absorption is an indication that emesis or lavage is effective only in the first few hours. Cathartics and charcoal are generally not effective.
- Correct acidosis, fluid/electrolyte balance and respiratory depression in the usual manner. Systemic acidosis (below 7.2) can be treated with intravenous sodium bicarbonate solution.
- Ethanol therapy prolongs the half-life of ethylene glycol and reduces the formation of toxic metabolites.
- Pyridoxine and thiamine are cofactors for ethylene glycol metabolism and should be given (50 to 100 mg respectively) intramuscularly, four times per day for 2 days.
- Magnesium is also a cofactor and should be replenished. The status of 4-methylpyrazole, in the treatment regime, is still uncertain. For clearance of the material and its metabolites, haemodialysis is much superior to peritoneal dialysis.

[Ellenhorn and Barceloux: Medical Toxicology]

It has been suggested that there is a need for establishing a new biological exposure limit before a workshift that is clearly below 100 mmol ethoxy-acetic acids per mole creatinine in morning urine of people occupationally exposed to ethylene glycol ethers. This arises from the finding that an increase in urinary stones may be associated with such exposures.

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Laitinen J., et al: Occupational & Environmental Medicine 1996; 53, 595-600

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

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

Special hazards arising from the substrate or mixture

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

Fire Fighting	<ul style="list-style-type: none"> ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ Avoid spraying water onto liquid pools. ▸ DO NOT approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▸ Combustible. ▸ Slight fire hazard when exposed to heat or flame. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke. ▸ Mists containing combustible materials may be explosive. <p>Combustion products include: carbon dioxide (CO₂) nitrogen oxides (NO_x) other pyrolysis products typical of burning organic material.</p>
HAZCHEM	2X

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none"> ▸ Remove all ignition sources. ▸ Clean up all spills immediately. ▸ Avoid breathing vapours and contact with skin and eyes. ▸ Control personal contact with the substance, by using protective equipment. ▸ Contain and absorb spill with sand, earth, inert material or vermiculite. ▸ Wipe up. ▸ Place in a suitable, labelled container for waste disposal.
Major Spills	<ul style="list-style-type: none"> ▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Stop leak if safe to do so. ▸ Contain spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Neutralise/decontaminate residue (see Section 13 for specific agent). ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▸ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ Avoid smoking, naked lights or ignition sources. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	<ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ No smoking, naked lights or ignition sources. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ DO NOT use aluminium or galvanised containers ▶ Lined metal can, lined metal pail/ can. ▶ Plastic pail. ▶ Polyliner drum. ▶ Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Avoid reaction with oxidising agents

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	ethylene glycol monobutyl ether	2-Butoxyethanol	20 ppm / 96.9 mg/m3	242 mg/m3 / 50 ppm	Not Available	Not Available
Australia Exposure Standards	2,6-di-tert-butyl-4-methylphenol	2,6-Di-tert-butyl-p-cresol	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	dimethylethanolamine	Dimethylaminoethanol	2 ppm / 7.4 mg/m3	22 mg/m3 / 6 ppm	Not Available	Not Available
Australia Exposure Standards	xylene	Xylene (o-, m-, p-isomers)	80 ppm / 350 mg/m3	655 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	cumene	Cumene	25 ppm / 125 mg/m3	375 mg/m3 / 75 ppm	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
ethylene glycol monobutyl ether	Butoxyethanol, 2-; (Glycol ether EB)	60 ppm	120 ppm	700 ppm
2,6-di-tert-butyl-4-methylphenol	Bis(1,1-dimethylethyl)-4-methylphenol, 2,6-; (BHT (food grade); 2,6-Di-tert-butyl-p-cresol)	6 mg/m3	29 mg/m3	180 mg/m3
1H-benzotriazole	Benzotriazole	1.2 mg/m3	13 mg/m3	77 mg/m3

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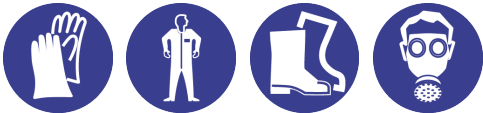
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dimethylethanolamine	Dimethylaminoethanol, 2-	3.7 ppm	40 ppm	72 ppm
1,2,4-trimethyl benzene	Permafluor E+	140 mg/m3	360 mg/m3	2,200 mg/m3
1,2,4-trimethyl benzene	Trimethylbenzene, 1,2,4-; (Pseudocumene)	Not Available	Not Available	480 ppm
1,3,5-trimethyl benzene	Mesitylene; (1,3,5-Trimethylbenzene)	Not Available	Not Available	480 ppm
xylene	Xylenes	Not Available	Not Available	Not Available
cumene	Cumene; (Isopropyl benzene)	Not Available	Not Available	Not Available
1,2,3-trimethyl benzene	Trimethylbenzene, 1,2,3-	Not Available	Not Available	480 ppm

Ingredient	Original IDLH	Revised IDLH
ethylene glycol monobutyl ether	700 ppm	Not Available
caprylic/ capric triglyceride	Not Available	Not Available
2,6-di-tert-butyl-4-methylphenol	Not Available	Not Available
1H-benzotriazole	Not Available	Not Available
dimethylethanolamine	Not Available	Not Available
1,2,4-trimethyl benzene	Not Available	Not Available
1,3,5-trimethyl benzene	Not Available	Not Available
xylene	900 ppm	Not Available
cumene	900 ppm	Not Available
1,2,3-trimethyl benzene	Not Available	Not Available

MATERIAL DATA

Exposure controls

Appropriate engineering controls	Use in a well-ventilated area General exhaust is adequate under normal operating conditions.
Personal protection	
Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields; or as required, ▶ Chemical goggles. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ Eyewash unit. ▶ Barrier cream. ▶ Skin cleansing cream.

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

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone,

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computer-generated selection:

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Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
SARANEX-23	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.

approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	AK-AUS P2	-	AK-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AK-AUS / Class 1 P2	-
up to 100 x ES	-	AK-2 P2	AK-PAPR-2 P2 ^

^ - Full-face

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO₂), G = Agricultural chemicals, K = Ammonia(NH₃), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

- ▶ Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- ▶ The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- ▶ Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Liquid with characteristic odour; partly mixes with water. Colour varies.		
Physical state	Liquid	Relative density (Water = 1)	0.75
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Applicable	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	2.03 @ 40C
Initial boiling point and boiling range (°C)	135-210	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	62 (CC)	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Combustible.	Oxidising properties	Not Available
Upper Explosive Limit (%)	10	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	1.0	Volatile Component (%vol)	>90
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Partly miscible	pH as a solution (1%)	Not Available

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Vapour density (Air = 1) >1

VOC g/L Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	<p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation hazard is increased at higher temperatures.</p> <p>Ethylene glycol monobutyl ether (2-butoxyethanol) and its metabolite butoxyacetic acid are haemolytic agents, causing red blood cell destruction.</p> <p>On the basis of industrial experience and volunteer short-term exposure humans are shown to be less susceptible than experimental animals to exposure. In 8-hour exposures at concentrations of 200 or 100 ppm no objective effects were seen other than raised urinary excretion of the metabolite butoxyacetic acid. No increased osmotic fragility of the red blood cell is observed. Subjectively these concentrations were uncomfortable with mild eye, nose and throat irritation occurring. No clinical signs of adverse effects nor subjective complaints were produced when male volunteers were exposed for 2 hours to 20 ppm during light physical exercise. Other studies have established that the most sensitive indicators of toxic effect observed from many of the glycol ethers is an increase in erythrocyte osmotic fragility in rats. This appears to be related to the development of haemoglobinuria at higher exposure levels.</p>
Ingestion	<p>Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.</p> <p>Severe acute exposure to ethylene glycol monobutyl ether, by ingestion, may cause kidney damage, haemoglobinuria, (blood in urine) and is potentially fatal.</p> <p>Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.</p> <p>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).</p>
Skin Contact	<p>Skin contact with the material may be harmful; systemic effects may result following absorption.</p> <p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Open cuts, abraded or irritated skin should not be exposed to this material</p>
Eye	<p>Evidence exists, or practical experience predicts, that the material may cause severe 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. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>
Chronic	<p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>Studies with some ethylene glycol ethers and their esters indicate reproductive changes, testicular atrophy, infertility and kidney function changes. The metabolic acetic acid derivatives of the glycol ethers (alkoxyacetic acids), not the ether itself, have been found to be the proximal reproductive toxin in animals. The potency of these metabolites decrease significantly as the chain length of the ether increases. Consequently glycol ethers with longer substituents (e.g diethylene glycols, triethylene glycols) have not generally been associated with reproductive effects. One of the most sensitive indicators of toxic effects observed from many of the glycol ethers is an increase in the erythrocytic osmotic fragility in rats. This appears to be related to the development of haemoglobinuria (blood in the urine) at higher exposure levels or as a result of chronic exposure. Ethylene glycol ethers and acetates are mainly metabolised to alkoxyacetic acids but there is also a minor pathway through ethylene glycol to oxalic acid. The main pathway of ethylene glycol ethers</p>

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is associated with significant clinical or experimental health effects, but the minor pathway is also interesting because formation of urinary stones depends principally upon urinary concentration of oxalate and calcium. In one study (1) the tendency to form urinary stones was 2.4 times higher amongst silk-screen printers exposed to ethylene glycol ethers, than among office workers. (1) Laitinen J., et al: Occupational Environmental Medicine 1996, 53 595-600

Ethanol Shield	TOXICITY	IRRITATION
	Not Available	Not Available
ethylene glycol monobutyl ether	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 100 mg SEVERE
	Inhalation (rat) LC50: 449.48655 mg/l/4h ^[2]	Eye (rabbit): 100 mg/24h-moderate
	Oral (rat) LD50: 250 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit): 500 mg, open; mild
		Skin: adverse effect observed (irritating) ^[1]
caprylic/ capric triglyceride	TOXICITY	IRRITATION
	Oral (rat) LD50: >2000 mg/kg ^[2]	Eye (rabbit): 100 mg/24 h - mild
2,6-di-tert-butyl-4-methylphenol	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 100 mg/24h-moderate
	Oral (rat) LD50: 890 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin (human): 500 mg/48h - mild
		Skin (rabbit): 500 mg/48h-moderate
		Skin: no adverse effect observed (not irritating) ^[1]
1H-benzotriazole	TOXICITY	IRRITATION
	dermal (rat) LD50: >1000 mg/kg ^[2]	Eye (rabbit): moderate *
	Inhalation (rat) LC50: 1.4 mg/l/4h*** ^[2]	Eye: adverse effect observed (irritating) ^[1]
	Oral (rat) LD50: 560 mg/kg ^[2]	Skin (rabbit): slight *
dimethylethanolamine	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 1219.3 mg/kg ^[2]	Eye (rabbit): 0.75 mg(open)-SEVERE
	Inhalation (rat) LC50: 1639.127619 mg/l/4hE ^[2]	Skin (rabbit): 445 mg(open)-mild
1,2,4-trimethyl benzene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >3160 mg/kg ^[2]	Not Available
	Inhalation (rat) LC50: 18 mg/l/4hd ^[2]	
1,3,5-trimethyl benzene	TOXICITY	IRRITATION
	Oral (rat) LD50: 5000 mg/kg ^[1]	Eye (rabbit): 500 mg/24h mild
		Eye: adverse effect observed (irritating) ^[1]
xylene	TOXICITY	IRRITATION
	Inhalation (rat) LC50: 24 mg/l/4hd ^[2]	Skin (rabbit): 20 mg/24h moderate
		Skin: adverse effect observed (irritating) ^[1]
	Oral (rat) LD50: 3523-8700 mg/kg ^[2]	Eye (human): 200 ppm irritant
		Eye (rabbit): 5 mg/24h SEVERE
xylene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1700 mg/kg ^[2]	Eye (rabbit): 87 mg mild
	Inhalation (rat) LC50: 4994.295 mg/l/4h ^[2]	Eye: adverse effect observed (irritating) ^[1]
xylene	TOXICITY	IRRITATION
	Oral (rat) LD50: 3523-8700 mg/kg ^[2]	Skin (rabbit): 500 mg/24h moderate

Continued...

Ethanol Shield

		Skin: adverse effect observed (irritating) ^[1]
cumene	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 2000 mg/kg ^[2]	Eye (rabbit): 500 mg/24h mild
	Inhalation (rat) LC50: 39 mg/l/4H ^[2]	Eye (rabbit): 86 mg mild
	Oral (rat) LD50: 1400 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin (rabbit): 10 mg/24h mild
		Skin (rabbit): 100 mg/24h moderate
		Skin: no adverse effect observed (not irritating) ^[1]
1,2,3-trimethyl benzene	TOXICITY	IRRITATION
	Not Available	Not Available
Legend:	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. * Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

ETHYLENE GLYCOL MONOBUTYL ETHER

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

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

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

Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m³) for EGHE, LC50 > 400ppm (2620 mg/m³) for EGBEA to LC50 > 2132 ppm (9061 mg/m³) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitizers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE *in vitro* than those of rats.

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

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

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

Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes). Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m³ and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m³), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m³), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m³) indicate that the members of the category are not teratogenic.

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

Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetotoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats.

Teratogenic effects were not observed in other species.

Ethanol Shield

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

Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility. Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions (1). The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of erythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration -dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma. 1: NTP Toxicology Program Technical report Series 484, March 2000.

For ethylene glycol:

Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol.

dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO₂, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO₂, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

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

Cardiovascular Effects. Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12- 24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol. Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases. Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.

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

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

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

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

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

Ethanol Shield

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

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

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

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

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

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

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

CAPRYLIC/ CAPRIC TRIGLYCERIDE

Not sensitising in guinea pig assay * IUCLID [Henkel]* Medium chain triglycerides (MCTs) exhibit very low levels of toxicity in a variety of laboratory animals and in humans when administered orally, parenterally or by the dermal route. There is no evidence that MCTs are sensitizers and they show little evidence that they are ocular or dermal irritants. The data strongly suggest that MCTs would pose little or no risk from toxicity when consumed as a supplement in a balanced diet at levels up to 15% of the dietary calories or about 50% of the dietary fat. MCTs are essentially non-toxic in the acute toxicity tests conducted in several species of animals. In ocular and dermal irritation testing, MCTs exhibited virtually no potential as ocular or dermal irritants, even with prolonged eye or skin exposure. MCTs exhibit no capacity for induction of hyper-sensitivity. 90-day toxicity tests did not result in notable toxicity, whether the product was administered in the diet up to 9375 mg/kg body weight/day in rats or by intramuscular injection (up to 0.5 ml/kg/day, rabbits). The toxicity NOAELs for two 3-month feeding studies in rats were, respectively, equal to or greater than 3125 mg/kg body weight/ day and equal to or greater than 9375 mg/kg body weight/day in the diet. There was no evidence that dietary administration of MCTs adversely affected the reproductive performance of rats or resulted in maternal toxicity, foetal toxicity or teratogenic effects at doses up to 4.28 g/kg body weight/day (iv). Another study, in rats, using a caprylic capric triglyceride, confirmed that MCTs would not pose a concern with regard to potential developmental or reproductive effects at dietary levels up to 12,500 mg/kg body weight/day. There was no evidence that dietary administration of MCTs adversely affected the reproductive performance of pigs or resulted in maternal toxicity, foetal toxicity or teratogenic effects at doses up to 9375 mg/kg body weight/day in the diet. In rabbits following iv administration, the maternal and foetal NOAELs were between 1.0 and 4.28 g/kg body weight/day, with toxicity being associated with nutritional deficit in the dams. A 2-yr study in rats, conducted with a closely related compound (tricaprylin, a triglyceride with C8 fatty acids), provided no evidence of a carcinogenic effect when the material was administered by oral gavage at levels up to 10 ml/kg (9.54 g/kg) per day. The toxicity NOAEL, based on data from this study, was 2.5 ml/kg/day (2.38 g/kg body weight/day). Although tricaprylin was found to be positive in one of the strains of *Salmonella typhimurium* in the presence of metabolic activation in an Ames microbial mutagenicity assay, the results of the carcinogenicity test with tricaprylin and mutagenicity tests with caprylic acid indicate that MCTs do not have the potential to be carcinogenic or mutagenic. The safety of human dietary consumption of MCTs, up to levels of 1 g/kg, has been confirmed in several clinical trials. MCTs have been used as 'Foods For Special Dietary Use' in a number of MCT-containing products used for total parenteral nutrition contain approximately 20% MCTs, and depending on patient size and needs, are given in quantities of 1000 to 3000 ml/day. Thus, under maximum exposure conditions, a patient would receive 200-600 ml MCTs per day for up to several months. This would translate to 3.0 to 9.0 g/kg body weight/day (assume 70 kg body weight). Proposed uses in food would include MCTs at over a range of 4 to 67% of the food (for example granola bars -4%, muffins 8.3%, cheese 12-23%, mayonnaise -35% or margarine - 67% based on product preparation needs While there is an increase in the alveolar acetone levels in diabetic patients fed MCTs, there is no evidence to suggest that consumption of moderate levels of MCTs would contribute to ketosis in these patients. Studies in rats support the evidence for the absence of the risk for ketosis. In patients with cirrhosis or other liver disease there is the potential for higher circulating levels of free fatty acids due to reduced hepatic metabolism. However, there is no evidence that the consumption of moderate levels of MCTs would contribute to CNS effects such as hepatic encephalopathy in these patients. In the cases of the diabetic or the cirrhotic patient, the consumption of MCTs could not account for such an elevation of ketone bodies or of free fatty acids as would be required to trigger adverse effects. Studies of MCTs are consistent with regard to the observations that MCTs can be administered by various routes at relatively high dose levels, especially in the diet or by oral gavage, without significant adverse effect. NOAEL values from dietary studies appear to be consistently of the order of 3000-5000 mg/kg body weight/ day and have been reported as high as 12000 mg/kg body weight/day. Similarly, humans receiving MCTs parenterally have tolerated doses of 3.0-9.0 g/kg body weight/day for periods of several months without adverse effects. A standard 2500 cal/day diet, in which 30% of the dietary calories is fat would include about 83.3 g fat per day. If 15% of the dietary calories, or 50% fat, were constituted of MCTs, the daily dietary intake of MCTs would be 41.7 g/day. For a 60-kg individual that would be about 0.7 g/kg body weight/day MCT. Compared to the lowest daily dose for TPN, about 200 ml or 3.2 g/kg body weight/day, the dietary intake would be 4.6-fold less than the intake used for TPN.

Ethanol Shield

For Group E aliphatic esters (polyol esters):

According to a classification scheme described by the American Chemistry Council's Aliphatic Esters Panel, Group E substances are esters of monoacids, mainly common fatty acids, and trihydroxy or polyhydroxyalcohols or polyols, such as pentaerythritol (PE), 2-ethyl-2-(hydroxymethyl)-1,3-propanediol or trimethylolpropane (TMP), and dipentaerythritol (diPE). The Group E substances often are referred to as "polyol esters". The polyol esters are unique in their chemical characteristics since they lack beta-tertiary hydrogen atoms, thus leading to stability against oxidation and elimination. The fatty acids often range from C5-C10 to as high as C18 (e.g., oleic, stearic, isostearic, tall oil fatty acids) in carbon number and generally are derived from naturally occurring sources. Group E esters may have multiple ester linkages and may include mixed esters derived from different carbon-length fatty acid mixtures. The lack of beta-tertiary hydrogen atoms in the structure of the polyol esters makes them characteristically and chemically stable against oxidation and elimination in comparison to other ester classes or groups. For these reasons, trimethylolpropane (TMP) and pentaerythritol (PE) esters with fatty acids of C5 to C10 carbon-chain length have applications as synthetic lubricants for passenger car motor oil and military and civilian jet engines. TMP and PE esters of C18 acids (e.g., isostearic and oleic acids) also have found use in synthetic lubricant applications, including refrigeration lubricants and hydraulic fluids. Because of their higher thermal stability characteristics, they also find use in a variety of high temperature applications such as industrial oven chain oils, high temperature greases, fire resistant transformer coolants and turbine engines. Polyol esters that are extensively esterified also have greater polarity, less volatility and enhanced lubricity characteristics. Acute toxicity: Depending on the degree of esterification, the polyol esters can be resistant or slow towards chemical or enzymatic hydrolysis (i.e., esterase or lipases) as a result of steric hindrance. PE and diPE esters that are capable of being enzymatically hydrolyzed will generate pentaerythritol or dipentaerythritol, and the corresponding fatty acids which, for most of the Group E esters, are comprised mainly of oleic, linoleic and stearic acids as well as the fatty acids in the C5-10 carbon-length. Similarly, TMP esters can undergo metabolism to yield trimethylolpropane (2-ethyl-2-hydroxymethyl-1,3-propanediol) and fatty acid constituents. Pentaerythritol and trimethylolpropane have been reported to have a low order of toxicity. The acute oral LD50 for these substances was greater than 2000 mg/kg indicating a relatively low order of toxicity. The similarity in the low order of toxicity for these substances is consistent with their similar chemical structure and physicochemical properties.

Metabolic studies of polyglyceryl esters indicated that these esters are hydrolyzed in the gastrointestinal (GI) tract, and utilization and digestibility studies supported the assumption that the fatty acid moiety is metabolized in the normal manner. Analytical studies have produced no evidence of accumulation of the polyglycerol moiety in body tissues. In an acute dermal toxicity study in rats, the LD50 of 1,2,3-propanetriol, homopolymer, diisooctadecanoate was >5000 mg/kg. Low toxicity was reported in acute oral studies. In rats, the LD50 >2000 mg/kg for polyglyceryl-3 caprate, polyglyceryl-3 caprylate, polyglyceryl-4 caprate, diisostearyl polyglyceryl-3 dimer dilinoleate, and the LD50 was >5000 mg/kg for polyglyceryl-3 iso-stearate, polyglyceryl-3-oleate, polyglyceryl-2 diisostearate and polyglyceryl-3 diisostearate. The ability to enhance skin penetration was examined for several of the polyglyceryl fatty acid esters.

Repeat dose toxicity: Polyol esters are generally well tolerated by rats in 28-day oral toxicity studies. NOAEL for these substances was 1000 mg/kg/day in Sprague-Dawley rats. The TMP ester of heptanoic and octanoic acid did not produce signs of overt systemic toxicity at any dose levels tested (i.e., 100, 300, and 1000 mg/kg/day). There were no treatment-related clinical in-life, functional observation battery, or gross postmortem findings. There were no treatment related mortality, and no adverse effects on body weight, food consumption, clinical laboratory parameters, or organ weights. However, there were increased numbers of hyaline droplets in the proximal cortical tubular epithelium of the 300 and 1000 mg/kg/day in male rats. Based on these findings (hyaline droplets), the NOAEL for this polyol ester was established at 100 mg/kg/day for male rats. Hyaline droplet formation observed in the male kidneys is believed to be a sex/species condition specific to only male rats, which has little relevance to humans.

The results from these repeated dose dermal toxicity studies suggest that polyol esters exhibit a low order of toxicity following repeated application. This may be attributable to similarities in their chemical structures, physicochemical properties, and common metabolic pathways (i.e., esters can be enzymatically hydrolyzed to the corresponding polyalcohol and the corresponding fatty acids). The polyol, hexanedioic acid, mixed esters with decanoic acid, heptanoic acid, octanoic acid and PE, was applied to the skin of groups of 10 (male and female) rats for five days a week for four (4) weeks at dose levels of 0, 125, 500 and 2000 mg/kg/day. Treated animals exhibited no signs indicative of systemic toxicity. No visible signs of irritation were observed at treatment sites. Microscopically, treated skin (viz., greater than or equal to 500 mg/kg/day) exhibited a dose-related increased incidence and severity of hyperplasia and hyperkeratosis of the epidermis and sebaceous gland hyperplasia. These effects were reversible. None of the minor changes in haematology and serum chemistry parameters were considered biologically significant. High dose females (2000 mg/kg/day) exhibited a significant increase in relative adrenal and brain weights when compared to the controls. These differences were attributed to the lower final body weight of the female animals. The NOAEL in this study for systemic toxicity was established as 500 mg/kg/day and 125 mg/kg/day for skin irritation.

Reproductive and developmental toxicity: Since metabolism of the polyol esters can occur, leading to the generation of the corresponding fatty acids and the polyol alcohol (such as pentaerythritol, trimethylolpropane, and dipentaerythritol), the issue of whether these metabolites may pose any potential reproductive/developmental toxicity concerns is important. However, the polyol alcohols such as pentaerythritol, trimethylolpropane, and dipentaerythritol, would be expected to undergo further metabolism, conjugation and excretion in the urine. Available evidence indicates that these ester hydrolysates (i.e., hydrolysis products), primarily fatty acids (e.g., heptanoic, octanoic, and decanoic acids) and secondarily the polyol alcohols should exhibit a low order of reproductive toxicity. It can be concluded that this group of high molecular weight polyol esters should not produce profound reproductive effects in rodents.

Genotoxicity: Polyols tested for genetic activity in the Salmonella assay, have been found to be inactive. Several polyol esters have been adequately tested for chromosomal mutation in the in vitro mammalian chromosome aberration assay, and all were inactive. Two TMP esters were also tested for in vivo chromosomal aberration in rats, and both demonstrated no activity. Thus, it is unlikely that these substances are chromosomal mutagens.

Carcinogenicity: In a 2-yr study, 28 male and 28 female rats were fed 5% polyglyceryl ester in the diet. No adverse effects on body weight, feed consumption, haematology values, or survival rate were noted. Liver function tests and renal function tests performed at 59 and 104 wks of the study were comparable between the test group and a control group fed 5% ground nut oil. The carcass fat contained no polyglycerol, and the levels of free fatty acid, unsaponifiable

Ethanol Shield

2,6-DI-TERT-BUTYL-
4-METHYLPHENOL

residue and fatty acid composition of carcass fat were not different from the controls. Organ weights, tumour incidence and tumour distribution were similar in control and test groups. A complete histological examination of major organs showed nothing remarkable

for bridged alkyl phenols:

Acute toxicity: Acute oral and dermal toxicity data are available for all but two of the substances in the group. The data show that acute toxicity of these substances is low. The testing for acute toxicity spans five decades

Repeat dose toxicity: Repeat dose studies on the members of this category include both subchronic and chronic exposures. The liver is identified as the target organ in rats for all of the substances tested. NOAEL's or NOEL's in rats for 13- week studies ranged from 100 ppm (approximately 5 mg/kg/day) to 500 ppm (approximately 25 mg/kg/day) while NOAEL's or NOEL's in rats for chronic studies were the same, 25 mg/kg/day (500 ppm).

Reproductive toxicity: Evaluation of effects on reproduction for the bridged alkyl phenols is supplemented by histopathological data on male and female reproductive organs in repeated dose studies. The data on the effects of bridged alkyl phenols on reproduction and reproductive organs span the range of structures and molecular weights. While not all of the data for reproductive effects are from reproduction studies, microscopic evaluations of reproductive organs along with other short-term tests for reproductive effects provide adequate data to evaluate the effects of these bridged alkyl phenols on reproduction. It can be concluded that reproductive toxicity is low.

Typically a two-year chronic feeding study provides data for 4,4'-thiobis-6-(t-butyl-m-cresol) (96-69-5). No adverse effects were noted on reproductive organs

Genotoxicity: Data from bacterial reverse mutation assays and *in vitro* and *in vivo* chromosome aberration studies were reviewed. Adequate bacterial gene mutation assays have been conducted with all of the category chemicals except two. Chromosome aberration studies, *in vitro* and/or *in vivo*, are available for all but two substances. The mutagenicity data span the range of structures and molecular weights and data can be bridged from other members of the group to meet any outstanding requirements. The weight of evidence for mutagenic potential for this category indicates these substances are not mutagenic.

Carcinogenicity: The mutagenicity data combined with the animal data plus the long historical use of BHT (128-37-0) indicate that the chemicals in this class are not expected to exhibit any significant potential to cause cancer. The weight of the evidence indicates that these chemicals are not genotoxic.

The Bridged Alkyl Phenols Category consists of a group of chemicals in which two molecules of mono or di-substituted alkyl (C1, C4, and/or C9) phenols are "bridged" or linked by a single atom (carbon or sulfur). The carbon atom linking the alkyl phenol groups contains hydrogen, propyl, or methyl substitutions. CAS No. 128-37-0 (BHT) is included in this category for data purposes because it is an alkyl phenol with a single carbon group such as the ones that link the phenol groups

For hindered phenols:

Available data shows that acute toxicity of these substances is low.

Mutagenicity. Data from bacterial reverse mutation assays and *in vitro* and *in vivo* chromosome aberration studies were reviewed. All assays, with and without metabolic activation, were negative. The weight of evidence for mutagenic potential for this category indicates these substances are not mutagenic.

In Vitro Chromosome Aberration Studies. *In vitro* chromosome aberration studies are available for several members. All except 2,6-di-tert-butyl-p-cresol were negative

In Vivo Chromosome Aberration Studies. *In vivo* studies evaluating chromosome damage are available for six of the hindered phenols. All *in vivo* evaluations were negative.

Repeated Dose Toxicity. Repeated dose toxicity data of approximately three months (90-day, 12- and 13-week) are available for some of the substances in this group. The liver was the target organ in rats for almost all of the substances with subchronic toxicity data in that species. Other target organs included thyroid and kidney and mesenteric lymph nodes. NOAELs in rats ranged from 100 ppm (approximately 5 mg/kg/day) to 10,000 ppm (500 mg/kg/day)

Carcinogenicity: Data is available for 2,6-di-tert-butyl-p-cresol (128-37-0); and 4,4'-thiobis-6-(t-butyl-m-cresol) (96-69-5). Liver adenomas were reported for 2,6-di-tert-butyl-p-cresol (128-37-0) and a NOAEL was established for the study at 25 mg/kg/day. 4,4'-Thiobis-6-(t-butyl-m-cresol) (96-69-5) was not carcinogenic in rats or mice, but the kidney was identified as a target organ in female rats

NOTE: 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.

* Degussa SDS Effects such as behavioral changes, reduction in body weight gain, and decrement in body weight have been observed after long-term administration of BHT to mice and rats. Toxic effects may be attributed more to BHT metabolites than to their parent compound, only a few studies have focused on their carcinogenicity and toxicity, and not only on that of BHT. The metabolite BHT-QM (syn: 2,6-di-tert-butyl-1,4-methylene-2,5-cyclohexadien-1-one, CAS RN: 2607-52-5) is a very reactive compound which is considered to play a significant role in hepatotoxicity, pneumotoxicity, and skin tumor promotion in mice. In addition, it was reported that another quinone derivative, BHT-OH(t)QM (syn 2-tert-butyl-6-(2-hydroxy-tert-butyl-4-methylene-2,5-cyclohexadien-1-one, CAS RN: 124755-19-7), is chemically more reactive than BHT-QM, and it has been recognized as the principal metabolite responsible for lung tumor promotion activity of BHT in mice. BHT has been reported to exert prooxidant effects under certain conditions. Thus, when BHT was added in excess to a wheat seedling medium in aerobic conditions, an enhancement of the generation rate of superoxide anion was observed. This is a reactive particle that may damage cellular structures at high concentrations. In addition, an increase in hepatic microsomal lipid peroxidation was observed in rats fed with diets containing 0.2% of BHT for 30 days. Due to this ability of BHT to exert prooxidant effects at high concentrations, it has been used to induce experimental models of oxidative stress in several animals and fungi in order to study the protective effects of other compounds. Quinone methide derivatives form adducts with several proteins, including enzymes that protect cells from oxidative stress; this prooxidant state can also lead to cell oxidative damage. It must be noted that relationships between chronic oxidative stress and tumor promotion are well known. Some authors have reported that at high aeration rate, BHT can react with molecular oxygen rather than with the reactive oxygen species present, yielding BHT-phenoxyl radical and superoxide anion. In addition, the phenolic radical itself may undergo redox recycling which can be a critical factor depending on the

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	<p>reductant involved. However, it has to be noted that BHT-phenoxy radical has been reported to be relatively stable. Furthermore, the potential reactivity of BHT-derived metabolites should be taken into account; some studies reported that not only BHT but also its metabolites, such as BHT-Q and BHT-QM, can act as prooxidant. As BHT undergoes several reactions during biotransformation, a large number of intermediate metabolites have been identified. However, their nature and concentration depend on the environmental conditions and on the animal species. Although the changes undergone by BHT during in vivo digestion processes have not been studied, after submission of a fluid deep-frying fat containing BHT and BHT-QM to an in vitro gastrointestinal digestion model, both these were detected in the digested samples. These results indicate that BHT and its toxic metabolite could remain bioaccessible for intestinal absorption. Studies concerning BHT metabolism have shown that, unlike other synthetic antioxidants, BHT is a potent inducer of the microsomal monooxygenase system and its major route of degradation is oxidation catalyzed by cytochrome P450. Studies have reported potential toxicity derived from the ingestion or administration of BHT. As for acute oral toxicity, although this is considered low in animals, it must be noted that 2 clinical cases were reported in patients who suffered acute neurotoxicity and gastritis after ingesting a high dose of BHT (4 and 80 g without medical prescription) to cure recurrent genital herpes. Regarding short-term subchronic toxicity studies, it has been reported that BHT causes dose-related increase in the incidence and severity.</p>
1H-BENZOTRIAZOLE	<p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>Bacterial mutagenicity: E. coli positive. Ames positive; HGPRT negative; micronucleus test (mouse) negative **** * [Ciba Geigy] ** [Bayer] *** Merck **** Benzotriazoles Coalition Synthetic Organic Chemical Manufacturers Association December, 2001</p>
DIMETHYLETHANOLAMINE	<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"> ▶ Many amine-based compounds can induce histamine liberation, which, in turn, can trigger allergic and other physiological effects, including bronchoconstriction or bronchial asthma and rhinitis. ▶ Systemic symptoms include headache, nausea, faintness, anxiety, a decrease in blood pressure, tachycardia (rapid heartbeat), itching, erythema (reddening of the skin), urticaria (hives), and facial edema (swelling). Systemic effects (those affecting the body) that are related to the pharmacological action of amines are usually transient. <p>Typically, there are four routes of possible or potential exposure: inhalation, skin contact, eye contact, and ingestion.</p> <p>Inhalation:</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.</p> <p>Products with higher vapour pressures have a greater potential for higher airborne concentrations. This increases the probability of worker exposure.</p> <p>Higher concentrations of certain amines can produce severe respiratory irritation, characterised by nasal discharge, coughing, difficulty in breathing, and chest pains.</p> <p>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.</p> <p>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.</p> <p>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>Skin Contact:</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>Eye Contact:</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.)</p> <p>Exposed persons may experience excessive tearing, burning, conjunctivitis, and corneal swelling.</p> <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>Ingestion:</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>

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Material aspirated (due to vomiting) can damage the bronchial tubes and the lungs.

Affected persons also may experience pain in the chest or abdomen, nausea, bleeding of the throat and the gastrointestinal tract, diarrhea, dizziness, drowsiness, thirst, circulatory collapse, coma, and even death.

Polyurethane Amine Catalysts: Guidelines for Safe Handling and Disposal; Technical Bulletin June 2000

Alliance for Polyurethanes Industry

For dimethylethanolamine (DMAE) and selected salts and esters:

Toxicology:

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

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

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

A large number of adverse health effects are associated with DMAE. These include cardiovascular, neurological, and/or psychological effects. Specific attribution of adverse effects to DMAE is unlikely, as many of these products also contained Ephedra vulgaris alkaloids and other Ephedra spp. Ephedra alkaloids cause similar cardiovascular and neurological effects reported for DMAE.

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

A meta-analysis of randomized controlled trials indicated that DMAE was no more effective than placebo in the treatment of tardive dyskinesia. Rather, there was a significantly increased risk of adverse events associated with the DMAE treatment.

DMAE treatment increases the concentration of choline in both the plasma and the brain of treated rats; the mechanism for this phenomenon was unknown. Since it was known that DMAE inhibits the influx of choline to the brain across the blood brain barrier, it is possible that DMAE also inhibited the efflux of choline from the brain, resulting in an accumulation in the brain.

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

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

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

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

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

Intravenous administration of DMAE (175 to 350 mg/kg; 1.95 to 3.90 mmol/kg) resulted in dose-dependant psychoanaleptic effects (as demonstrated by spontaneous running in mice) and an influence on conditioned reflexes in rats.

DMAE appears to exert a central vasomotor stimulant effect. Intracerebroventricular (ICV) administration of DMAE (0.1 to 2.0 mg; 1.0 to 20 μ mol) resulted in potentiation of the carotid occlusion response (all doses) resulting in an increase in blood pressure in dogs (higher doses). This effect was not abolished by atropine sulfate (ICV).

With meclofenoxate (centrophenoquine hydrochloride) treatment (10 to 40 mg/kg body weight; 0.040 to 0.16 mmol/kg), a significant dose-dependent reduction in both blood pressure (up to 49.7+/-0.39 mmHg reduction) and heart rate (up to 71 +/-4.5% reduction) was observed in the old rats at the 40 mg/kg (0.16 mmol/kg) dose level

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

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

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

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

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

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

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

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

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

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

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

1,2,4-TRIMETHYL BENZENE

CHEMWATCH 2325 1,3,5-trimethylbenzene

1,3,5-TRIMETHYL BENZENE

CHEMWATCH 12171 1,2,4-trimethylbenzene

XYLENE

Reproductive effector in rats

CUMENE

For aromatic terpenes:

Acute toxicity: Mammalian LD50 for p-cymene have shown it to have low toxic potential. Similar studies with cumene have concurred with these results

In general, the studies indicate that p-cymene (p-methylisopropylbenzene) or cumene (isopropylbenzene) is rapidly absorbed by oral or inhalation routes. They undergo oxidation (hydroxylation) of the side chain isopropyl substituent and, in the case of p-cymene, the methyl substituent to yield polar oxygenated metabolites. These metabolites are either excreted unchanged in the urine or undergo Phase II conjugation with glucuronic acid and/or glycine followed by excretion in the urine. Unchanged p-cymene or cumene were not detected in the urine or faeces.

Humans (5 males and 5 females/group) exposed to an atmosphere containing 49, 98, or 147 ppm cumene for 7 hours

Ethanol Shield

showed 64% absorption at 0.5 hours and 45% at 7 hours. Maximum excretion is observed at 6 to 8 hours and is essentially complete at 48 hours. Approximately 35% of the dose inhaled was excreted as 2-phenyl-2-propanol

Repeat Dose Toxicity: Subacute Studies: Groups of 7 to 12 male rats were exposed to 0, 50, or 250 ppm of p-cymene for 6 hours/day, 5 days/week for 4 weeks with an 8-week recovery period. there was no overt toxicity in the treated rats and no effect on body weight or terminal weight of the brain, cerebellum or whole brain. There was also no effect on regional enzyme activities, regional protein synthesis or regional neurotransmitter concentrations.

Cumene has been tested by the National Toxicology Program (NTP) in both rats and mice. Animals were exposed to up to 4,000 ppm cumene by whole-body inhalation for 12-13 days over a period of 16-17 days. In rats, all animals died at 4,000 ppm, and about half the animals died at the next exposure concentration (2,000 ppm). Varying degrees of ataxia were reported in surviving rats exposed to 500 to 2,000 ppm cumene. Increased relative liver and kidney weights were reported in rats exposed to cumene. In exposed male rats, hyaline droplets in the renal cortical tubules were reported. At 2,000 ppm, superlative inflammation of the lung was reported in 40% of the rats. In mice, all animals died at the 2 highest exposures (2,000 and 4,000 ppm). At 1,000 ppm, 80% of the female mice died and male mice showed varying degrees of ataxia. Increased relative liver and kidney weights were reported in mice exposed to cumene. Decreased thymus weight was reported in male mice exposed to 1,000 ppm of cumene. No histopathological findings accompanied the organ weight changes. A NOAEL of 1,000 ppm was determined for female rats and male mice and a NOAEL of 500 ppm was determined for female mice based on mortality and histopathological findings.

Chronic toxicity: The US EPA concluded that there is some evidence that suggests that cumene is not likely to produce a carcinogenic response (i.e., numerous genotoxic tests, including gene mutation, chromosomal aberration, and primary DNA damage tests, all but one of which were negative or not reproducible) In addition, EPA noted that cumene does not appear to metabolise to highly reactive chemical species and in terms of metabolism, cumene is analogous to methyl benzene for which a 2-year inhalation study was conducted by NTP and no evidence of carcinogenic activity was reported in either rats or mice.

Given that the only structural difference between p-cymene and cumene is the presence of a second alkyl substituent (isopropylbenzene versus p-methylisopropylbenzene), similar conclusions can be drawn for p-cymene, particularly since the pharmacokinetic, metabolic and toxicologic data that are available support this conclusion.

Reproductive toxicity: Taking into consideration the rapid metabolism and excretion of cumene, the US EPA concluded, "cumene has low potential for reproductive toxicity."

Developmental toxicity: Even at maternally toxic concentrations exposure to cumene vapor did not produce developmental toxicity in rats. However the US EPA determined that the changes in gestational parameters of the rabbits, though not significant, were consistent in indicating possible developmental effects and therefore set the NOAEL in rabbits for both developmental and maternal effects at 1,206 ppm and the LOAEL at 2,297 ppm, respectively (as reported in EPA, 1997). Since both cumene and p-cymene exhibit such similar pharmacokinetic and metabolic profiles, and show no evidence of toxicity at levels of exposure similar to those experienced by humans, further teratogenic or developmental testing is not recommended

Genotoxicity: The genotoxicity database on p-cymene and cumene shows no mutagenic potential in the Ames assay. In cytogenetic assays, there is no evidence of a genotoxic potential in vitro. In whole animals, the genotoxicity results for cumene are mixed showing weakly positive results in micronuclei induction in rats, but no evidence of genotoxicity in mice.

Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen

[National Toxicology Program: U.S. Dep. of Health & Human Services 2002]

WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.

Cumene is reasonably anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity from studies in experimental animals. Cumene caused tumours at several tissue sites, including lung and liver in mice and kidney in male rats. Several proposed mechanisms of carcinogenesis support the relevance to humans of lung and liver tumours in experimental animals. Specifically, there is evidence that humans and experimental animals metabolise cumene through similar metabolic pathways. There is also evidence that cumene is genotoxic in some tissues, based on findings of DNA damage in rodent lung and liver. Furthermore, mutations of the K-ras oncogene and p53 tumor-suppressor gene observed in cumene-induced lung tumours in mice, along with altered expression of many other genes, resemble molecular alterations found in human lung and other cancers. The relevance of the kidney tumors to cancer in humans is uncertain; there is evidence that a species-specific mechanism not relevant to humans contributes to their induction, but it is possible that other mechanisms relevant to humans, such as genotoxicity, may also contribute to kidney-tumour formation in male rats.

ETHYLENE GLYCOL MONOBUTYL ETHER & DIMETHYLETHANOLAMINE & XYLENE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
ETHYLENE GLYCOL MONOBUTYL ETHER & DIMETHYLETHANOLAMINE	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.
CAPRYLIC/ CAPRIC TRIGLYCERIDE & 1,3,5- TRIMETHYL BENZENE	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
CAPRYLIC/ CAPRIC TRIGLYCERIDE & 2,6-DI- TERT-BUTYL- 4-METHYLPHENOL & 1,3,5-TRIMETHYL	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

Continued...

Ethanol Shield

BENZENE & XYLENE & CUMENE	
2,6-DI-TERT-BUTYL-4-METHYLPHENOL & 1H-BENZOTRIAZOLE & DIMETHYLETHANOLAMINE & 1,2,4-TRIMETHYL BENZENE & 1,3,5-TRIMETHYL BENZENE & CUMENE & 1,2,3-TRIMETHYL BENZENE	<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>
2,6-DI-TERT-BUTYL-4-METHYLPHENOL & XYLENE	<p>The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.</p>
1,2,4-TRIMETHYL BENZENE & 1,3,5-TRIMETHYL BENZENE & 1,2,3-TRIMETHYL BENZENE	<p>For trimethylbenzenes: Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure. Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal. Following oral administration of the chemical to rats, 62.6% of the dose was recovered as urinary metabolites indicating substantial absorption. 1,2,4-Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. In the blood stream, approximately 85% of the chemical is bound to red blood cells. Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion. After a single oral dose to rats of 1200 mg/kg, urinary metabolites consisted of approximately 43.2% glycine, 6.6% glucuronic, and 12.9% sulfuric acid conjugates. The two principle metabolites excreted by rabbits after oral administration of 438 mg/kg/day for 5 days were 2,4-dimethylbenzoic acid and 3,4-dimethylhippuric acid. The major routes of excretion of 1,2,4-trimethylbenzene are exhalation of parent compound and elimination of urinary metabolites. Half-times for urinary metabolites were reported as 9.5 hours for glycine, 22.9 hours for glucuronide, and 37.6 hours for sulfuric acid conjugates.</p> <p>Acute Toxicity Direct contact with liquid 1,2,4-trimethylbenzene is irritating to the skin and breathing the vapor is irritating to the respiratory tract causing pneumonitis. Breathing high concentrations of the chemical vapor causes headache, fatigue, and drowsiness. In humans liquid 1,2,4-trimethylbenzene is irritating to the skin and inhalation of vapor causes chemical pneumonitis. High concentrations of vapor (5000-9000 ppm) cause headache, fatigue, and drowsiness. The concentration of 5000 ppm is roughly equivalent to a total of 221 mg/kg assuming a 30 minute exposure period (see end note 1). 2. Animals - Mice exposed to 8130-9140 ppm 1,2,4-trimethylbenzene (no duration given) had loss of righting response and loss of reflexes. Direct dermal contact with the chemical (no species given) causes vasodilation, erythema, and irritation (U.S. EPA). Seven of 10 rats died after an oral dose of 2.5 mL of a mixture of trimethylbenzenes in olive oil (average dose approximately 4.4 g/kg). Rats and mice were exposed by inhalation to a coal tar distillate containing about 70% 1,3,5- and 1,2,4-trimethylbenzene; no pathological changes were noted in either species after exposure to 1800-2000 ppm for up to 48 continuous hours, or in rats after 14 exposures of 8 hours each at the same exposure levels. No effects were reported for rats exposed to a mixture of trimethylbenzenes at 1700 ppm for 10 to 21 days.</p> <p>Neurotoxicity 1,2,4-Trimethylbenzene depresses the central nervous system. Exposure to solvent mixtures containing the chemical causes headache, fatigue, nervousness, and drowsiness. Occupationally, workers exposed to a solvent containing 50% 1,2,4-trimethylbenzene had nervousness, headaches, drowsiness, and vertigo (U.S. EPA). Headache, fatigue, and drowsiness were reported for workers exposed (no dose given) to paint thinner containing 80% 1,2,4- and 1,3,5-trimethylbenzenes.</p> <p>Results of the developmental toxicity study indicate that the C9 fraction caused adverse neurological effects at the highest dose (1500 ppm) tested.</p> <p>Subchronic/Chronic Toxicity Long-term exposure to solvents containing 1,2,4-trimethylbenzene may cause nervousness, tension, and bronchitis. Painters that worked for several years with a solvent containing 50% 1,2,4- and 30% 1,3,5-trimethylbenzene showed nervousness, tension and anxiety, asthmatic bronchitis, anemia, and alterations in blood clotting; haematological effects may have been due to trace amounts of benzene.</p> <p>Rats given 1,2,4-trimethylbenzene orally at doses of 0.5 or 2.0 g/kg/day, 5 days/week for 4 weeks. All rats exposed to the high dose died and 1 rat in the low dose died (no times given); no other effects were reported. Rats exposed by inhalation to 1700 ppm of a trimethylbenzene isomeric mixture for 4 months had decreased weight gain, lymphopenia and neutrophilia.</p> <p>Genotoxicity: Results of mutagenicity testing, indicate that the C9 fraction does not induce gene mutations in prokaryotes (Salmonella typhimurium/mammalian microsome assay); or in mammalian cells in culture (in Chinese hamster ovary cells with and without activation). The C9 fraction does not induce chromosome mutations in Chinese hamster ovary cells with and without activation; does not induce chromosome aberrations in the bone marrow of Sprague-Dawley rats exposed by inhalation (6 hours/day for 5 days); and does not induce sister chromatid exchange in Chinese hamster ovary cells with and without activation.</p> <p>Developmental/Reproductive Toxicity: A three-generation reproductive study on the C9 fraction was conducted. CD rats (30/sex/group) were exposed by inhalation to the C9 fraction at concentrations of 0, 100, 500, or 1500 ppm (0, 100, 500, or 1500 mg/kg/day) for 6 hours/day, 5 days/week. There was evidence of parental and reproductive toxicity at all dose levels. Indicators of parental toxicity included reduced body weights, increased salivation, hunched posture, aggressive behavior, and death. Indicators of adverse reproductive system effects included reduced litter size and reduced pup body weight. The LOEL was 100 ppm; a no-observed-effect level was not established. Developmental toxicity, including possible developmental neurotoxicity, was evident in rats in a 3-generation reproductive study.</p>

Continued...

Ethanol Shield

	No effects on fecundity or fertility occurred in rats treated dermally with up to 0.3 mL/rat/day of a mixture of trimethylbenzenes, 4-6 hours/day, 5 days/week over one generation
1,2,4-TRIMETHYL BENZENE & 1,3,5- TRIMETHYL BENZENE	Other Toxicity data is available for CHEMWATCH 12172 1,2,3-trimethylbenzene

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

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

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Ethanol Shield	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available

ethylene glycol monobutyl ether	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1-700mg/L	2
	EC50	48	Crustacea	ca. 1-800mg/L	2
	EC50	72	Algae or other aquatic plants	1-840mg/L	2
	NOEC	24	Crustacea	>1-mg/L	2

caprylic/ capric triglyceride	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	≥53mg/L	1
	EC50	48	Crustacea	>0.01mg/L	2
	EC50	72	Algae or other aquatic plants	>0.449mg/L	2
	EC10	72	Algae or other aquatic plants	>0.449mg/L	2
2,6-di-tert-butyl- 4-methylphenol	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.199mg/L	2
	EC50	48	Crustacea	>0.17mg/L	2
	EC50	96	Algae or other aquatic plants	0.228mg/L	3
	NOEC	504	Crustacea	0.023mg/L	2

1H-benzotriazole	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	46.868mg/L	3
	EC50	48	Crustacea	8.58mg/L	2
	EC50	96	Algae or other aquatic plants	18.560mg/L	3
	EC10	504	Crustacea	0.4mg/L	2
dimethylethanolamine	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	88-131mg/L	1
	EC50	48	Crustacea	98.37mg/L	2
	EC50	72	Algae or other aquatic plants	≈35mg/L	1
	EC20	72	Algae or other aquatic plants	18mg/L	1
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	NOEC	96	Fish	100mg/L	1

Continued...

Ethanol Shield

1,2,4-trimethyl benzene	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1.318mg/L	3
	EC50	48	Crustacea	ca.6.14mg/L	2
	EC50	96	Algae or other aquatic plants	2.154mg/L	3
1,3,5-trimethyl benzene	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1.318mg/L	3
	EC50	48	Crustacea	13mg/L	5
	EC50	96	Algae or other aquatic plants	2.154mg/L	3
xylene	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	2.6mg/L	2
	EC50	48	Crustacea	1.8mg/L	2
	EC50	72	Algae or other aquatic plants	3.2mg/L	2
cumene	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1.784mg/L	3
	EC50	48	Crustacea	0.6mg/L	2
	EC50	72	Algae or other aquatic plants	1.29mg/L	2
1,2,3-trimethyl benzene	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1.318mg/L	3
	EC50	96	Algae or other aquatic plants	2.154mg/L	3
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			

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

For ethylene glycol monoalkyl ethers and their acetates:

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

Environmental fate:

The ethers, like other simple glycol ethers possess no functional groups that are readily subject to hydrolysis in the presence of waters. The acetates possess an ester group that hydrolyses in neutral ambient water under abiotic conditions.

Level III fugacity modeling indicates that category members, when released to air and water, will partition predominately to water and, to a lesser extent, to air and soil. Estimates of soil and sediment partition coefficients (Kocs ranging from 1- 10) suggest that category members would exhibit high soil mobility. Estimated bioconcentration factors (log BCF) range from 0.463 to 0.732. Biodegradation studies indicate that all category members are readily biodegradable. The physical chemistry and environmental fate properties indicate that category members will not persist or bioconcentrate in the environment.

Ecotoxicity:

Glycol ether acetates do not hydrolyse rapidly into their corresponding glycol ethers in water under environmental conditions. The LC50 or EC50 values for EGHE are lower than those for EGPE and EGBE (which have shorter chain lengths and lower log Kow values). Overall, the LC50 values for the glycol ethers in aquatic species range from 94 to > 5000 mg/L. For EGHE, the 96-hour LC50 for *Brachydanio rerio* (zebra fish) is between 94 and mg/L, the 48-hour EC50 for *Daphnia magna* was 145 mg/L and the 72-hour EC50 values for biomass and growth rate of algae (*Scenedesmus subspicatus*) were 98 and 198 mg/L, respectively. LC50/EC50 values for EGPE and EGBE in aquatic species are 835 mg/l or greater.

Aquatic toxicity data for EGBEA show a 96-hour LC50 of 28.3 mg/L for rainbow trout (*Oncorhynchus mykiss*), a 48-hour LC50 of 37-143 mg/L for *Daphnia magna*, a 72-hour EC50 of greater than 500 mg/L for biomass or growth rate of algae (*Scenedesmus subspicatus* and *Pseudokirchneriella subcapitata*, respectively), and a 7-day EC10 of 30.4 mg/L and a NOEC of 16.4 mg/L for reproduction in *Ceriodaphnia dubia*.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ethylene glycol monobutyl ether	LOW (Half-life = 56 days)	LOW (Half-life = 1.37 days)
2,6-di-tert-butyl-4-methylphenol	HIGH	HIGH
1H-benzotriazole	HIGH	HIGH
dimethylethanolamine	LOW	LOW

Continued...

Ethanol Shield

1,2,4-trimethyl benzene	LOW (Half-life = 56 days)	LOW (Half-life = 0.67 days)
1,3,5-trimethyl benzene	HIGH	HIGH
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
cumene	HIGH	HIGH
1,2,3-trimethyl benzene	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
ethylene glycol monobutyl ether	LOW (BCF = 2.51)
2,6-di-tert-butyl-4-methylphenol	HIGH (BCF = 2500)
1H-benzotriazole	LOW (BCF = 15)
dimethylethanolamine	LOW (LogKOW = -0.9351)
1,2,4-trimethyl benzene	LOW (BCF = 275)
1,3,5-trimethyl benzene	LOW (BCF = 342)
xylene	MEDIUM (BCF = 740)
cumene	LOW (BCF = 35.5)
1,2,3-trimethyl benzene	LOW (BCF = 259)

Mobility in soil

Ingredient	Mobility
ethylene glycol monobutyl ether	HIGH (KOC = 1)
2,6-di-tert-butyl-4-methylphenol	LOW (KOC = 23030)
1H-benzotriazole	LOW (KOC = 996.2)
dimethylethanolamine	HIGH (KOC = 1.602)
1,2,4-trimethyl benzene	LOW (KOC = 717.6)
1,3,5-trimethyl benzene	LOW (KOC = 703)
cumene	LOW (KOC = 817.2)
1,2,3-trimethyl benzene	LOW (KOC = 732.5)



SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Authority for disposal. ▶ Bury or incinerate residue at an approved site. ▶ Recycle containers if possible, or dispose of in an authorised landfill.
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SECTION 14 TRANSPORT INFORMATION

Labels Required

	
Marine Pollutant	
HAZCHEM	2X

Land transport (ADG)

UN number	2810	
UN proper shipping name	TOXIC LIQUID, ORGANIC, N.O.S. (contains ethylene glycol monobutyl ether)	
Transport hazard class(es)	Class	6.1
	Subrisk	Not Applicable
Packing group	III	
Environmental hazard	Environmentally hazardous	
Special precautions for user	Special provisions	223 274
	Limited quantity	5 L

Air transport (ICAO-IATA / DGR)

UN number	2810	
UN proper shipping name	Toxic liquid, organic, n.o.s. * (contains ethylene glycol monobutyl ether)	
Transport hazard class(es)	ICAO/IATA Class	6.1
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	6L
Packing group	III	
Environmental hazard	Environmentally hazardous	
Special precautions for user	Special provisions	A3 A4 A137
	Cargo Only Packing Instructions	663
	Cargo Only Maximum Qty / Pack	220 L
	Passenger and Cargo Packing Instructions	655
	Passenger and Cargo Maximum Qty / Pack	60 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y642
	Passenger and Cargo Limited Maximum Qty / Pack	2 L

Sea transport (IMDG-Code / GGVSee)

UN number	2810	
UN proper shipping name	TOXIC LIQUID, ORGANIC, N.O.S. (contains ethylene glycol monobutyl ether)	
Transport hazard class(es)	IMDG Class	6.1
	IMDG Subrisk	Not Applicable
Packing group	III	
Environmental hazard	Marine Pollutant	
Special precautions for user	EMS Number	F-A , S-A
	Special provisions	223 274
	Limited Quantities	5 L

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

Not Applicable

SECTION 15 REGULATORY INFORMATION**Safety, health and environmental regulations / legislation specific for the substance or mixture**

ETHYLENE GLYCOL MONOBUTYL ETHER(111-76-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Continued...

Ethanol Shield

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List
 Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes
 Australia Exposure Standards
 Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
 Australia Inventory of Chemical Substances (AICS)
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix F (Part 3)
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Index
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Part 2, Section Seven - Appendix I

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
 GESAMP/EHS Composite List - GESAMP Hazard Profiles
 IMO IBC Code Chapter 17: Summary of minimum requirements
 IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
 International Air Transport Association (IATA) Dangerous Goods Regulations
 International Maritime Dangerous Goods Requirements (IMDG Code)
 United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

CAPRYLIC/ CAPRIC TRIGLYCERIDE(73398-61-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)
 IMO IBC Code Chapter 17: Summary of minimum requirements

IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk

2,6-DI-TERT-BUTYL-4-METHYLPHENOL(128-37-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List
 Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes
 Australia Exposure Standards
 Australia Inventory of Chemical Substances (AICS)
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix F (Part 3)
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Index
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2

GESAMP/EHS Composite List - GESAMP Hazard Profiles
 IMO IBC Code Chapter 17: Summary of minimum requirements
 IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
 International Air Transport Association (IATA) Dangerous Goods Regulations
 International Maritime Dangerous Goods Requirements (IMDG Code)
 United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

1H-BENZOTRIAZOLE(95-14-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List
 Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes
 Australia Inventory of Chemical Substances (AICS)
 GESAMP/EHS Composite List - GESAMP Hazard Profiles
 IMO IBC Code Chapter 17: Summary of minimum requirements

IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk
 International Air Transport Association (IATA) Dangerous Goods Regulations
 International Maritime Dangerous Goods Requirements (IMDG Code)
 United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

DIMETHYLETHANOLAMINE(108-01-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List
 Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes
 Australia Exposure Standards
 Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
 Australia Inventory of Chemical Substances (AICS)
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix F (Part 3)
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Index

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
 GESAMP/EHS Composite List - GESAMP Hazard Profiles
 IMO IBC Code Chapter 17: Summary of minimum requirements
 IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk
 International Air Transport Association (IATA) Dangerous Goods Regulations
 International Maritime Dangerous Goods Requirements (IMDG Code)
 United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

1,2,4-TRIMETHYL BENZENE(95-63-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Ethanol Shield

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List
Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Index
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7
GESAMP/EHS Composite List - GESAMP Hazard Profiles

IMO IBC Code Chapter 17: Summary of minimum requirements
IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk
IMO Provisional Categorization of Liquid Substances - List 2: Pollutant only mixtures containing at least 99% by weight of components already assessed by IMO
IMO Provisional Categorization of Liquid Substances - List 3: (Trade-named) mixtures containing at least 99% by weight of components already assessed by IMO, presenting safety hazards
International Air Transport Association (IATA) Dangerous Goods Regulations
International Maritime Dangerous Goods Requirements (IMDG Code)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

1,3,5-TRIMETHYL BENZENE(108-67-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List
Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Index
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7
GESAMP/EHS Composite List - GESAMP Hazard Profiles

IMO IBC Code Chapter 17: Summary of minimum requirements
IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk
IMO Provisional Categorization of Liquid Substances - List 2: Pollutant only mixtures containing at least 99% by weight of components already assessed by IMO
IMO Provisional Categorization of Liquid Substances - List 3: (Trade-named) mixtures containing at least 99% by weight of components already assessed by IMO, presenting safety hazards
International Air Transport Association (IATA) Dangerous Goods Regulations
International Maritime Dangerous Goods Requirements (IMDG Code)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

XYLENE(1330-20-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List
Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes
Australia Exposure Standards
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Hazardous chemicals which may require Health Monitoring
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix F (Part 3)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Index
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Part 2, Section Seven - Appendix I
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7
GESAMP/EHS Composite List - GESAMP Hazard Profiles
IMO IBC Code Chapter 17: Summary of minimum requirements
IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk
IMO Provisional Categorization of Liquid Substances - List 3: (Trade-named) mixtures containing at least 99% by weight of components already assessed by IMO, presenting safety hazards
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Air Transport Association (IATA) Dangerous Goods Regulations
International Maritime Dangerous Goods Requirements (IMDG Code)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

CUMENE(98-82-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List
Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes
Australia Exposure Standards
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
GESAMP/EHS Composite List - GESAMP Hazard Profiles

IMO IBC Code Chapter 17: Summary of minimum requirements
IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk
IMO Provisional Categorization of Liquid Substances - List 2: Pollutant only mixtures containing at least 99% by weight of components already assessed by IMO
IMO Provisional Categorization of Liquid Substances - List 3: (Trade-named) mixtures containing at least 99% by weight of components already assessed by IMO, presenting safety hazards
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Air Transport Association (IATA) Dangerous Goods Regulations
International Maritime Dangerous Goods Requirements (IMDG Code)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

1,2,3-TRIMETHYL BENZENE(526-73-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Ethanol Shield

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List
 Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes
 Australia Inventory of Chemical Substances (AICS)
 GESAMP/EHS Composite List - GESAMP Hazard Profiles
 IMO IBC Code Chapter 17: Summary of minimum requirements
 IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk

IMO Provisional Categorization of Liquid Substances - List 2: Pollutant only mixtures containing at least 99% by weight of components already assessed by IMO
 IMO Provisional Categorization of Liquid Substances - List 3: (Trade-named) mixtures containing at least 99% by weight of components already assessed by IMO, presenting safety hazards
 International Air Transport Association (IATA) Dangerous Goods Regulations
 International Maritime Dangerous Goods Requirements (IMDG Code)
 United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

National Inventory Status

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (xylene; 1H-benzotriazole; 1,3,5-trimethyl benzene; 1,2,3-trimethyl benzene; cumene; 1,2,4-trimethyl benzene; caprylic/ capric triglyceride; ethylene glycol monobutyl ether; dimethylethanolamine)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (1,2,3-trimethyl benzene)
Vietnam - NCI	Yes
Russia - ARIPS	No (1,2,3-trimethyl benzene; caprylic/ capric triglyceride)
Thailand - TECl	No (1,2,4-trimethyl benzene)
Legend:	<p>Yes = All CAS declared ingredients are on the inventory</p> <p>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)</p>

SECTION 16 OTHER INFORMATION

Revision Date	30/07/2019
Initial Date	23/07/2019

SDS Version Summary

Version	Issue Date	Sections Updated
4.1.1.1	29/07/2019	Classification, Ingredients
5.1.1.1	30/07/2019	Physical Properties

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.

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.

Continued...

IDLH: Immediately Dangerous to Life or Health Concentrations

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

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