

EVIC 1800A GLOBAL POLYURETHANE PART A (LEAD FREE)

Chemwatch Material Safety Data Sheet
Issue Date: 25-Sep-2008
NA317TC

CHEMWATCH 5093-13
Version No:4
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Section 2 - HAZARDS IDENTIFICATION

POISONS SCHEDULE

S5

RISK

- » Highly flammable.
- » Harmful by inhalation and in contact with skin.
- » Limited evidence of a carcinogenic effect.
- » May impair fertility.
- » May cause harm to the unborn child.
- » HARMFUL- May cause lung damage if swallowed.

SAFETY

- » Keep locked up.
- » Keep away from sources of ignition. No smoking.
- » Do not breathe gas/fumes/vapour/spray.
- » In case of insufficient ventilation wear suitable respiratory equipment.
- » Use only in well ventilated areas.
- » Keep container in a well ventilated place.
- » Avoid exposure - obtain special instructions before use.
- » To clean the floor and all objects contaminated by this material use water and detergent.
- » Keep container tightly closed.
- » This material and its container must be disposed of in a safe way.
- » Keep away from food drink and animal feeding stuffs.
- » In case of contact with eyes rinse with plenty of water and contact Doctor or Poisons Information Centre.
- » This material and its container must be disposed of as hazardous waste.

Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

NAME	CAS RN	%
resin unregulated		30-60
titanium dioxide	13463-67-7	30-60
aromatic solvents		1-10
2- ethoxyethyl acetate	111-15-9	1-10
alkyl ester		1-10
alkyl ketone		1-10
propylene glycol monomethyl ether acetate, beta- isomer	70657-70-4	1-10
additives		1-10

Section 4 - FIRST AID MEASURES

SWALLOWED

- If swallowed do NOT induce vomiting.
- If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.
- Observe the patient carefully.
- Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.
- Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.
- Seek medical advice.
- Avoid giving milk or oils.
- Avoid giving alcohol.
- If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

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Section 4 - FIRST AID MEASURES

EYE

» If this product comes in contact with the eyes:

- Wash out immediately with fresh running water.
- Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.
- If pain persists or recurs seek medical attention.
- Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

SKIN

» If skin contact occurs:

- Immediately remove all contaminated clothing, including footwear.
- Flush skin and hair with running water (and soap if available).
- Seek medical attention in event of irritation.

INHALED

- If fumes or combustion products are inhaled remove from contaminated area.
- Lay patient down. Keep warm and rested.
- Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.
- Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.
- Transport to hospital, or doctor.

NOTES TO PHYSICIAN

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

Treat symptomatically.

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. 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	End of shift	
	2 mg/min	Last 4 hrs of shift	

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

- Hepatic metabolism produces ethylene glycol as a metabolite.
- Clinical presentation, following severe intoxication, resembles that of ethylene glycol exposures.

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Section 4 - FIRST AID MEASURES

- Monitoring the urinary excretion of the alkoxyacetic acid metabolites may be a useful indication of exposure. [Ellenhorn and Barceloux: Medical Toxicology].

Section 5 - FIRE FIGHTING MEASURES

EXTINGUISHING MEDIA

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

FIRE FIGHTING

- Alert Fire Brigade and tell them location and nature of hazard.
- May be violently or explosively reactive.
- Wear breathing apparatus plus protective gloves.
- Prevent, by any means available, spillage from entering drains or water course.
- Consider evacuation (or protect in place).
- Fight fire from a safe distance, with adequate cover.
- If safe, switch off electrical equipment until vapour fire hazard removed.
- Use water delivered as a fine spray to control the fire and cool adjacent area.
- Avoid spraying water onto liquid pools.
- Do not approach containers suspected to be hot.
- Cool fire exposed containers with water spray from a protected location.
- If safe to do so, remove containers from path of fire.

FIRE/EXPLOSION HAZARD

- Liquid and vapour are highly flammable.
 - Severe fire hazard when exposed to heat, flame and/or oxidisers.
 - Vapour may travel a considerable distance to source of ignition.
 - Heating may cause expansion or decomposition leading to violent rupture of containers.
 - On combustion, may emit toxic fumes of carbon monoxide (CO).
- Combustion products include: carbon dioxide (CO₂), other pyrolysis products typical of burning organic material.
Contains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions.

FIRE INCOMPATIBILITY

- Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result.

HAZCHEM: None

Section 6 - ACCIDENTAL RELEASE MEASURES

EMERGENCY PROCEDURES

MINOR SPILLS

- Remove all ignition sources.
- Clean up all spills immediately.
- Avoid breathing vapours and contact with skin and eyes.
- Control personal contact by using protective equipment.
- Contain and absorb small quantities with vermiculite or other absorbent material.
- Wipe up.
- Collect residues in a flammable waste container.

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Section 6 - ACCIDENTAL RELEASE MEASURES

MAJOR SPILLS

» Chemical Class: aromatic hydrocarbons

For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
LAND SPILL - SMALL				
Feathers - pillow	1	throw	pitchfork	DGC, RT
cross- linked polymer - particulate	2	shovel	shovel	R, W, SS
cross- linked polymer- pillow	2	throw	pitchfork	R, DGC, RT
sorbent clay - particulate	3	shovel	shovel	R, I, P,
treated clay/ treated natural organic - particulate	3	shovel	shovel	R, I
wood fibre - pillow	4	throw	pitchfork	R, P, DGC, RT
LAND SPILL - MEDIUM				
cross- linked polymer - particulate	1	blower	skid loader	R, W, SS
treated clay/ treated natural organic - particulate	2	blower	skid loader	R, I
sorbent clay - particulate	3	blower	skid loader	R, I, P
polypropylene - particulate	3	blower	skid loader	W, SS, DGC
feathers - pillow	3	throw	skid loader	DGC, RT
expanded mineral - particulate	4	blower	skid loader	R, I, W, P, DGC

Legend

DGC: Not effective where ground cover is dense

R; Not reusable

I: Not incinerable

P: Effectiveness reduced when rainy

RT: Not effective where terrain is rugged

SS: Not for use within environmentally sensitive sites

W: Effectiveness reduced when windy

Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;

R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988.

- Clear area of personnel and move upwind.
- Alert Fire Brigade and tell them location and nature of hazard.
- May be violently or explosively reactive.
- Wear breathing apparatus plus protective gloves.
- Prevent, by any means available, spillage from entering drains or water course.
- Consider evacuation (or protect in place).
- 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.

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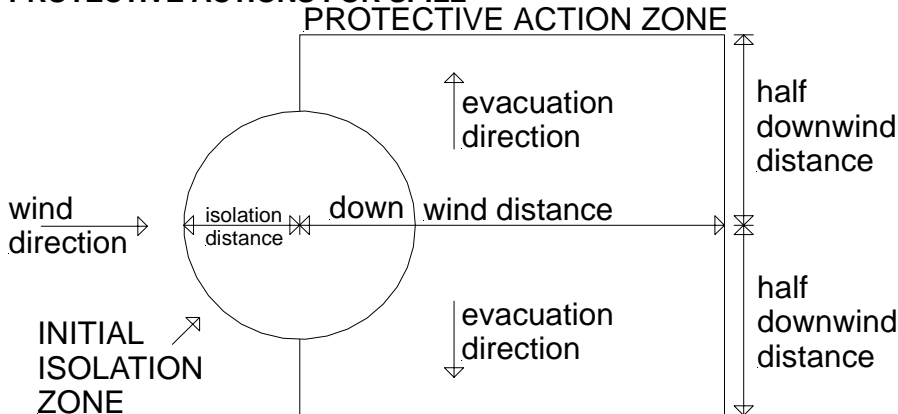
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Section 6 - ACCIDENTAL RELEASE MEASURES

- Contain spill with sand, earth or vermiculite.
- Use only spark-free shovels and explosion proof equipment.
- Collect recoverable product into labelled containers for recycling.
- Absorb remaining product with sand, earth or vermiculite.
- Collect solid residues and seal in labelled drums for disposal.
- Wash area and prevent runoff into drains.
- If contamination of drains or waterways occurs, advise emergency services.

PROTECTIVE ACTIONS FOR SPILL



From IERG (Canada/Australia)

Isolation Distance	25 metres
Downwind Protection Distance	300 metres
IERG Number	14

FOOTNOTES

- 1 PROTECTIVE ACTION ZONE is defined as the area in which people are at risk of harmful exposure. This zone assumes that random changes in wind direction confines the vapour plume to an area within 30 degrees on either side of the predominant wind direction, resulting in a crosswind protective action distance equal to the downwind protective action distance.
- 2 PROTECTIVE ACTIONS should be initiated to the extent possible, beginning with those closest to the spill and working away from the site in the downwind direction. Within the protective action zone a level of vapour concentration may exist resulting in nearly all unprotected persons becoming incapacitated and unable to take protective action and/or incurring serious or irreversible health effects.
- 3 INITIAL ISOLATION ZONE is determined as an area, including upwind of the incident, within which a high probability of localised wind reversal may expose nearly all persons without appropriate protection to life-threatening concentrations of the material.
- 4 SMALL SPILLS involve a leaking package of 200 litres (55 US gallons) or less, such as a drum (jerrican or box with inner containers). Larger packages leaking less than 200 litres and compressed gas leaking from a small cylinder are also considered "small spills".
LARGE SPILLS involve many small leaking packages or a leaking package of greater than 200 litres, such as a cargo tank, portable tank or a "one-tonne" compressed gas cylinder.
- 5 Guide 128 is taken from the US DOT emergency response guide book.
- 6 IERG information is derived from CANUTEC - Transport Canada.

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

Section 7 - HANDLING AND STORAGE

PROCEDURE FOR HANDLING

- Containers, even those that have been emptied, may contain explosive vapours.
 - Do NOT cut, drill, grind, weld or perform similar operations on or near containers.
- Contains low boiling substance:

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Section 7 - HANDLING AND STORAGE

Storage in sealed containers may result in pressure buildup causing violent rupture of containers not rated appropriately.

- Check for bulging containers.

- Vent periodically

- Always release caps or seals slowly to ensure slow dissipation of vapours.

- DO NOT allow clothing wet with material to stay in contact with skin.

The tendency of many ethers to form explosive peroxides is well documented. Ethers lacking non-methyl hydrogen atoms adjacent to the ether link are thought to be relatively safe

- DO NOT concentrate by evaporation, or evaporate extracts to dryness, as residues may contain explosive peroxides with DETONATION potential.

- Any static discharge is also a source of hazard.

- Before any distillation process remove trace peroxides by shaking with excess 5% aqueous ferrous sulfate solution or by percolation through a column of activated alumina.

- Distillation results in uninhibited ether distillate with considerably increased hazard because of risk of peroxide formation on storage.

- Add inhibitor to any distillate as required.

- When solvents have been freed from peroxides by percolation through columns of activated alumina, the absorbed peroxides must promptly be desorbed by treatment with polar solvents such as methanol or water, which should then be disposed of safely.

- Electrostatic discharge may be generated during pumping - this may result in fire.

- Ensure electrical continuity by bonding and grounding (earthing) all equipment.

- Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (≤ 1 m/sec until fill pipe submerged to twice its diameter, then ≤ 7 m/sec).

- Avoid splash filling.

- Do NOT use compressed air for filling discharging or handling operations.

- Avoid all personal contact, including inhalation.

- Wear protective clothing when risk of exposure occurs.

- Use in a well-ventilated area.

- Prevent concentration in hollows and sumps.

- DO NOT enter confined spaces until atmosphere has been checked.

- Avoid smoking, naked lights, heat or ignition sources.

- When handling, DO NOT eat, drink or smoke.

- Vapour may ignite on pumping or pouring due to static electricity.

- DO NOT use plastic buckets.

- Earth and secure metal containers when dispensing or pouring product.

- Use spark-free tools when handling.

- Avoid contact with incompatible materials.

- Keep containers securely sealed.

- Avoid physical damage to containers.

- Always wash hands with soap and water after handling.

- Work clothes should be laundered separately.

- Use good occupational work practice.

- Observe manufacturer's storing and handling recommendations.

- Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.

SUITABLE CONTAINER

- Packing as supplied by manufacturer.

- Plastic containers may only be used if approved for flammable liquid.

- Check that containers are clearly labelled and free from leaks.

- For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : Where a can is to be used as an inner package, the can must have a screwed enclosure.

- For materials with a viscosity of at least 2680 cSt. (23 deg. C)

- For manufactured product having a viscosity of at least 250 cSt. (23 deg. C)

- Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C)

- (i) : Removable head packaging;

- (ii) : Cans with friction closures and

- (iii) : low pressure tubes and cartridges may be used.

- Where combination packages are used, and the inner packages are of glass, there must be sufficient inert

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cushioning material in contact with inner and outer packages

• In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.

STORAGE INCOMPATIBILITY

» Propylene glycol monomethyl ether acetate:

- may polymerise unless properly inhibited due to peroxide formation
- should be isolated from UV light, high temperatures, free radical initiators
- may react with strong oxidisers to produce fire and/ or explosion
- reacts violently with sodium peroxide, uranium fluoride
- is incompatible with sulfuric acid, nitric acid, caustics, aliphatic amines, isocyanates, boranes.
- Avoid reaction with oxidising agents.

STORAGE REQUIREMENTS

- Store in original containers in approved flame-proof area.
- No smoking, naked lights, heat or ignition sources.
- DO NOT store in pits, depressions, basements or areas where vapours may be trapped.
- Keep containers securely sealed.
- Store away from incompatible materials in a cool, dry well ventilated area.
- Protect containers against physical damage and check regularly for leaks.
- Observe manufacturer's storing and handling recommendations.

Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

EXPOSURE CONTROLS

Source	Material	TWA ppm	TWA mg/m ³
Australia Exposure Standards	titanium dioxide (Titanium dioxide (a))		10
Australia Exposure Standards	2- ethoxyethyl acetate (2-Ethoxyethyl acetate)	5	27

The following materials had no OELs on our records

- propylene glycol monomethyl ether acetate, beta- isomer: CAS:70657- 70- 4

EMERGENCY EXPOSURE LIMITS

Material	Revised IDLH Value (mg/m ³)	Revised IDLH Value (ppm)
titanium dioxide	5, 000	
2- ethoxyethyl acetate		500

MATERIAL DATA

» 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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Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

INGREDIENT DATA

TITANIUM DIOXIDE:

» It is the goal of the ACGIH (and other Agencies) to recommend TLVs (or their equivalent) for all substances for which there is evidence of health effects at airborne concentrations encountered in the workplace.

At this time no TLV has been established, even though this material may produce adverse health effects (as evidenced in animal experiments or clinical experience). Airborne concentrations must be maintained as low as is practically possible and occupational exposure must be kept to a minimum.

NOTE: The ACGIH occupational exposure standard for Particles Not Otherwise Specified (P.N.O.S) does NOT apply

Sensory irritants are chemicals that produce temporary and undesirable side-effects on the eyes, nose or throat. Historically occupational exposure standards for these irritants have been based on observation of workers' responses to various airborne concentrations. Present day expectations require that nearly every individual should be protected against even minor sensory irritation and exposure standards are established using uncertainty factors or safety factors of 5 to 10 or more. On occasion animal no-observable-effect-levels (NOEL) are used to determine these limits where human results are unavailable. An additional approach, typically used by the TLV committee (USA) in determining respiratory standards for this group of chemicals, has been to assign ceiling values (TLV C) to rapidly acting irritants and to assign short-term exposure limits (TLV STELs) when the weight of evidence from irritation, bioaccumulation and other endpoints combine to warrant such a limit. In contrast the MAK Commission (Germany) uses a five-category system based on intensive odour, local irritation, and elimination half-life. However this system is being replaced to be consistent with the European Union (EU) Scientific Committee for Occupational Exposure Limits (SCOEL); this is more closely allied to that of the USA.

OSHA (USA) concluded that exposure to sensory irritants can:

- cause inflammation
- cause increased susceptibility to other irritants and infectious agents
- lead to permanent injury or dysfunction
- permit greater absorption of hazardous substances and
- acclimate the worker to the irritant warning properties of these substances thus increasing the risk of

overexposure.

Animal exposed by inhalation to 10 mg/m³ titanium dioxide show no significant fibrosis, possibly reversible tissue reaction. The architecture of lung air spaces remains intact.

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

2-ETHOXYETHYL ACETATE:

Odour Threshold Value: 0.06 ppm (detection), 0.13 ppm (recognition) This substance is readily hydrolysed in the body yielding ethylene glycol monoethyl ether which is a putative reproductive toxin. The TLV-TWA is thought to be protective against testicular effects.

PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, BETA-ISOMER:

MAK Value: 5 ppm, 28 mg/m³

PERSONAL PROTECTION

EYE

- 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 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].

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HANDS/FEET

- Wear chemical protective gloves, eg. PVC.
 - Wear safety footwear or safety gumboots, eg. Rubber.
- Suitability and durability of glove type is dependent on usage. Factors such as:
- frequency and duration of contact,
 - chemical resistance of glove material,
 - glove thickness and
 - dexterity,
- are important in the selection of gloves.

OTHER

- Overalls.
- PVC Apron.
- PVC protective suit may be required if exposure severe.
- Eyewash unit.
- Ensure there is ready access to a safety shower.
- Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity.

RESPIRATOR

» Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Breathing Zone Level ppm (volume)	Maximum Protection Factor	Half- face Respirator	Full- Face Respirator
1000	10	A- AUS	-
1000	50	-	A- AUS
5000	50	Airline *	-
5000	100	-	A- 2
10000	100	-	A- 3
	100+		Airline**

* - Continuous Flow

** - Continuous-flow or positive pressure demand.

The local concentration of material, quantity and conditions of use determine the type of personal protective equipment required. For further information consult site specific CHEMWATCH data (if available), or your Occupational Health and Safety Advisor.

ENGINEERING CONTROLS

» For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant.

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:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25- 0.5 m/s (50- 100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5- 1 m/s (100- 200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1- 2.5 m/s (200- 500 f/min.)

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Within each range the appropriate value depends on:

Lower end of the range

- 1: Room air currents minimal or favourable to capture
- 2: Contaminants of low toxicity or of nuisance value only.
- 3: Intermittent, low production.
- 4: Large hood or large air mass in motion

Upper end of the range

- 1: Disturbing room air currents
- 2: Contaminants of high toxicity
- 3: High production, heavy use
- 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.

Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

APPEARANCE

» Note that all of the monopropylene glycol ethers may exist in two isomeric forms, alpha or beta. The alpha form, which is thermodynamically favored during synthesis, consists of a secondary alcohol configuration. The beta form consists of a primary alcohol. The two isomeric forms are shown above. The di- and tripropylene glycol ethers may form up to 4 and 8 isomeric forms, respectively. Even so, all isomers exhibit either the "alpha" or "beta" configuration, existing as secondary or primary alcohols, respectively. The distribution of isomeric forms for the di- and tripropylene glycols, as with the mono-PGEs, also results in predominantly the alpha form (i.e., a secondary alcohol). It should be noted that only the alpha isomer and isomeric mixtures (consisting predominantly of the alpha isomer) are produced commercially; the purified beta isomer is not produced at this time.

White highly flammable liquid with a solvent odour; does not mix with water.

PHYSICAL PROPERTIES

Liquid.

Does not mix with water.

Sinks in water.

Molecular Weight: Not Available
Melting Range (°C): Not Available
Solubility in water (g/L): Immiscible
pH (1% solution): Not Applicable
Volatile Component (%vol): Not Available
Relative Vapour Density (air=1): >1
Lower Explosive Limit (%): Not Available
Autoignition Temp (°C): Not Available
State: Liquid

Boiling Range (°C): 78- 157
Specific Gravity (water= 1): 1.37- 1.73
pH (as supplied): Not Applicable
Vapour Pressure (kPa): Not Available
Evaporation Rate: Not Available
Flash Point (°C): 0
Upper Explosive Limit (%): Not Available
Decomposition Temp (°C): Not Available
Viscosity: Not Available

Section 10 - CHEMICAL STABILITY AND REACTIVITY INFORMATION

CONDITIONS CONTRIBUTING TO INSTABILITY

- Presence of incompatible materials.
- Product is considered stable.
- Hazardous polymerisation will not occur.

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

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Section 11 - TOXICOLOGICAL INFORMATION

POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

SWALLOWED

» 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).

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

EYE

» Limited evidence or practical experience suggests, that the material may cause moderate 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. Repeated or prolonged exposure may cause moderate inflammation (similar to windburn) characterised by a temporary redness of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.

Undiluted propylene glycol monomethyl ether acetate (PGMEA) causes moderate discomfort, slight conjunctival redness and slight corneal injury in rabbits.

SKIN

» Skin contact with the material may be harmful; systemic effects may result following absorption.

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.

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

Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.

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

INHALED

» 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.

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.

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CHRONIC HEALTH EFFECTS

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

There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: - clear evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects.

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:

- 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.

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative effects involving organs or biochemical systems.

Long term exposure to the dusts of titanium and several of its compounds produces chronic lung disease (fibrosis) in animals. Radiological evidence exists amongst titanium dioxide workers suggesting chronic lung changes which resemble a slight form of silicosis. Workers chronically exposed to titanium or titanium dioxide dusts show a high incidence of chronic bronchitis (endobronchitis and peribronchitis). Early stages of this disease are characterised by impaired pulmonary respiration and ventilatory capacity and by reduced blood alkalinity. Cardiac changes characteristic of pulmonary disease (with hypertrophy of the right auricle) have also been observed amongst workers. The largest of the cohort studies was among white male production workers in the titanium dioxide industry in six European countries. The study indicated a slightly increased risk for lung cancer compared with the general population. However, there was no evidence of an exposure-response relationship within the cohort. No increase in the mortality rates for kidney cancer was found when the cohort was compared with the general population, but there was a suggestion of an exposure-response relationship in internal analyses. The other cohort studies, both of which were conducted in the USA, did not report an increased risk for lung cancer or cancer at any other site; no results for kidney cancer were reported, presumably because there were few cases.

One population-based case-control study conducted in Montreal did not indicate an increased risk for lung or kidney cancer.

In summary, the studies do not suggest an association between occupational exposure to titanium dioxide as it occurred in recent decades in western Europe and North America and risk for cancer.

All the studies had methodological limitations; misclassification of exposure could not be ruled out. None of the studies was designed to assess the impact of particle size (fine or ultrafine) or the potential effect of the coating compounds on the risk for lung cancer.

An increased incidence of lung adenomas in rats of both sexes and of cystic keratinising lesions, diagnosed as squamous cell carcinomas in female rats, was seen in animals subject to high doses of inhaled titanium dioxide. Intratracheal delivery of titanium dioxide in combination with benz[a]pyrene produced an increase in benign and malignant tumours of the larynx, trachea and lungs in hamsters.

Squamous cell carcinomas developed after exposure to 250 mg/m³ for 6 hours/day, 5 days/week for 2 years in rats; the type of carcinoma that developed was considered to be a unique experimentally induced tumour and to be of questionable relevance for extrapolation of the results to humans. Given the extremely high level of dust in the lungs, the carcinomas were postulated to be the result of saturation of the normal pulmonary clearance mechanisms. At 50 mg/m³, massive accumulations of dust-laden macrophages, foamy dust cells and free particles were considered indicative of such overload.

TOXICITY AND IRRITATION

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

» The material may produce moderate eye irritation leading to 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 (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.

For ethylene glycol:

Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow.

Following absorption, ethylene glycol is distributed throughout the body according to total body water. In

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

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

Cardiovascular Effects. Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12- 24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol.

Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases. Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.

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

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

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

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

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

Neurological Effects: Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to

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unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation.

Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion.

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

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

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

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

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

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,

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000 mg/m³ for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC₅₀ is >2,040 mg/m³. For PnB, the 4-hour LC₅₀ was >651 ppm (>3,412 mg/m³), 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/m³ (600 ppm) for PnB and 2,010 mg/m³ (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m³ (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m³ (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/m³) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m³). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m³), with decreased body weights occurring at 3000 ppm (11058 mg/m³). 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].

TITANIUM DIOXIDE:

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

TOXICITY

Oral (Rat) LD₅₀: >20000 mg/kg *

Oral (Mouse) LD₅₀: >10000 mg/kg *

» The material may produce moderate eye irritation leading to 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 (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

IRRITATION

Skin (human): 0.3 mg /3D (int)- Mild *

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intracellular oedema of the epidermis.

For titanium dioxide:

Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals.

(General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin.

Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes.

However, the workers in these studies were also exposed to asbestos and/or silica.

No data were available on genotoxic effects in titanium dioxide-exposed humans.

Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts.

Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium.

Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light.

Animal carcinogenicity data

Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats.

In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative.

Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice.

In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative.

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* IUCLID

2-ETHOXYETHYL ACETATE:

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

TOXICITY

Oral (rat) LD50: 2900 mg/kg

Inhalation (rat) LC50: 12100 mg/m³/8 h Dermal (rabbit):420

mg(open)- Mild

Dermal (rabbit) LD50: 10500 mg/kg

Inhalation (rat) TClO: 50 ppm/6 h

» 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.

Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).

IRRITATION

Eye (rabbit): 40 mg - Moderate

PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE, BETA-ISOMER:

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

TOXICITY

Oral (rat) LD50: 8532 mg/kg

Dermal (rabbit) LD50: >5000 mg/kg* [CCINFO]*

Inhalation (rat) LC50: 4345 ppm/6h

» Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

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

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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/m³ for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m³. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m³), 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/m³ (600 ppm) for PnB and 2,010 mg/m³ (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m³ (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m³ (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/m³) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m³). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m³), with decreased body weights occurring at 3000 ppm (11058 mg/m³). 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].

No data for material.

Data for isomer only

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Section 11 - TOXICOLOGICAL INFORMATION

as its alpha isomer; propylene glycol monomethyl ether acetate:

CARCINOGEN

titanium dioxide	International Agency for Research on Cancer (IARC) Carcinogens	Group	2B
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SKIN

2- ethoxyethyl acetate	Australia Exposure Standards - Skin	Notes	Sk
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Section 12 - ECOLOGICAL INFORMATION

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

Section 13 - DISPOSAL CONSIDERATIONS

- Containers may still present a chemical hazard/ danger when empty.
- Return to supplier for reuse/ recycling if possible.

Otherwise:

- If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.

- Where possible retain label warnings and MSDS and observe all notices pertaining to the product.

Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.

A Hierarchy of Controls seems to be common - the user should investigate:

- Reduction,
- Reuse
- Recycling
- Disposal (if all else fails)

This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. 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.

- DO NOT allow wash water from cleaning or process equipment to enter drains.
- It may be necessary to collect all wash water for treatment before disposal.
- In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
- Where in doubt contact the responsible authority.
- Recycle wherever possible.
- Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified.
- Dispose of by: Burial in a licenced land-fill or Incineration in a licenced apparatus (after admixture with suitable combustible material).
- Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

Section 14 - TRANSPORTATION INFORMATION

Labels Required: FLAMMABLE LIQUID
HAZCHEM: 3[Y]E (ADG6)

UNDG:

Dangerous Goods Class: 3	Subrisk: None
UN Number: 1263	Packing Group: II
Shipping Name: PAINT RELATED MATERIAL (including paint thinning or	

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Section 14 - TRANSPORTATION INFORMATION

reducing compound)

Air Transport IATA:

ICAO/IATA Class:	3	ICAO/IATA Subrisk:	None
UN/ID Number:	1263	Packing Group:	II
Special provisions:	A3 A72		
Shipping name:	PAINT RELATED MATERIAL		

Maritime Transport IMDG:

IMDG Class:	3	IMDG Subrisk:	None
UN Number:	1263	Packing Group:	II
EMS Number:	F- E, S- E	Special provisions:	163 944
Limited Quantities:	5 L	Marine Pollutant:	Not Determined

Shipping Name: PAINT (including paint, lacquer, enamel, stain, shellac solutions, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)

Section 15 - REGULATORY INFORMATION

POISONS SCHEDULE: S5

REGULATIONS

Evic 1800A Global Polyurethane Part A (Lead Free) (CAS: None):
No regulations applicable

titanium dioxide (CAS: 13463-67-7) is found on the following regulatory lists;

- Australia Exposure Standards
- Australia High Volume Industrial Chemical List (HVICL)
- Australia Inventory of Chemical Substances (AICS)
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 4
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 5
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 7
- Australia Therapeutic Goods Administration (TGA) Substances that may be used as active ingredients in Listed medicines
- Australia Therapeutic Goods Administration (TGA) Sunscreening agents permitted as active ingredients in listed products

CODEX General Standard for Food Additives (GSFA) - Additives Permitted for Use in Food in General, Unless Otherwise Specified, in Accordance with GMP

- IMO IBC Code Chapter 17: Summary of minimum requirements
- International Agency for Research on Cancer (IARC) Carcinogens
- OECD Representative List of High Production Volume (HPV) Chemicals

titanium dioxide (CAS: 1317-70-0) is found on the following regulatory lists;

- Australia Inventory of Chemical Substances (AICS)
- OECD Representative List of High Production Volume (HPV) Chemicals

titanium dioxide (CAS: 1317-80-2) is found on the following regulatory lists;

- Australia Inventory of Chemical Substances (AICS)
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 4
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 5
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 7
- GESAMP/EHS Composite List of Hazard Profiles - Hazard evaluation of substances transported by ships
- OECD Representative List of High Production Volume (HPV) Chemicals

titanium dioxide (CAS: 1309-63-3) is found on the following regulatory lists;

- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 4
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 5
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 7

titanium dioxide (CAS: 62338-64-1) is found on the following regulatory lists;

- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 4
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 5
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 7

2-ethoxyethyl acetate (CAS: 111-15-9) is found on the following regulatory lists;

- Australia Exposure Standards
- Australia Hazardous Substances
- Australia Inventory of Chemical Substances (AICS)
- Australia National Pollutant Inventory
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix E (Part 2)
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix F (Part 3)
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix I
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 6
- GESAMP/EHS Composite List of Hazard Profiles - Hazard evaluation of substances transported by ships
- IMO IBC Code Chapter 17: Summary of minimum requirements
- IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk

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Section 15 - REGULATORY INFORMATION

OECD Representative List of High Production Volume (HPV) Chemicals

propylene glycol monomethyl ether acetate, beta-isomer (CAS: 70657-70-4) is found on the following regulatory lists:

Australia Hazardous Substances

Australia Inventory of Chemical Substances (AICS)

No data available for titanium dioxide as CAS: 12188-41-9, CAS: 100292-32-8, CAS: 101239-53-6, CAS: 116788-85-3, CAS: 12000-59-8, CAS: 12701-76-7, CAS: 12767-65-6, CAS: 12789-63-8, CAS: 1344-29-2, CAS: 185323-71-1, CAS: 185828-91-5, CAS: 188357-76-8, CAS: 188357-79-1, CAS: 195740-11-5, CAS: 221548-98-7, CAS: 224963-00-2, CAS: 246178-32-5, CAS: 252962-41-7, CAS: 37230-92-5, CAS: 37230-94-7, CAS: 37230-95-8, CAS: 37230-96-9, CAS: 39320-58-6, CAS: 39360-64-0, CAS: 39379-02-7, CAS: 416845-43-7, CAS: 494848-07-6, CAS: 494848-23-6, CAS: 494851-77-3, CAS: 494851-98-8, CAS: 55068-84-3, CAS: 55068-85-4, CAS: 552316-51-5, CAS: 767341-00-4, CAS: 97929-50-5, CAS: 98084-96-9.

Section 16 - OTHER INFORMATION

INGREDIENTS WITH MULTIPLE CAS NUMBERS

Ingredient Name	CAS
titanium dioxide	13463- 67- 7, 1317- 70- 0, 1317- 80- 2, 12188- 41- 9, 1309- 63- 3, 100292- 32- 8, 101239- 53- 6, 116788- 85- 3, 12000- 59- 8, 12701- 76- 7, 12767- 65- 6, 12789- 63- 8, 1344- 29- 2, 185323- 71- 1, 185828- 91- 5, 188357- 76- 8, 188357- 79- 1, 195740- 11- 5, 221548- 98- 7, 224963- 00- 2, 246178- 32- 5, 252962- 41- 7, 37230- 92- 5, 37230- 94- 7, 37230- 95- 8, 37230- 96- 9, 39320- 58- 6, 39360- 64- 0, 39379- 02- 7, 416845- 43- 7, 494848- 07- 6, 494848- 23- 6, 494851- 77- 3, 494851- 98- 8, 55068- 84- 3, 55068- 85- 4, 552316- 51- 5, 62338- 64- 1, 767341- 00- 4, 97929- 50- 5, 98084- 96- 9

REPRODUCTIVE HEALTH GUIDELINES

» Established occupational exposure limits frequently do not take into consideration reproductive end points that are clearly below the thresholds for other toxic effects. Occupational reproductive guidelines (ORGs) have been suggested as an additional standard. These have been established after a literature search for reproductive no-observed-adverse effect-level (NOAEL) and the lowest-observed-adverse-effect-level (LOAEL). In addition the US EPA's procedures for risk assessment for hazard identification and dose-response assessment as applied by NIOSH were used in the creation of such limits. Uncertainty factors (UFs) have also been incorporated.

Ingredient	ORG	UF	Endpoint	CR	Adeq TLV
propylene glycol monomethyl ether acetate, beta- isomer	7.06 mg/m3	100	D	NA	-

» These exposure guidelines have been derived from a screening level of risk assessment and should not be construed as unequivocally safe limits. ORGS represent an 8-hour time-weighted average unless specified otherwise.

CR = Cancer Risk/10000; UF = Uncertainty factor:

TLV believed to be adequate to protect reproductive health:

LOD: Limit of detection

Toxic endpoints have also been identified as:

D = Developmental; R = Reproductive; TC = Transplacental carcinogen

Jankovic J., Drake F.: A Screening Method for Occupational Reproductive

American Industrial Hygiene Association Journal 57: 641-649 (1996).

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

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

www.chemwatch.net/references.

» The (M)SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by

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Section 16 - OTHER INFORMATION

reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

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