

## ColorSpec Caliper HT, 300g (ColorSpec Caliper HT)

### Motor Active

Chemwatch Hazard Alert Code: 4

Chemwatch: 41-9271

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Safety Data Sheet according to WHS and ADG requirements

Initial Date: **Not Available**

L.GHS.AUS.EN

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

### Product Identifier

<b>Product name</b>	ColorSpec Caliper HT, 300g (ColorSpec Caliper HT)
<b>Synonyms</b>	Product Code: CSCHT300G
<b>Proper shipping name</b>	AEROSOLS
<b>Other means of identification</b>	Not Available

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

<b>Relevant identified uses</b>	Application is by spray atomisation from a hand held aerosol pack Use according to manufacturer's directions.
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### Details of the supplier of the safety data sheet

<b>Registered company name</b>	Motor Active
<b>Address</b>	35 Slough Business Park, Holker Street Silverwater NSW 2128 Australia
<b>Telephone</b>	+61 2 9737 9422 1800 350 622
<b>Fax</b>	+61 2 9737 9414
<b>Website</b>	www.motoractive.com.au
<b>Email</b>	andrew.spira@motoractive.com.au

### Emergency telephone number

<b>Association / Organisation</b>	MotorActive
<b>Emergency telephone numbers</b>	+61 2 9737 9422 (For General Information Monday to Friday 8:30am to 5:pm)
<b>Other emergency telephone numbers</b>	13 11 26 (In Case of Emergency contact: Poison Information Hotline)

## SECTION 2 HAZARDS IDENTIFICATION

### Classification of the substance or mixture

**HAZARDOUS CHEMICAL. DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.**

#### CHEMWATCH HAZARD RATINGS

	Min	Max
Flammability	4	
Toxicity	1	
Body Contact	2	
Reactivity	1	
Chronic	3	

0 = Minimum  
1 = Low  
2 = Moderate  
3 = High  
4 = Extreme

<b>Poisons Schedule</b>	Not Applicable
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ColorSpec Caliper HT, 300g (ColorSpec Caliper HT)

<b>Classification</b> [1]	Aerosols Category 1, Eye Irritation Category 2A, Reproductive Toxicity Category 1B, Specific target organ toxicity - single exposure Category 3 (narcotic effects)
<b>Legend:</b>	1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

**Label elements**

<b>GHS label elements</b>	
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<b>SIGNAL WORD</b>	<b>DANGER</b>
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**Hazard statement(s)**

<b>H222</b>	Extremely flammable aerosol.
<b>H319</b>	Causes serious eye irritation.
<b>H360</b>	May damage fertility or the unborn child.
<b>H336</b>	May cause drowsiness or dizziness.
<b>AUH044</b>	Risk of explosion if heated under confinement
<b>AUH066</b>	Repeated exposure may cause skin dryness and cracking

**Precautionary statement(s) Prevention**

<b>P201</b>	Obtain special instructions before use.
<b>P210</b>	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
<b>P211</b>	Do not spray on an open flame or other ignition source.
<b>P251</b>	Pressurized container: Do not pierce or burn, even after use.
<b>P271</b>	Use only outdoors or in a well-ventilated area.
<b>P281</b>	Use personal protective equipment as required.
<b>P261</b>	Avoid breathing mist/vapours/spray.
<b>P280</b>	Wear protective gloves/protective clothing/eye protection/face protection.

**Precautionary statement(s) Response**

<b>P308+P313</b>	IF exposed or concerned: Get medical advice/attention.
<b>P305+P351+P338</b>	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
<b>P312</b>	Call a POISON CENTER or doctor/physician if you feel unwell.
<b>P337+P313</b>	If eye irritation persists: Get medical advice/attention.
<b>P304+P340</b>	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.

**Precautionary statement(s) Storage**

<b>P405</b>	Store locked up.
<b>P410+P412</b>	Protect from sunlight. Do not expose to temperatures exceeding 50 °C/122 °F.
<b>P403+P233</b>	Store in a well-ventilated place. Keep container tightly closed.

**Precautionary statement(s) Disposal**

<b>P501</b>	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
67-64-1	10-30	<u>acetone</u>

## ColorSpec Caliper HT, 300g (ColorSpec Caliper HT)

108-65-6	10-30	<u>propylene glycol monomethyl ether acetate, alpha-isomer</u>
1330-20-7	<1	<u>xylene</u>
115-10-6	30-60	<u>dimethyl ether</u>

## SECTION 4 FIRST AID MEASURES

## Description of first aid measures

<b>Eye Contact</b>	<p>If aerosols come in contact with the eyes:</p> <ul style="list-style-type: none"> <li>▶ Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh running water.</li> <li>▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.</li> <li>▶ Transport to hospital or doctor without delay.</li> <li>▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> </ul>
<b>Skin Contact</b>	<p>If solids or aerosol mists are deposited upon the skin:</p> <ul style="list-style-type: none"> <li>▶ Flush skin and hair with running water (and soap if available).</li> <li>▶ Remove any adhering solids with industrial skin cleansing cream.</li> <li>▶ <b>DO NOT use solvents.</b></li> <li>▶ Seek medical attention in the event of irritation.</li> </ul>
<b>Inhalation</b>	<p>If aerosols, fumes or combustion products are inhaled:</p> <ul style="list-style-type: none"> <li>▶ Remove to fresh air.</li> <li>▶ Lay patient down. Keep warm and rested.</li> <li>▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.</li> <li>▶ If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.</li> <li>▶ Transport to hospital, or doctor.</li> </ul>
<b>Ingestion</b>	<ul style="list-style-type: none"> <li>▶ Avoid giving milk or oils.</li> <li>▶ Avoid giving alcohol.</li> <li>▶ Not considered a normal route of entry.</li> </ul>

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

Treat symptomatically.  
for lower alkyl ethers:

## BASIC TREATMENT

- ▶ Establish a patent airway with suction where necessary.
- ▶ Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- ▶ Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- ▶ A low-stimulus environment must be maintained.
- ▶ Monitor and treat, where necessary, for shock.
- ▶ Anticipate and treat, where necessary, for seizures.
- ▶ **DO NOT use emetics.** Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.

## ADVANCED TREATMENT

- ▶ Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- ▶ Positive-pressure ventilation using a bag-valve mask might be of use.
- ▶ Monitor and treat, where necessary, for arrhythmias.
- ▶ Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- ▶ Drug therapy should be considered for pulmonary oedema.
- ▶ Hypotension without signs of hypovolaemia may require vasopressors.
- ▶ Treat seizures with diazepam.
- ▶ Proparacaine hydrochloride should be used to assist eye irrigation.

## EMERGENCY DEPARTMENT

- ▶ Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- ▶ Ethers may produce anion gap acidosis. Hyperventilation and bicarbonate therapy might be indicated.
- ▶ Haemodialysis might be considered in patients with impaired renal function.
- ▶ Consult a toxicologist as necessary.

BRONSTEIN, A.C. and CURRANCE, P.L.

EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

for simple esters:

## BASIC TREATMENT

- Establish a patent airway with suction where necessary.
- Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- Monitor and treat, where necessary, for pulmonary oedema .
- Monitor and treat, where necessary, for shock.
- **DO NOT use emetics.** Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.
- Give activated charcoal.

## ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Treat seizures with diazepam.
- Proparacaine hydrochloride should be used to assist eye irrigation.

## EMERGENCY DEPARTMENT

- Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- Consult a toxicologist as necessary.

BRONSTEIN, A.C. and CURRANCE, P.L. *EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994*

For acute or short term repeated exposures to acetone:

- Symptoms of acetone exposure approximate ethanol intoxication.
- About 20% is expired by the lungs and the rest is metabolised. Alveolar air half-life is about 4 hours following two hour inhalation at levels near the Exposure Standard; in overdose, saturable metabolism and limited clearance, prolong the elimination half-life to 25-30 hours.
- There are no known antidotes and treatment should involve the usual methods of decontamination followed by supportive care.

[Ellenhorn and Barceloux: Medical Toxicology]

Management:

Measurement of serum and urine acetone concentrations may be useful to monitor the severity of ingestion or inhalation.

Inhalation Management:

- Maintain a clear airway, give humidified oxygen and ventilate if necessary.
- If respiratory irritation occurs, assess respiratory function and, if necessary, perform chest X-rays to check for chemical pneumonitis.
- Consider the use of steroids to reduce the inflammatory response.
- Treat pulmonary oedema with PEEP or CPAP ventilation.

Dermal Management:

- Remove any remaining contaminated clothing, place in double sealed, clear bags, label and store in secure area away from patients and staff.
- Irrigate with copious amounts of water.
- An emollient may be required.

Eye Management:

- Irrigate thoroughly with running water or saline for 15 minutes.
- Stain with fluorescein and refer to an ophthalmologist if there is any uptake of the stain.

Oral Management:

- No **GASTRIC LAVAGE OR EMETIC**
- Encourage oral fluids.

Systemic Management:

- Monitor blood glucose and arterial pH.
- Ventilate if respiratory depression occurs.
- If patient unconscious, monitor renal function.
- Symptomatic and supportive care.

The Chemical Incident Management Handbook:

Guy's and St. Thomas' Hospital Trust, 2000

## BIOLOGICAL EXPOSURE INDEX

These represent the determinants observed in specimens collected from a healthy worker exposed at the Exposure Standard (ES or TLV):

Determinant	Sampling Time	Index	Comments
Acetone in urine	End of shift	50 mg/L	NS

NS: Non-specific determinant; also observed after exposure to other material

For acute or short term repeated exposures to xylene:

- Gastro-intestinal absorption is significant with ingestions. For ingestions exceeding 1-2 ml (xylene)/kg, intubation and lavage with cuffed endotracheal tube is recommended. The use of charcoal and cathartics is equivocal.
- Pulmonary absorption is rapid with about 60-65% retained at rest.
- Primary threat to life from ingestion and/or inhalation, is respiratory failure.
- Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen.

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- ▶ Patients with inadequate tidal volumes or poor arterial blood gases ( $pO_2 < 50$  mm Hg or  $pCO_2 > 50$  mm Hg) should be intubated.
- ▶ Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- ▶ A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- ▶ Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.

## BIOLOGICAL EXPOSURE INDEX - BEI

These represent the determinants observed in specimens collected from a healthy worker exposed at the Exposure Standard (ES or TLV):

Determinant	Index	Sampling Time	Comments
Methylhippu-ric acids in urine	1.5 gm/gm creatinine 2 mg/min	End of shift Last 4 hrs of shift	

## SECTION 5 FIREFIGHTING MEASURES

## Extinguishing media

## SMALL FIRE:

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

## LARGE FIRE:

- ▶ Water spray or fog.

## Special hazards arising from the substrate or mixture

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"> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ May be violently or explosively reactive.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ If safe, switch off electrical equipment until vapour fire hazard removed.</li> <li>▶ Use water delivered as a fine spray to control fire and cool adjacent area.</li> <li>▶ <b>DO NOT</b> approach containers suspected to be hot.</li> <li>▶ Cool fire exposed containers with water spray from a protected location.</li> <li>▶ If safe to do so, remove containers from path of fire.</li> <li>▶ Equipment should be thoroughly decontaminated after use.</li> </ul>
Fire/Explosion Hazard	<p>Combustion products include; carbon dioxide (CO<sub>2</sub>)</p> <ul style="list-style-type: none"> <li>▶ Liquid and vapour are highly flammable.</li> <li>▶ Severe fire hazard when exposed to heat or flame.</li> <li>▶ Vapour forms an explosive mixture with air.</li> <li>▶ Severe explosion hazard, in the form of vapour, when exposed to flame or spark.</li> <li>▶ Vapour may travel a considerable distance to source of ignition.</li> <li>▶ Heating may cause expansion or decomposition with violent container rupture.</li> <li>▶ Aerosol cans may explode on exposure to naked flames.</li> <li>▶ Rupturing containers may rocket and scatter burning materials.</li> <li>▶ Hazards may not be restricted to pressure effects.</li> <li>▶ May emit acrid, poisonous or corrosive fumes.</li> <li>▶ On combustion, may emit toxic fumes of carbon monoxide (CO).</li> </ul> <p>, carbon monoxide (CO) nitrogen oxides (NO<sub>x</sub>) other pyrolysis products typical of burning organic material <b>Contains low boiling substance:</b> Closed containers may rupture due to pressure buildup under fire conditions.</p>

## SECTION 6 ACCIDENTAL RELEASE MEASURES

## Personal precautions, protective equipment and emergency procedures

Minor Spills	<ul style="list-style-type: none"> <li>▶ Clean up all spills immediately.</li> <li>▶ Avoid breathing vapours and contact with skin and eyes.</li> <li>▶ Wear protective clothing, impervious gloves and safety glasses.</li> <li>▶ Shut off all possible sources of ignition and increase ventilation.</li> <li>▶ Wipe up.</li> <li>▶ If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated.</li> <li>▶ Undamaged cans should be gathered and stowed safely.</li> </ul>
Major Spills	<ul style="list-style-type: none"> <li>▶ <b>DO NOT exert excessive pressure on valve; DO NOT attempt to operate damaged valve.</b></li> <li>▶ Clear area of personnel and move upwind.</li> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> </ul>

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- ▶ May be violently or explosively reactive.
- ▶ Wear breathing apparatus plus protective gloves.
- ▶ Prevent, by any means available, spillage from entering drains or water courses
- ▶ No smoking, naked lights or ignition sources.
- ▶ Increase ventilation.
- ▶ Stop leak if safe to do so.
- ▶ Water spray or fog may be used to disperse / absorb vapour.
- ▶ Absorb or cover spill with sand, earth, inert materials or vermiculite.
- ▶ If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated.
- ▶ Undamaged cans should be gathered and stowed safely.
- ▶ Collect residues and seal in labelled drums for disposal.
- ▶ Remove leaking cylinders to a safe place if possible.
- ▶ Release pressure under safe, controlled conditions by opening the valve.

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

## SECTION 7 HANDLING AND STORAGE

### Precautions for safe handling

<b>Safe handling</b>	<ul style="list-style-type: none"> <li>▶ <b>DO NOT allow clothing wet with material to stay in contact with skin</b></li> <li>▶ Avoid all personal contact, including inhalation.</li> <li>▶ Wear protective clothing when risk of exposure occurs.</li> <li>▶ Use in a well-ventilated area.</li> <li>▶ Prevent concentration in hollows and sumps.</li> <li>▶ <b>DO NOT enter confined spaces until atmosphere has been checked.</b></li> <li>▶ Avoid smoking, naked lights or ignition sources.</li> <li>▶ Avoid contact with incompatible materials.</li> <li>▶ <b>When handling, DO NOT eat, drink or smoke.</b></li> <li>▶ <b>DO NOT incinerate or puncture aerosol cans.</b></li> <li>▶ <b>DO NOT spray directly on humans, exposed food or food utensils.</b></li> <li>▶ Avoid physical damage to containers.</li> <li>▶ Always wash hands with soap and water after handling.</li> <li>▶ Work clothes should be laundered separately.</li> <li>▶ Use good occupational work practice.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.</li> </ul>
<b>Other information</b>	<ul style="list-style-type: none"> <li>▶ Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can</li> <li>▶ Store in original containers in approved flammable liquid storage area.</li> <li>▶ <b>DO NOT store in pits, depressions, basements or areas where vapours may be trapped.</b></li> <li>▶ No smoking, naked lights, heat or ignition sources.</li> <li>▶ Keep containers securely sealed. Contents under pressure.</li> <li>▶ Store away from incompatible materials.</li> <li>▶ Store in a cool, dry, well ventilated area.</li> <li>▶ Avoid storage at temperatures higher than 40 deg C.</li> <li>▶ Store in an upright position.</li> <li>▶ Protect containers against physical damage.</li> <li>▶ Check regularly for spills and leaks.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> </ul>

### Conditions for safe storage, including any incompatibilities

<b>Suitable container</b>	<ul style="list-style-type: none"> <li>▶ Aerosol dispenser.</li> <li>▶ Check that containers are clearly labelled.</li> </ul>
<b>Storage incompatibility</b>	<ul style="list-style-type: none"> <li>▶ Avoid reaction with oxidising agents</li> </ul>

## 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	acetone	Acetone	1185 mg/m <sup>3</sup> / 500 ppm	2375 mg/m <sup>3</sup> / 1000 ppm	Not Available	Not Available

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Australia Exposure Standards	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxy-2-propanol acetate	274 mg/m <sup>3</sup> / 50 ppm	548 mg/m <sup>3</sup> / 100 ppm	Not Available	Sk
Australia Exposure Standards	xylene	Xylene (o-, m-, p-isomers)	350 mg/m <sup>3</sup> / 80 ppm	655 mg/m <sup>3</sup> / 150 ppm	Not Available	Not Available
Australia Exposure Standards	dimethyl ether	Dimethyl ether	760 mg/m <sup>3</sup> / 400 ppm	950 mg/m <sup>3</sup> / 500 ppm	Not Available	Not Available

**EMERGENCY LIMITS**

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
acetone	Acetone	Not Available	Not Available	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Propylene glycol monomethyl ether acetate, alpha-isomer; (1-Methoxypropyl-2-acetate)	Not Available	Not Available	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Propylene glycol monomethyl ether acetate, beta-isomer; (2-Methoxypropyl-1-acetate)	Not Available	Not Available	Not Available
xylene	Xylenes	Not Available	Not Available	Not Available
dimethyl ether	Methyl ether; (Dimethyl ether)	1,000 ppm	1000 ppm	7200 ppm

Ingredient	Original IDLH	Revised IDLH
acetone	20,000 ppm	2,500 [LEL] ppm
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available
xylene	1,000 ppm	900 ppm
dimethyl ether	Not Available	Not Available

**MATERIAL DATA**

for dimethyl ether:

The no-effect-level for dimethyl ether is somewhere between 2000 ppm (rabbits) and 50,000 ppm (humans) with possible cardiac sensitisation occurring around 200,000 ppm (dogs). The AIHA has adopted a safety factor of 100 in respect to the 50,000 ppm level in its recommendation for a workplace environmental exposure level (WEEL) which is thought to protect against both narcotic and sensitising effects. This level is consistent with the TLV-TWA of 400 ppm for diethyl ether and should be easily achievable using current technologies. The use of the traditionally allowable excursion of 1.25 to the level of 6.25 ppm is felt to be more than adequate as an upper safe limit of exposure.

Human data:

50,000 ppm (12 mins): Feelings of mild intoxication.

75,000 ppm (12 mins): As above plus slight lack of attenuation.

82,000 ppm (12 mins): Some incoordination, slight blurring of vision

(30 mins): As above plus analgesia of the face and rushing of blood to the face.

100,000 ppm (10-20 mins): Narcotic symptoms; (64 mins): Sickness (assumed to be nausea)

144,000 ppm (36 mins): Unconsciousness

Odour Threshold Value: 3.6 ppm (detection), 699 ppm (recognition)

Saturation vapour concentration: 237000 ppm @ 20 C

NOTE: Detector tubes measuring in excess of 40 ppm, are available.

Exposure at or below the recommended TLV-TWA is thought to protect the worker against mild irritation associated with brief exposures and the bioaccumulation, chronic irritation of the respiratory tract and headaches associated with long-term acetone exposures. The NIOSH REL-TWA is substantially lower and has taken into account slight irritation experienced by volunteer subjects at 300 ppm. Mild irritation to acclimatised workers begins at about 750 ppm - unacclimatised subjects will experience irritation at about 350-500 ppm but acclimatisation can occur rapidly. Disagreement between the peak bodies is based largely on the view by ACGIH that widespread use of acetone, without evidence of significant adverse health effects at higher concentrations, allows acceptance of a higher limit.

Half-life of acetone in blood is 3 hours which means that no adjustment for shift-length has to be made with reference to the standard 8 hour/day, 40 hours per week because body clearance occurs within any shift with low potential for accumulation.

A STEL has been established to prevent excursions of acetone vapours that could cause depression of the central nervous system.

Odour Safety Factor(OSF)

OSF=38 (ACETONE)

for propylene glycol monomethyl ether acetate (PGMEA)

Saturated vapour concentration: 4868 ppm at 20 C.

A two-week inhalation study found nasal effects to the nasal mucosa in animals at concentrations up to 3000 ppm. Differences in the teratogenic potential of the alpha (commercial grade) and beta isomers of PGMEA may be explained by the formation of different metabolites. The beta-isomer is thought to be oxidised to methoxypropionic acid, a homologue to methoxyacetic acid which is a known teratogen. The alpha- form is conjugated and excreted. PGMEA mixture (containing 2% to 5% beta isomer) is a mild skin and eye irritant, produces mild central nervous system effects in animals at 3000 ppm and produces mild CNS impairment and upper respiratory tract and eye irritation in humans at 1000 ppm. In rats exposed to 3000 ppm PGMEA produced slight foetotoxic effects (delayed sternbral ossification) - no effects on foetal development were seen in rabbits exposed at 3000 ppm.

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for xylenes:

IDLH Level: 900 ppm

Odour Threshold Value: 20 ppm (detection), 40 ppm (recognition)

NOTE: Detector tubes for o-xylene, measuring in excess of 10 ppm, are available commercially. (m-xylene and p-xylene give almost the same response).

Xylene vapour is an irritant to the eyes, mucous membranes and skin and causes narcosis at high concentrations. Exposure to doses sufficiently high to produce intoxication and unconsciousness also produces transient liver and kidney toxicity. Neurologic impairment is NOT evident amongst volunteers inhaling up to 400 ppm though complaints of ocular and upper respiratory tract irritation occur at 200 ppm for 3 to 5 minutes.

Exposure to xylene at or below the recommended TLV-TWA and STEL is thought to minimise the risk of irritant effects and to produce neither significant narcosis or chronic injury. An earlier skin notation was deleted because percutaneous absorption is gradual and protracted and does not substantially contribute to the dose received by inhalation.

Odour Safety Factor(OSF)

OSF=4 (XYLENE)

## Exposure controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.

The basic types of engineering controls are:

Process controls which involve changing the way a job activity or process is done to reduce the risk.

Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.

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

General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection.

Provide adequate ventilation in warehouse or closed storage areas.

Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Speed:
aerosols, (released at low velocity into zone of active generation)	0.5-1 m/s
direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

## Personal protection



## Eye and face protection

- ▶ Safety glasses with side shields.
- ▶ Chemical goggles.
- ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of 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]

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	<ul style="list-style-type: none"> <li>▶ Close fitting gas tight goggles</li> </ul> <p><b>DO NOT wear contact lenses.</b></p> <ul style="list-style-type: none"> <li>▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lens or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]</li> </ul>
<b>Skin protection</b>	See Hand protection below
<b>Hands/feet protection</b>	<p>For esters:</p> <ul style="list-style-type: none"> <li>▶ Do NOT use natural rubber, butyl rubber, EPDM or polystyrene-containing materials.</li> <li>▶ No special equipment needed when handling small quantities.</li> <li>▶ <b>OTHERWISE:</b></li> <li>▶ For potentially moderate exposures:</li> <li>▶ Wear general protective gloves, eg. light weight rubber gloves.</li> <li>▶ For potentially heavy exposures:</li> <li>▶ Wear chemical protective gloves, eg. PVC. and safety footwear.</li> </ul>
<b>Body protection</b>	See Other protection below
<b>Other protection</b>	<p>No special equipment needed when handling small quantities.</p> <p><b>OTHERWISE:</b></p> <ul style="list-style-type: none"> <li>▶ Overalls.</li> <li>▶ Skin cleansing cream.</li> <li>▶ Eyewash unit.</li> <li>▶ Do not spray on hot surfaces.</li> <li>▶ The clothing worn by process operators insulated from earth may develop static charges far higher (up to 100 times) than the minimum ignition energies for various flammable gas-air mixtures. This holds true for a wide range of clothing materials including cotton.</li> <li>▶ Avoid dangerous levels of charge by ensuring a low resistivity of the surface material worn outermost.</li> </ul> <p>BREITHERICK: Handbook of Reactive Chemical Hazards.</p>
<b>Thermal hazards</b>	Not Available

## Recommended material(s)

## GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

**"Forsberg Clothing Performance Index".**

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

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Material	CPI
BUTYL	A
NEOPRENE	B

\* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

## Respiratory protection

Type AX 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, 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	AX-AUS / Class 1	-	AX-PAPR-AUS / Class 1
up to 50 x ES	Air-line*	-	-
up to 100 x ES	-	AX-3	-
100+ x ES	-	Air-line**	-

\* - Continuous-flow; \*\* - Continuous-flow or positive pressure demand  
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)

## SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

## Information on basic physical and chemical properties

<b>Appearance</b>	Supplied as an aerosol pack. Contents under <b>PRESSURE</b> . Contains highly flammable ether propellant. 28pge Clear thin flammable liquid with a strong solvent odour.		
<b>Physical state</b>	Liquid	<b>Relative density</b>	0.75-0.85

Continued...

		(Water = 1)	
<b>Odour</b>	Not Available	<b>Partition coefficient n-octanol / water</b>	Not Available
<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	Not Available
<b>pH (as supplied)</b>	Not Applicable	<b>Decomposition temperature</b>	Not Available
<b>Melting point / freezing point (°C)</b>	Not Available	<b>Viscosity (cSt)</b>	Not Available
<b>Initial boiling point and boiling range (°C)</b>	-25 to 145	<b>Molecular weight (g/mol)</b>	Not Applicable
<b>Flash point (°C)</b>	-41 (OC)	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	Not Available	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	HIGHLY FLAMMABLE.	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	19	<b>Surface Tension (dyn/cm or mN/m)</b>	Not Available
<b>Lower Explosive Limit (%)</b>	1	<b>Volatile Component (%vol)</b>	Not Available
<b>Vapour pressure (kPa)</b>	510 @20C	<b>Gas group</b>	Not Available
<b>Solubility in water (g/L)</b>	Not Available	<b>pH as a solution (1%)</b>	Not Applicable
<b>Vapour density (Air = 1)</b>	Not Available	<b>VOC g/L</b>	Not Available

## SECTION 10 STABILITY AND REACTIVITY

<b>Reactivity</b>	See section 7
<b>Chemical stability</b>	<ul style="list-style-type: none"> <li>▶ Elevated temperatures.</li> <li>▶ Presence of open flame.</li> <li>▶ Product is considered stable.</li> <li>▶ Hazardous polymerisation will not occur.</li> </ul>
<b>Possibility of hazardous reactions</b>	See section 7
<b>Conditions to avoid</b>	See section 7
<b>Incompatible materials</b>	See section 7
<b>Hazardous decomposition products</b>	See section 5

## SECTION 11 TOXICOLOGICAL INFORMATION

### Information on toxicological effects

	<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>
<b>Inhaled</b>	<p>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>The acute toxicity of inhaled alkylbenzenes is best described by central nervous system depression. As a rule, these compounds may also act as general anaesthetics.</p> <p>Systemic poisoning produced by general anaesthesia is characterised by lightheadedness, nervousness, apprehension, euphoria, confusion, dizziness, drowsiness, tinnitus, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness and respiratory depression and arrest. Cardiac arrest may result from cardiovascular collapse. Bradycardia, and hypotension may also be produced.</p> <p>Inhaled alkylbenzene vapours cause death in animals at air levels that are relatively similar (typically LC50s are in the range 5000 -8000 ppm for 4 to 8 hour exposures). It is likely that acute inhalation exposure to alkylbenzenes resembles that to general anaesthetics.</p>

Continued...

Alkylbenzenes are not generally toxic other than at high levels of exposure. This may be because their metabolites have a low order of toxicity and are easily excreted. There is little or no evidence to suggest that metabolic pathways can become saturated leading to spillover to alternate pathways. Nor is there evidence that toxic reactive intermediates, which may produce subsequent toxic or mutagenic effects, are formed

Common, generalised symptoms associated with toxic gas inhalation include:

- ▶ central nervous system effects such as depression, headache, confusion, dizziness, progressive stupor, coma and seizures;
- ▶ respiratory system complications may include acute pulmonary oedema, dyspnoea, stridor, tachypnoea, bronchospasm, wheezing and other reactive airway symptoms, and respiratory arrest;
- ▶ cardiovascular effects may include cardiovascular collapse, arrhythmias and cardiac arrest;
- ▶ gastrointestinal effects may also be present and may include mucous membrane irritation, nausea and vomiting (sometimes bloody), and abdominal pain.

Ethers produce narcosis following inhalation.

Inhalation of lower alkyl ethers may result in central nervous system depression or stimulation, intoxication, headache, dizziness, weakness, blurred vision, seizures and possible coma. Cardiovascular involvement may produce hypotension, bradycardia and cardiovascular collapse, whilst respiratory symptoms might include irritation of nose and throat, cough, laryngeal spasm, pharyngitis, irregular respiration, depression, pulmonary oedema and respiratory arrest. Nausea, vomiting and salivation might also indicate overexposure.

Convulsions, respiratory distress or paralysis, asphyxia, pneumonitis, and unconsciousness are all serious manifestations of poisoning. Fatalities have been reported. Kidney and liver damage with interstitial cystitis may result from massive exposures.

Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure.

Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination

Mice exposed at up to 3000 ppm PGMEA 6 hr/day for a total of 9 days during an 11-day period showed no pronounced effect on the weights of liver, kidneys, heart, spleen, thymus or testes. Histopathological examination revealed degeneration of the olfactory epithelium in mice exposed at 300 ppm for the same time. Rats, similarly failed to show changes in internal organs and did not show olfactory epithelium degeneration until 3000 ppm. The no-effect level in rats was 1000 ppm.

**WARNING: Intentional misuse by concentrating/inhaling contents may be lethal.**

Systemic effects of acetone inhalation exposure include central nervous system depression, light-headedness, incoherent speech, ataxia, stupor, hypotension, tachycardia, metabolic acidosis, hyperglycaemia and ketosis. Rarely, convulsions and tubular necrosis may be evident. Other symptoms of exposure may include restlessness, headache, vomiting, low blood-pressure and rapid and irregular pulse, eye and throat irritation, weakness of the legs and dizziness. Inhalation of high concentrations may produce dryness of the mouth and throat, nausea, uncoordinated movement, loss of coordinated speech, drowsiness and, in severe cases, coma. Inhalation of acetone vapours over long periods causes irritation of the respiratory tract, coughing and headache. Rats exposed to 52200 ppm vapour for 1 hour showed clear signs of narcosis; fatalities occurred at 126600 ppm.

### Ingestion

Accidental ingestion of the material may be damaging to the health of the individual.

Not normally a hazard due to physical form of product.

Considered an unlikely route of entry in commercial/industrial environments

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.

Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).

Ingestion of alkyl ethers may produce symptoms similar to those produced following inhalation.

### Skin Contact

Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.

Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.

Repeated application of commercial grade PGMEA to the skin of rabbits for 2-weeks caused slight redness and very slight exfoliation.

Spray mist may produce discomfort

Alkyl ethers may defat and dehydrate the skin producing dermatoses. Absorption may produce headache, dizziness, and central nervous system depression.

Open cuts, abraded or irritated skin should not be exposed to this material

The material may produce mild skin irritation; limited evidence or practical experience suggests, that the material either:

- ▶ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or
- ▶ produces significant, but mild, 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 (non allergic). 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.

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<p><b>Eye</b></p>	<p>Direct contact with the eye may not cause irritation because of the extreme volatility of the gas; however concentrated atmospheres may produce irritation after brief exposures..</p> <p>Eye contact with alkyl ethers (vapours or liquid) may produce irritation, redness and lachrymation.</p> <p>Undiluted propylene glycol monomethyl ether acetate (PGMEA) causes moderate discomfort, slight conjunctival redness and slight corneal injury in rabbits</p> <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>
<p><b>Chronic</b></p>	<p>There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of:</p> <ul style="list-style-type: none"> <li>- clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.</li> </ul> <p>Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>Repeated exposure to higher concentrations of propylene glycol monomethyl ether acetate (PGMEA) (1000 ppm and above) causes mild liver and kidney damage in animals.</p> <p>A minor component, 2-methoxy-1-propyl acetate (the beta-isomer) produced birth defects on inhalation exposure of pregnant rabbits at 545 ppm, but not at 145 or 36 ppm; maternal and embryo/foetal toxicity on inhalation exposure of pregnant rats at 2710 ppm, but not at 545 or 110 ppm; and no adverse effects on dermal exposure of pregnant rabbits at applied dosages of 1000 and 2000 mg/kg of body weight per day during the critical period or embryo/foetal development. In a further study, no developmental effects were seen following exposure of pregnant rats at air concentrations of commercial propylene glycol monomethyl ether acetate (containing 3-5% of the minor component) up to 4000 ppm; slight maternal effects were seen at 5000 ppm and greater.</p> <p>Exposure of pregnant rats and rabbits to the parent glycol ether, propylene glycol monomethyl ether which contained comparable amounts of the primary isomer, 2-methoxy-1-propanol, did not produce teratogenic effects at concentrations up to 3000 ppm. Foetotoxic effects were seen in rat foetuses but not in rabbit foetuses at this concentration and maternal toxicity was noted in both species at this concentration</p> <p>Principal route of occupational exposure to the gas is by inhalation.</p> <p>Chronic exposure to alkyl ethers may result in loss of appetite, excessive thirst, fatigue, and weight loss</p> <p>Prolonged or repeated contact with xylenes may cause defatting dermatitis with drying and cracking. Chronic inhalation of xylenes has been associated with central nervous system effects, loss of appetite, nausea, ringing in the ears, irritability, thirst anaemia, mucosal bleeding, enlarged liver and hyperplasia. Exposure may produce kidney and liver damage. In chronic occupational exposure, xylene (usually mixed with other solvents) has produced irreversible damage to the central nervous system and ototoxicity (damages hearing and increases sensitivity to noise), probably due to neurotoxic mechanisms.</p> <p>Industrial workers exposed to xylene with a maximum level of ethyl benzene of 0.06 mg/l (14 ppm) reported headaches and irritability and tired quickly. Functional nervous system disturbances were found in some workers employed for over 7 years whilst other workers had enlarged livers.</p> <p>Xylene has been classed as a developmental toxin in some jurisdictions.</p> <p>Small excess risks of spontaneous abortion and congenital malformation were reported amongst women exposed to xylene in the first trimester of pregnancy. In all cases, however, the women were also been exposed to other substances. Evaluation of workers chronically exposed to xylene has demonstrated lack of genotoxicity. Exposure to xylene has been associated with increased risks of haemopoietic malignancies but, again, simultaneous exposure to other substances (including benzene) complicates the picture. A long-term gavage study to mixed xylenes (containing 17% ethyl benzene) found no evidence of carcinogenic activity in rats and mice of either sex.</p> <p>Workers exposed to 700 ppm acetone for 3 hours/day for 7-15 years showed inflammation of the respiratory tract, stomach and duodenum, attacks of giddiness and loss of strength. Exposure to acetone may enhance liver toxicity of chlorinated solvents.</p>

<p><b>ColorSpec Caliper HT, 300g (ColorSpec Caliper HT)</b></p>	<p><b>TOXICITY</b></p> <p>Not Available</p>	<p><b>IRRITATION</b></p> <p>Not Available</p>
<p><b>acetone</b></p>	<p><b>TOXICITY</b></p> <p>Dermal (rabbit) LD50: 20000 mg/kg<sup>[2]</sup></p> <p>Inhalation (rat) LC50: 50.1 mg/L/8 hr<sup>[2]</sup></p> <p>Oral (rat) LD50: 5800 mg/kgE<sup>[2]</sup></p>	<p><b>IRRITATION</b></p> <p>Eye (human): 500 ppm - irritant</p> <p>Eye (rabbit): 20mg/24hr - moderate</p> <p>Eye (rabbit): 3.95 mg - SEVERE</p> <p>Skin (rabbit): 500 mg/24hr - mild</p> <p>Skin (rabbit):395mg (open) - mild</p>
<p><b>propylene glycol monomethyl ether acetate, alpha-isomer</b></p>	<p><b>TOXICITY</b></p> <p>dermal (rat) LD50: &gt;2000 mg/kg<sup>[1]</sup></p>	<p><b>IRRITATION</b></p> <p>* [CCINFO]</p>

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	Inhalation (rat) LC50: 4345 ppm/6h <sup>[2]</sup>	Nil reported
	Oral (rat) LD50: >14.1 ml <sup>[1]</sup>	
<b>xylene</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >1700 mg/kg <sup>[2]</sup>	Eye (human): 200 ppm irritant
	Inhalation (rat) LC50: 5000 ppm/4h <sup>[2]</sup>	Eye (rabbit): 5 mg/24h SEVERE
	Oral (rat) LD50: 4300 mg/kg <sup>[2]</sup>	Eye (rabbit): 87 mg mild
		Skin (rabbit):500 mg/24h moderate
<b>dimethyl ether</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Inhalation (rat) LC50: 309 mg/L/4H <sup>[2]</sup>	Nil reported
<b>Legend:</b>	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

<b>ColorSpec Caliper HT, 300g (ColorSpec Caliper HT)</b>	<p>for propylene glycol ethers (PGEs):</p> <p>Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM).</p> <p>Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids.</p> <p>Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).</p> <p>This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.</p> <p>Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body.</p> <p>As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces.</p> <p>As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from &gt;3,000 mg/kg (PnB) to &gt;5,000 mg/kg (DPMA). Dermal LD50s are all &gt; 2,000 mg/kg (PnB, &amp; DPnB; where no deaths occurred), and ranging up to &gt;15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m<sup>3</sup> for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is &gt;2,040 mg/m<sup>3</sup>. For PnB, the 4-hour LC50 was &gt;651 ppm (&gt;3,412 mg/m<sup>3</sup>), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating</p> <p>None are skin sensitisers.</p> <p>In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested).</p> <p>Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m<sup>3</sup> (600 ppm) for PnB and 2,010 mg/m<sup>3</sup> (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m<sup>3</sup> (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m<sup>3</sup> (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members.</p> <p>One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m<sup>3</sup>) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m<sup>3</sup>). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m<sup>3</sup>), with decreased body weights occurring at 3000 ppm (11058 mg/m<sup>3</sup>). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects</p>
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## ColorSpec Caliper HT, 300g (ColorSpec Caliper HT)

were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health.

In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity.

The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.

A BASF report (in ECETOC ) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects.

The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.I]

for acetone:

The acute toxicity of acetone is low. Acetone is not a skin irritant or sensitiser but is a defatting agent to the skin. Acetone is an eye irritant. The subchronic toxicity of acetone has been examined in mice and rats that were administered acetone in the drinking water and again in rats treated by oral gavage. Acetone-induced increases in relative kidney weight changes were observed in male and female rats used in the oral 13-week study. Acetone treatment caused increases in the relative liver weight in male and female rats that were not associated with histopathologic effects and the effects may have been associated with microsomal enzyme induction. Haematologic effects consistent with macrocytic anaemia were also noted in male rats along with hyperpigmentation in the spleen. The most notable findings in the mice were increased liver and decreased spleen weights. Overall, the no-observed-effect-levels in the drinking water study were 1% for male rats (900 mg/kg/d) and male mice (2258 mg/kg/d), 2% for female mice (5945 mg/kg/d), and 5% for female rats (3100 mg/kg/d). For developmental effects, a statistically significant reduction in foetal weight, and a slight, but statistically significant increase in the percent incidence of later resorptions were seen in mice at 15,665 mg/m<sup>3</sup> and in rats at 26,100 mg/m<sup>3</sup>. The no-observable-effect level for developmental toxicity was determined to be 5220 mg/m<sup>3</sup> for both rats and mice. Teratogenic effects were not observed in rats and mice tested at 26,110 and 15,665 mg/m<sup>3</sup>, respectively. Lifetime dermal carcinogenicity studies in mice treated with up to 0.2 mL of acetone did not reveal any increase in organ tumor incidence relative to untreated control animals.

The scientific literature contains many different studies that have measured either the neurobehavioural performance or neurophysiological response of humans exposed to acetone. Effect levels ranging from about 600 to greater than 2375 mg/m<sup>3</sup> have been reported. Neurobehavioral studies with acetone-exposed employees have recently shown that 8-hr exposures in excess of 2375 mg/m<sup>3</sup> were not associated with any dose-related changes in response time, vigilance, or digit span scores. Clinical case studies, controlled human volunteer studies, animal research, and occupational field evaluations all indicate that the NOAEL for this effect is 2375 mg/m<sup>3</sup> or greater.

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.

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**ACETONE****PROPYLENE GLYCOL  
MONOMETHYL ETHER  
ACETATE, ALPHA-  
ISOMER**

for propylene glycol ethers (PGEs):

Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM).

Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the

developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids.

Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).

This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.

Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body.

As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces.

As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m<sup>3</sup> for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m<sup>3</sup>. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m<sup>3</sup>), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating. None are skin sensitizers.

In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested).

Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m<sup>3</sup> (600 ppm) for PnB and 2,010 mg/m<sup>3</sup> (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m<sup>3</sup> (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m<sup>3</sup> (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members. One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m<sup>3</sup>) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m<sup>3</sup>). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m<sup>3</sup>), with decreased body weights occurring at 3000 ppm (11058 mg/m<sup>3</sup>). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health.

In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity.

The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.

A BASF report (in ECETOC ) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects.

The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.I]

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**XYLENE**

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

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis

ColorSpec Caliper HT, 300g (ColorSpec Caliper HT)

(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. 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.  
Reproductive effector in rats

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: **X** – Data available but does not fill the criteria for classification  
**✓** – Data required to make classification available  
**⊖** – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
acetone	EC50	384	Crustacea	97.013mg/L	3
acetone	EC50	48	Crustacea	>100mg/L	4
acetone	EC50	96	Algae or other aquatic plants	20.565mg/L	4
acetone	LC50	96	Fish	>100mg/L	4
acetone	NOEC	96	Algae or other aquatic plants	4.950mg/L	4
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	96	Algae or other aquatic plants	9.337mg/L	3
propylene glycol monomethyl ether acetate, alpha-isomer	LC50	96	Fish	100mg/L	1
propylene glycol monomethyl ether acetate, alpha-isomer	NOEC	336	Fish	47.5mg/L	2
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	48	Crustacea	373mg/L	2
propylene glycol monomethyl ether acetate, alpha-isomer	EC50	504	Crustacea	>100mg/L	2
xylene	EC50	24	Crustacea	0.711mg/L	4
xylene	LC50	96	Fish	0.0013404mg/L	4
xylene	EC50	48	Crustacea	>3.4mg/L	2
xylene	EC50	72	Algae or other aquatic plants	4.6mg/L	2
xylene	NOEC	73	Algae or other aquatic plants	0.44mg/L	2
dimethyl ether	NOEC	48	Crustacea	>4000mg/L	1
dimethyl ether	EC50	384	Crustacea	46.027mg/L	3
dimethyl ether	LC50	96	Fish	200.592mg/L	3
dimethyl ether	EC50	48	Crustacea	>4400.0mg/L	2
dimethyl ether	EC50	96	Algae or other aquatic plants	154.917mg/L	2

Legend:

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

for propylene glycol ethers:

Environmental fate:

Continued...

Most are liquids at room temperature and all are water-soluble.

Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM)

Environmental fate: Log octanol-water partition coefficients (log Kow's) range from 0.309 for TPM to 1.523 for DPnB. Calculated BCFs range from 1.47 for DPnB to 3.16 for DPMA and TPM, indicating low bioaccumulation. Henry's Law Constants, which indicate propensity to partition from water to air, are low for all category members, ranging from  $5.7 \times 10^{-9}$  atm-m<sup>3</sup>/mole for TPM to  $2.7 \times 10^{-9}$  atm-m<sup>3</sup>/mole for PnB. Fugacity modeling indicates that most propylene glycol ethers are likely to partition roughly equally into the soil and water compartments in the environment with small to negligible amounts remaining in other environmental compartments (air, sediment, and aquatic biota). Propylene glycol ethers are unlikely to persist in the environment. Once in air, the half-life of the category members due to direct reactions with photochemically generated hydroxyl radicals, range from 2.0 hours for TPM to 4.6 hours for PnB. In water, most members of this family are "readily biodegradable" under aerobic conditions. (DPMA degraded within 28 days (and within the specified 10-day window) but only using pre-adapted or "acclimated" inoculum.). In soil, biodegradation is rapid for PM and PMA.

#### Ecotoxicity:

Acute aquatic toxicity testing indicates low toxicity for both ethers and acetates. For ethers, effect concentrations are > 500 mg/L. For acetates, effect concentrations are > 151 mg/L.

Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. For example, there is an increase in toxicity as alkylation of the naphthalene structure increases. The order of most toxic to least in a study using grass shrimp (*Palaemonetes pugio*) and brown shrimp (*Penaeus aztecus*) was dimethylnaphthalenes > methylnaphthalenes > naphthalenes.

Studies conclude that the toxicity of an oil appears to be a function of its di-aromatic and tri-aromatic hydrocarbons, which includes three-ring hydrocarbons such as phenanthrene.

The heavier (4-, 5-, and 6-ring) PAHs are more persistent than the lighter (2- and 3-ring) PAHs and tend to have greater carcinogenic and other chronic impact potential. PAHs in general are more frequently associated with chronic risks. These risks include cancer and often are the result of exposures to complex mixtures of chronic-risk aromatics (such as PAHs, alkyl PAHs, benzenes, and alkyl benzenes), rather than exposures to low levels of a single compound.

Anthracene is a phototoxic PAH. UV light greatly increases the toxicity of anthracene to bluegill sunfish. Benchmarks developed in the absence of UV light may be under-protective, and biological resources in strong sunlight are at more risk than those that are not.

For xylenes :

log Koc : 2.05-3.08

Koc : 25.4-204

Half-life (hr) air : 0.24-42

Half-life (hr) H<sub>2</sub>O surface water : 24-672

Half-life (hr) H<sub>2</sub>O ground : 336-8640

Half-life (hr) soil : 52-672

Henry's Pa m<sup>3</sup>/mol: 637-879

Henry's atm m<sup>3</sup>/mol: 7.68E-03

BOD 5 if unstated: 1.4,1%

COD : 2.56,13%

ThOD : 3.125

BCF : 23

log BCF : 1.17-2.41

#### Environmental Fate

**Terrestrial fate:** Measured Koc values of 166 and 182, indicate that 3-xylene is expected to have moderate mobility in soil. Volatilisation of p-xylene is expected to be important from moist soil surfaces given a measured Henry's Law constant of  $7.18 \times 10^{-3}$  atm-cu m/mole. The potential for volatilisation of 3-xylene from dry soil surfaces may exist based on a measured vapor pressure of 8.29 mm Hg. p-Xylene may be degraded during its passage through soil). The extent of the degradation is expected to depend on its concentration, residence time in the soil, the nature of the soil, and whether resident microbial populations have been acclimated. p-Xylene, present in soil samples contaminated with jet fuel, was completely degraded aerobically within 5 days. In aquifer studies under anaerobic conditions, p-xylene was degraded, usually within several weeks, with the production of 3-methylbenzylfumaric acid, 3-methylbenzylsuccinic acid, 3-methylbenzoate, and 3-methylbenzaldehyde as metabolites.

**Aquatic fate:** Koc values indicate that p-xylene may adsorb to suspended solids and sediment in water. p-Xylene is expected to volatilise from water surfaces based on the measured Henry's Law constant. Estimated volatilisation half-lives for a model river and model lake are 3 hours and 4 days, respectively. BCF values of 14.8, 23.4, and 6, measured in goldfish, eels, and clams, respectively, indicate that bioconcentration in aquatic organisms is low. p-Xylene in water with added humic substances was 50% degraded following 3 hours irradiation suggesting that indirect photooxidation in the presence of humic acids may play an important role in the abiotic degradation of p-xylene. Although p-xylene is biodegradable and has been observed to degrade in pond water, there are insufficient data to assess the rate of this process in surface waters. p-Xylene has been observed to degrade in anaerobic and aerobic groundwater in several studies; however, it is known to persist for many years in groundwater, at least at sites where the concentration might have been quite high.

#### Atmospheric fate:

Most xylenes released to the environment will occur in the atmosphere and volatilisation is the dominant environmental fate process. In the ambient atmosphere, xylenes are expected to exist solely in the vapour phase. Xylenes are degraded in the atmosphere primarily by reaction with photochemically-produced hydroxyl radicals, with an estimated atmospheric lifetime of about 0.5 to 2 days. Xylenes' susceptibility to photochemical oxidation in the troposphere is to the extent that they may contribute to photochemical smog formation.

According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere and from its vapour pressure, p-xylene, is expected to exist solely as a vapour in the ambient atmosphere. Vapour-phase p-xylene is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be about 16 hours. A half-life of 1.0 hr in summer and 10 hr in winter was measured for the reaction of p-xylene with photochemically-produced hydroxyl radicals. p-Xylene has a moderately high photochemical reactivity under smog conditions, higher than the other xylene isomers, with loss rates varying from 9-42% per hr. The photooxidation of p-xylene results in the production of carbon monoxide, formaldehyde, glyoxal, methylglyoxal, 3-methylbenzyl nitrate, m-tolualdehyde, 4-nitro-3-xylene, 5-nitro-3-xylene, 2,6-dimethyl-p-benzoquinone, 2,4-dimethylphenol, 6-nitro-2,4-dimethylphenol, 2,6-dimethylphenol, and 4-nitro-2,6-dimethylphenol.

#### Ecotoxicity:

for xylenes

Fish LC50 (96 h) Pimephales promelas 13.4 mg/l; Oncorhynchus mykiss 8.05 mg/l; Lepomis macrochirus 16.1 mg/l (all flow through values); Pimephales promelas 26.7 (static)

Daphnia EC50 (96 h): 3.83 mg/l

Photobacterium phosphoreum EC50 (24 h): 0.0084 mg/l

Gammarus lacustris LC50 (48 h): 0.6 mg/l

For ketones:

Ketones, unless they are alpha, beta--unsaturated ketones, can be considered as narcosis or baseline toxicity compounds

Hydrolysis may also involve the addition of water to ketones to yield ketals under mild acid conditions. However, this addition of water is thermodynamically favorable only for low molecular weight ketones. This addition is an equilibrium reaction that is reversible upon a change of water concentration and the reaction ultimately leads to no permanent change in the structure of the ketone substrate. The higher molecular weight ketones do not form stable ketals. Therefore, the ketones are stable to water under ambient environmental conditions

Another possible reaction of ketones in water involves the enolic hydrogen on the carbons bonded to the carbonyl function. Under conditions of high pH (pH greater than 10), the enolic proton is abstracted by base (OH-) forming a carbanion intermediate that may react with other organic substrates (e.g., ketones, esters, aldehydes) containing a center for nucleophilic attack. The reactions, commonly recognized as condensation reactions, produce higher molecular weight products. Under ambient conditions of temperature, pH, and low concentration, these condensation reactions are unfavorable. Based on its reactions in air, it seems likely that ketones undergo photolysis in water. It is probable that ketones will be biodegraded to an appreciable degree by micro-organisms in soil and water. They are unlikely to bioconcentrate or biomagnify.

Most ethers are very resistant to hydrolysis, and the rate of cleavage of the carbon-oxygen bond by abiotic processes is expected to be insignificant. Direct photolysis will not be an important removal process since aliphatic ethers do not absorb light at wavelengths >290 nm

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

for acetone:

log Kow: -0.24

Half-life (hr) air: 312-1896

Half-life (hr) H2O surface water: 20

Henry's atm m3 /mol: 3.67E-05

BOD 5: 0.31-1.76,46-55%

COD: 1.12-2.07

ThOD: 2.2

BCF: 0.69

#### Environmental fate:

Acetone preferentially locates in the air compartment when released to the environment. A substantial amount of acetone can also be found in water, which is consistent with the high water to air partition coefficient and its small, but detectable, presence in rain water, sea water, and lake water samples. Very little acetone is expected to reside in soil, biota, or suspended solids. This is entirely consistent with the physical and chemical properties of acetone and with measurements showing a low propensity for soil absorption and a high preference for moving through the soil and into the ground water. In air, acetone is lost by photolysis and reaction with photochemically produced hydroxyl radicals; the estimated half-life of these combined processes is about 22 days. The relatively long half-life allows acetone to be transported long distances from its emission source.

Acetone is highly soluble and slightly persistent in water, with a half-life of about 20 hours; it is minimally toxic to aquatic life.

Acetone released to soil volatilises although some may leach into the ground where it rapidly biodegrades.

Acetone does not concentrate in the food chain.

Acetone meets the OECD definition of readily biodegradable which requires that the biological oxygen demand (BOD) is at least 70% of the theoretical oxygen demand (THOD) within the 28-day test period

Drinking Water Standard: none available.

Soil Guidelines: none available.

Air Quality Standards: none available.

#### Ecotoxicity:

Testing shows that acetone exhibits a low order of toxicity

Fish LC50: brook trout 6070 mg/l; fathead minnow 15000 mg/l

Bird LC0 (5 day): Japanese quail, ring-neck pheasant 40,000 mg/l

Daphnia magna LC50 (48 h): 15800 mg/l; NOEC 8500 mg/l

Aquatic invertebrate 2100 - 16700 mg/l

Aquatic plant NOEC: 5400-7500 mg/l

Daphnia magna chronic NOEC 1660 mg/l

Acetone vapors were shown to be relatively toxic to two types insects and their eggs. The time to 50% lethality (LT50) was found to be 51.2 hr and 67.9 hr when the flour beetle (*Tribolium confusum*) and the flour moth (*Ephesia kuehniella*) were exposed to an airborne acetone concentration of 61.5 mg/m3. The LT50 values for the eggs were 30-50% lower than for the adult. The direct application of acetone liquid to the body of the insects or surface of the eggs did not, however, cause any mortality.

The ability of acetone to inhibit cell multiplication has been examined in a wide variety of microorganisms. The results have generally indicated mild to minimal toxicity with NOECs greater than 1700 mg/L for exposures lasting from 6 hr to 4 days. Longer exposure periods of 7 to 8 days with bacteria produced mixed results; but overall the data indicate a low degree of toxicity for acetone. The only exception to these findings were the results obtained with the flagellated protozoa (*Entosiphon sulcatum*) which yielded a 3-day NOEC of 28 mg/L.

## Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
acetone	LOW (Half-life = 14 days)	MEDIUM (Half-life = 116.25 days)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW	LOW
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
dimethyl ether	LOW	LOW

**Bioaccumulative potential**

Ingredient	Bioaccumulation
acetone	LOW (BCF = 0.69)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW (LogKOW = 0.56)
xylene	MEDIUM (BCF = 740)
dimethyl ether	LOW (LogKOW = 0.1)

**Mobility in soil**

Ingredient	Mobility
acetone	HIGH (KOC = 1.981)
propylene glycol monomethyl ether acetate, alpha-isomer	HIGH (KOC = 1.838)
dimethyl ether	HIGH (KOC = 1.292)

**SECTION 13 DISPOSAL CONSIDERATIONS****Waste treatment methods**

<b>Product / Packaging disposal</b>	<p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> <li>▶ Reduction</li> <li>▶ Reuse</li> <li>▶ Recycling</li> <li>▶ Disposal (if all else fails)</li> </ul> <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> <li>▶ <b>DO NOT allow wash water from cleaning or process equipment to enter drains.</b></li> <li>▶ It may be necessary to collect all wash water for treatment before disposal.</li> <li>▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.</li> <li>▶ Where in doubt contact the responsible authority.</li> <li>▶ Consult State Land Waste Management Authority for disposal.</li> <li>▶ Discharge contents of damaged aerosol cans at an approved site.</li> <li>▶ Allow small quantities to evaporate.</li> <li>▶ <b>DO NOT incinerate or puncture aerosol cans.</b></li> <li>▶ Bury residues and emptied aerosol cans at an approved site.</li> </ul>
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**SECTION 14 TRANSPORT INFORMATION****Labels Required**

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

**Land transport (ADG)**

<b>UN number</b>	1950
<b>Packing group</b>	Not Applicable
<b>UN proper shipping name</b>	AEROSOLS
<b>Environmental hazard</b>	Not Applicable

ColorSpec Caliper HT, 300g (ColorSpec Caliper HT)

<b>Transport hazard class(es)</b>	Class	2.1
	Subrisk	Not Applicable
<b>Special precautions for user</b>	Special provisions	63 190 277 327 344
	Limited quantity	1000ml

**Air transport (ICAO-IATA / DGR)**

<b>UN number</b>	1950	
<b>Packing group</b>	Not Applicable	
<b>UN proper shipping name</b>	Aerosols	
<b>Environmental hazard</b>	Not Applicable	
<b>Transport hazard class(es)</b>	ICAO/IATA Class	2.1
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	10L
<b>Special precautions for user</b>	Special provisions	A145A167A802; A1A145A167A802
	Cargo Only Packing Instructions	203
	Cargo Only Maximum Qty / Pack	150 kg
	Passenger and Cargo Packing Instructions	203; Forbidden
	Passenger and Cargo Maximum Qty / Pack	75 kg; Forbidden
	Passenger and Cargo Limited Quantity Packing Instructions	Y203; Forbidden
	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G; Forbidden

**Sea transport (IMDG-Code / GGVSee)**

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

**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**

**ACETONE(67-64-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

**PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, ALPHA-ISOMER(108-65-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

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

Continued...

## Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

## Australia Inventory of Chemical Substances (AICS)

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

**DIMETHYL ETHER(115-10-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

## Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

## Australia Inventory of Chemical Substances (AICS)

International Air Transport Association (IATA) Dangerous Goods Regulations - Prohibited List Passenger and Cargo Aircraft

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (propylene glycol monomethyl ether acetate, alpha-isomer; acetone; xylene; dimethyl ether)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	Y
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
<b>Legend:</b>	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

**SECTION 16 OTHER INFORMATION****Other information****Ingredients with multiple cas numbers**

Name	CAS No
propylene glycol monomethyl ether acetate, alpha-isomer	108-65-6, 142300-82-1, 84540-57-8
dimethyl ether	115-10-6, 157621-61-9

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

A list of reference resources used to assist the committee may be found at:

[www.chemwatch.net](http://www.chemwatch.net)

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

**Definitions and abbreviations**

PC—TWA: Permissible Concentration-Time Weighted Average  
 PC—STEL: Permissible Concentration-Short Term Exposure Limit  
 IARC: International Agency for Research on Cancer  
 ACGIH: American Conference of Governmental Industrial Hygienists  
 STEL: Short Term Exposure Limit  
 TEEL: Temporary Emergency Exposure Limit.  
 IDLH: Immediately Dangerous to Life or Health Concentrations  
 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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**ColorSpec Caliper HT, 300g (ColorSpec Caliper HT)**

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