

127 UV WASH

Hazard Alert Code:
MODERATE

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

Issue Date: 13-Nov-2009

CD 2009/3

Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

PRODUCT NAME

127 UV Wash

PROPER SHIPPING NAME

HYDROCARBONS, LIQUID, N.O.S.(contains solvent naphtha petroleum, heavy aromatic)

PRODUCT USE

■ Used according to manufacturer's directions. The use of a quantity of material in an unventilated or confined space may result in increased exposure and an irritating atmosphere developing. Before starting consider control of exposure by mechanical ventilation. Printing industry use only.

SUPPLIER

Company: Hurst Australia Pty Ltd

Address:

10 Bellona Avenue

Regents Park

NSW, 2143

AUS

Telephone: +61 2 9644 6888

Emergency Tel: +61 2 9644 6888

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HAZARD RATINGS

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

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



Section 2 - HAZARDS IDENTIFICATION

STATEMENT OF HAZARDOUS NATURE

HAZARDOUS SUBSTANCE. DANGEROUS GOODS. According to the Criteria of NOHSC, and the ADG Code.

POISONS SCHEDULE

S5

RISK

- Flammable.
- Harmful by inhalation in contact with skin and if swallowed.
- Irritating to eyes and skin.
- Toxic to aquatic organisms may cause long-term adverse effects in the aquatic environment.
- HARMFUL - May cause lung damage if swallowed.
- Repeated exposure may cause skin dryness and cracking.
- Vapours may cause drowsiness and dizziness.
- Cumulative effects may result following exposure*.
- May produce discomfort of the respiratory system*.
- Limited evidence of a carcinogenic effect*.
- May be harmful to the foetus/ embryo*.

SAFETY

- Do not breathe gas/ fumes/ vapour/ spray.
- 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.
- If swallowed IMMEDIATELY contact Doctor or Poisons Information Centre (show this container or label).
- Use appropriate container to avoid environment contamination.
- Avoid release to the environment. Refer to special instructions/

127 UV WASH

Hazard Alert Code:
MODERATE

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

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CD 2009/3

- May possibly affect fertility*.

safety data sheets.

* (limited evidence).

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

Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

NAME	CAS RN	%
solvent naphtha petroleum, heavy aromatic	64742-94-5	30-60
ethylene glycol monobutyl ether	111-76-2	10-30
propylene glycol monomethyl ether - alpha isomer	107-98-2	10-30

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.

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.

For acute or short term repeated exposures to petroleum distillates or related hydrocarbons:

- Primary threat to life, from pure petroleum distillate 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 (pO₂ 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.
- Lavage is indicated in patients who require decontamination; ensure use of cuffed endotracheal tube in adult patients.

127 UV WASH

Hazard Alert Code:
MODERATE

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

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CD 2009/3

[Ellenhorn and Barceloux: Medical Toxicology]

Treat symptomatically.

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

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

[Ellenhorn and Barceloux: Medical Toxicology]. For acute or short term repeated exposures to ethylene glycol:

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

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

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

Section 5 - FIRE FIGHTING MEASURES

EXTINGUISHING MEDIA

- Do not use a water jet to fight fire.

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.
- If safe, switch off electrical equipment until vapour fire hazard removed.
- Use water delivered as a fine spray to control fire and cool adjacent area.
- Avoid spraying water onto liquid pools.
- DO NOT approach containers suspected to be hot.
- Cool fire exposed containers with water spray from a protected location.
- If safe to do so, remove containers from path of fire.

FIRE/EXPLOSION HAZARD

■

- Liquid and vapour are flammable.
- Moderate fire hazard when exposed to heat or flame.
- Vapour forms an explosive mixture with air.
- Moderate explosion hazard when exposed to heat or flame.
- 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 monoxide (CO), 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

3Y

Section 6 - ACCIDENTAL RELEASE MEASURES

EMERGENCY PROCEDURES

MINOR SPILLS

■

- Remove all ignition sources.
- Clean up all spills immediately.

127 UV WASH

Hazard Alert Code:
MODERATE

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

Issue Date: 13-Nov-2009

CD 2009/3

- 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.

MAJOR SPILLS

- Chemical Class: aliphatic hydrocarbons

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

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
LAND SPILL - SMALL				
cross-linked polymer - particulate	1	shovel	shovel	R, W, SS
cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT
wood fiber - pillow	2	throw	pitchfork	R, P, DGC, RT
treated wood fibre- pillow	2	throw	pitchfork	DGC, RT
sorbent clay - particulate	3	shovel	shovel	R, I, P
foamed glass - pillow	3	throw	pitchfork	R, P, DGC, RT
LAND SPILL - MEDIUM				
cross-linked polymer - particulate	1	blower	skiploader	R,W, SS
cross-linked polymer - pillow	2	throw	skiploader	R, DGC, RT
sorbent clay - particulate	3	blower	skiploader	R, I, P
polypropylene - particulate	3	blower	skiploader	W, SS, DGC
expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC
polypropylene - mat	4	throw	skiploader	DGC, RT

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.
- 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

127 UV WASH

Hazard Alert Code:
MODERATE

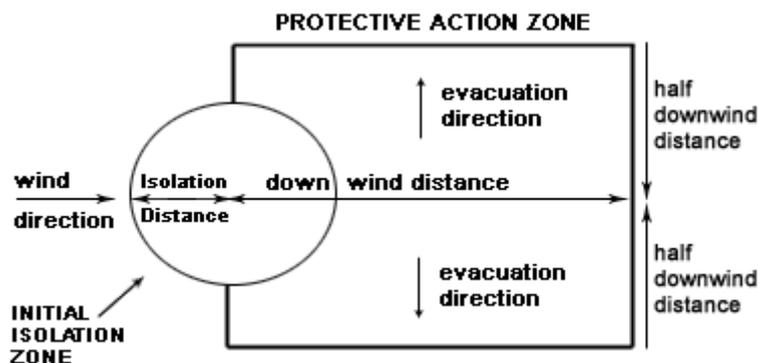
Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

Issue Date: 13-Nov-2009

CD 2009/3



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:
- 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 overexposure occurs.
- Use in a well-ventilated area.
- Prevent concentration in hollows and sumps.
- DO NOT enter confined spaces until atmosphere has been checked.
- Avoid smoking, naked lights or ignition sources.
- Avoid generation of static electricity.

127 UV WASH

Hazard Alert Code:
MODERATE

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

Issue Date: 13-Nov-2009

CD 2009/3

- DO NOT use plastic buckets.
- Earth all lines and equipment.
- Use spark-free tools when handling.
- Avoid contact with incompatible materials.
- When handling, DO NOT eat, drink or smoke.
- Keep containers securely sealed when not in use.
- Avoid physical damage to containers.
- Always wash hands with soap and water after handling.
- Work clothes should be laundered separately.
- Use good occupational work practice.
- Observe manufacturer's 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 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

- Avoid reaction with oxidising agents

STORAGE REQUIREMENTS

- Store in original containers in approved flammable liquid storage area.
 - Store away from incompatible materials in a cool, dry, well-ventilated area.
 - DO NOT store in pits, depressions, basements or areas where vapours may be trapped.
 - No smoking, naked lights, heat or ignition sources.
 - Storage areas should be clearly identified, well illuminated, clear of obstruction and accessible only to trained and authorised personnel - adequate security must be provided so that unauthorised personnel do not have access.
 - Store according to applicable regulations for flammable materials for storage tanks, containers, piping, buildings, rooms, cabinets, allowable quantities and minimum storage distances.
 - Use non-sparking ventilation systems, approved explosion proof equipment and intrinsically safe electrical systems.
 - Have appropriate extinguishing capability in storage area (e.g. portable fire extinguishers - dry chemical, foam or carbon dioxide) and flammable gas detectors.
 - Keep adsorbents for leaks and spills readily available.
 - Protect containers against physical damage and check regularly for leaks.
 - Observe manufacturer's storing and handling recommendations.
- In addition, for tank storages (where appropriate):
- Store in grounded, properly designed and approved vessels and away from incompatible materials.
 - For bulk storages, consider use of floating roof or nitrogen blanketed vessels; where venting to atmosphere is possible, equip storage tank vents with flame arrestors; inspect tank vents during winter conditions for vapour/ ice build-up.
 - Storage tanks should be above ground and diked to hold entire contents.

SAFE STORAGE WITH OTHER CLASSIFIED CHEMICALS

*X: Must not be stored together**O: May be stored together with specific preventions**+: May be stored together*

Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

127 UV WASH

Hazard Alert Code:
MODERATE

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

Issue Date: 13-Nov-2009

CD 2009/3

EXPOSURE CONTROLS

Source	Material	TWA ppm	TWA mg/m ³	STEL ppm	STEL mg/m ³	Peak ppm	Peak mg/m ³	TWA F/CC	Notes
Australia Exposure Standards	127 UV Wash (Propylene glycol monomethyl ether)	100	369	150	553				
Australia Exposure Standards	127 UV Wash (Petrol (gasoline))		900						(see Chapter 16)
Australia Exposure Standards	solvent naphtha petroleum, heavy aromatic (Petrol (gasoline))		900						(see Chapter 16)
Australia Exposure Standards	ethylene glycol monobutyl ether (2-Butoxyethanol)	20	96.9	50	242				Sk
Australia Exposure Standards	propylene glycol monomethyl ether - alpha isomer (Propylene glycol monomethyl ether)	100	369	150	553				

EMERGENCY EXPOSURE LIMITS

Material	Revised IDLH Value (mg/m ³)	Revised IDLH Value (ppm)
ethylene glycol monobutyl ether		700 [Unch]

MATERIAL DATA

127 UV WASH:

■ for propylene glycol monomethyl ether (PGME)

Odour Threshold: 10 ppm.

The TLV-TWA is protective against discomfort caused by odour, against eye and skin irritation, and chronic effects (including possible liver and kidney damage).

Individuals exposed to 100 ppm reported a transient unpleasant odour with slight eye irritation after about 1 or 2 hours. At 300 ppm, mild irritation of the eyes and nose developed within 5 minutes; some individuals found the irritation hardly bearable after about an hour. A concentration of 750 ppm was highly irritating. Signs of central nervous system depression developed at 1000 ppm. Neurological, clinical chemical and general medical examinations showed no other conspicuous toxicity.

Concentrations of the beta-isomer, 2-methoxy-1-propyl acetate are low in commercial grades of PGME and teratogenic effects associated with this isomer are expected to be absent.

Odour Safety Factor(OSF)

OSF=10 (propylene glycol monomethyl ether).

Odour threshold: 0.25 ppm.

The TLV-TWA is protective against ocular and upper respiratory tract irritation and is recommended for bulk handling of gasoline based on calculations of hydrocarbon content of gasoline vapour. A STEL is recommended to prevent mucous membrane and ocular irritation and prevention of acute depression of the central nervous system. Because of the wide variation in molecular weights of its components, the conversion of ppm to mg/m³ is approximate. Sweden recommends hexane type limits of 100 ppm and heptane and octane type limits of 300 ppm. Germany does not assign a value because of the widely differing compositions and resultant differences in toxic properties.

Odour Safety Factor (OSF)

OSF=0.042 (gasoline).

For ethylene glycol monobutyl ether (2-butoxyethanol)

Odour Threshold Value: 0.10 ppm (detection), 0.35 ppm (recognition)

Although rats appear to be more susceptible than other animals anaemia is not uncommon amongst humans following exposure. The TLV reflects the need to maintain exposures below levels found to cause blood changes in experimental animals. It is concluded that this limit will reduce the significant risk of irritation, haematologic effects and other systemic effects observed in humans and animals exposed to higher vapour concentrations. The toxic effects typical of some other glycol ethers (pancytopenia, testis atrophy and teratogenic effects) are not found with this substance.

Odour Safety Factor (OSF)

OSF=2E2 (2-BUTOXYETHANOL).

SOLVENT NAPHTHA PETROLEUM, HEAVY AROMATIC:

■ 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.

127 UV WASH

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Odour Safety Factor (OSF)

OSF=0.042 (gasoline).

REL TWA: 100 ppm [Manufacturer]

CEL TWA: 100 ppm, 550 mg/m³

ETHYLENE GLYCOL MONOBUTYL ETHER:

■ For ethylene glycol monobutyl ether (2-butoxyethanol)

Odour Threshold Value: 0.10 ppm (detection), 0.35 ppm (recognition)

Although rats appear to be more susceptible than other animals anaemia is not uncommon amongst humans following exposure. The TLV reflects the need to maintain exposures below levels found to cause blood changes in experimental animals. It is concluded that this limit will reduce the significant risk of irritation, haematologic effects and other systemic effects observed in humans and animals exposed to higher vapour concentrations. The toxic effects typical of some other glycol ethers (pancytopenia, testis atrophy and teratogenic effects) are not found with this substance.

Odour Safety Factor (OSF)

OSF=2E2 (2-BUTOXYETHANOL).

Exposed individuals are reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class A or B.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

Class	OSF	Description
A	550	Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities
B	26-550	As "A" for 50-90% of persons being distracted
C	1-26	As "A" for less than 50% of persons being distracted
D	0.18-1	10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached
E	<0.18	As "D" for less than 10% of persons aware of being tested

PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER:

■ for propylene glycol monomethyl ether (PGME)

Odour Threshold: 10 ppm.

The TLV-TWA is protective against discomfort caused by odour, against eye and skin irritation, and chronic effects (including possible liver and kidney damage).

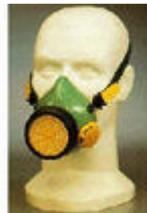
Individuals exposed to 100 ppm reported a transient unpleasant odour with slight eye irritation after about 1 or 2 hours. At 300 ppm, mild irritation of the eyes and nose developed within 5 minutes; some individuals found the irritation hardly bearable after about an hour. A concentration of 750 ppm was highly irritating. Signs of central nervous system depression developed at 1000 ppm. Neurological, clinical chemical and general medical examinations showed no other conspicuous toxicity.

Concentrations of the beta-isomer, 2-methoxy-1-propyl acetate are low in commercial grades of PGME and teratogenic effects associated with this isomer are expected to be absent.

Odour Safety Factor(OSF)

OSF=10 (propylene glycol monomethyl ether).

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

127 UV WASH

Hazard Alert Code:
MODERATE

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

Issue Date: 13-Nov-2009

CD 2009/3

thoroughly. [CDC NIOSH Current Intelligence Bulletin 59]

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.
- For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets), non sparking safety footwear.

RESPIRATOR

■ 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.

Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
10 x ES	A-AUS P	-	A-PAPR-AUS P
50 x ES	Air-line*	-	-
100 x ES	-	A-3 P	-
100+ x ES	-	Air-line**	-

* - 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.)
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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.)
--	----------------------------

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.

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

127 UV WASH

Hazard Alert Code:
MODERATE

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

Issue Date: 13-Nov-2009

CD 2009/3

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. Clear liquid with a mild characteristic odour; not miscible with water.

PHYSICAL PROPERTIES

Liquid.

Does not mix with water.

Floats on water.

Molecular Weight: Not Available	Boiling Range (°C): 145-200	Melting Range (°C): Not Available
Specific Gravity (water=1): 0.86	Solubility in water (g/L): Immiscible	pH (as supplied): Not Available
pH (1% solution): Not Available	Vapour Pressure (kPa): Not Available	Volatile Component (%vol): 100
Evaporation Rate: Not Applicable	Relative Vapour Density (air=1): >1	Flash Point (°C): 42
Lower Explosive Limit (%): Not Available	Upper Explosive Limit (%): Not Available	Autoignition Temp (°C): Not Available
Decomposition Temp (°C): Not Available	State: Liquid	Viscosity: Not Available

Material	Value
log Kow	0.76-0.83
log Kow	-0.53

Section 10 - CHEMICAL STABILITY

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.

Section 11 - TOXICOLOGICAL INFORMATION

POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

SWALLOWED

■ Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.

Swallowing of the liquid may cause aspiration into the lungs with the risk of chemical pneumonitis; serious consequences may result. (ICSC13733).

Ingestion of petroleum hydrocarbons can irritate the pharynx, oesophagus, stomach and small intestine, and cause swellings and ulcers of the mucous. Symptoms include a burning mouth and throat; larger amounts can cause nausea and vomiting, narcosis, weakness, dizziness, slow and shallow breathing, abdominal swelling, unconsciousness and convulsions. Damage to the heart muscle can produce heart beat irregularities, ventricular fibrillation (fatal) and ECG changes. The central nervous system can be depressed. Light species can cause a sharp tingling of the tongue and cause loss of sensation there. Aspiration can cause cough, gagging, pneumonia with swelling and bleeding.

Severe acute exposure to ethylene glycol monobutyl ether, by ingestion, may cause kidney damage, haemoglobinuria, (blood in urine) and is potentially fatal.

EYE

■ Direct eye contact with petroleum hydrocarbons can be painful, and the corneal epithelium may be temporarily damaged. Aromatic species can cause irritation and excessive tear secretion.

When instilled in rabbit eyes ethylene glycol monobutyl ether produced pain, conjunctival irritation, and transient corneal injury.

There is evidence that material may produce eye irritation in some persons and produce eye damage 24 hours or more after instillation. Severe inflammation may be expected with pain. There may be damage to the cornea. Unless treatment is prompt and adequate there may be permanent loss of vision. Conjunctivitis can occur following repeated exposure.

SKIN

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

The material may accentuate any pre-existing dermatitis condition.

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

Toxic amounts of PGME may be absorbed through the skin following extensive prolonged contact ; this may result in drowsiness.

Constant contact with the beta-isomer, on the skin of rabbits, for several weeks caused very mild, simple irritation. Dose rates of 10 mg/kg produced incomplete anaesthesia, depression, and slight increase in kidney weights in test animals.

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

Entry into the blood-stream, through, for example, cuts, abrasions or lesions, may produce systemic injury with harmful effects.

Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.

Ethylene glycol monobutyl ether (2-butoxyethanol) penetrates the skin easily and toxic effects via this route may be more likely than

127 UV WASH

**Hazard Alert Code:
MODERATE**

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

Issue Date: 13-Nov-2009

CD 2009/3

by inhalation. Percutaneous uptake rate in the guinea pig was estimated to be 0.25 $\mu\text{mole}/\text{min}/\text{cm}^2$.

Aromatic hydrocarbons may produce sensitivity and redness of the skin. They are not likely to be absorbed into the body through the skin but branched species are more likely to.

INHALED

■ Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by sleepiness, reduced alertness, loss of reflexes, lack of co-ordination, and vertigo.

There is some evidence to suggest that the material can cause respiratory irritation in some persons. The body's response to such irritation can cause further lung damage.

Inhalation hazard is increased at higher temperatures.

Inhaling high concentrations of mixed hydrocarbons can cause narcosis, with nausea, vomiting and lightheadedness. Low molecular weight (C2-C12) hydrocarbons can irritate mucous membranes and cause incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and stupor. Massive exposures can lead to severe central nervous system depression, deep coma and death. Convulsions can occur due to brain irritation and/or lack of oxygen. Permanent scarring may occur, with epileptic seizures and brain bleeds occurring months after exposure. Respiratory system effects include inflammation of the lungs with oedema and bleeding. Lighter species mainly cause kidney and nerve damage; the heavier paraffins and olefins are especially irritant to the respiratory system. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce sensation loss and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce multiple nerve damage. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue, vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons can sensitise the heart and may cause ventricular fibrillation, leading to death.

Central nervous system (CNS) depression may include general discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.

The odour of PGME becomes objectionable at 100 ppm and intolerable with anaesthetic effects at 1000 ppm. High vapour concentrations (above 1000 ppm) are intolerable due to severe eye, nose and throat irritation. Odour is transiently objectionable above 100 ppm. Obvious sedation, increased liver weights and reduced specific gravity of the urine were found in animals subject to concentrations of 3000 ppm PGME.

Inhalation may produce central nervous system depression. High concentrations of the beta-isomer produced slight growth depression and slight liver change and lung effects in rats and mice.

Ethylene glycol monobutyl ether (2-butoxyethanol) and its metabolite butoxyacetic acid are haemolytic agents, causing red blood cell destruction.

On the basis of industrial experience and volunteer short-term exposure humans are shown to be less susceptible than experimental animals to exposure. In 8-hour exposures at concentrations of 200 or 100 ppm no objective effects were seen other than raised urinary excretion of the metabolite butoxyacetic acid. No increased osmotic fragility of the red blood cell is observed. Subjectively these concentrations were uncomfortable with mild eye, nose and throat irritation occurring. No clinical signs of adverse effects nor subjective complaints were produced when male volunteers were exposed for 2 hours to 20 ppm during light physical exercise. Other studies have established that the most sensitive indicators of toxic effect observed from many of the glycol ethers is an increase in erythrocyte osmotic fragility in rats. This appears to be related to the development of haemoglobinuria at higher exposure levels.

Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful.

Acute effects from inhalation of high vapour concentrations may be chest and nasal irritation with coughing, sneezing, headache and even nausea.

CHRONIC HEALTH EFFECTS

■ Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.

Substance accumulation, in the human body, may occur and may cause some concern following repeated or long-term occupational exposure.

There is some evidence from animal testing that exposure to this material may result in toxic effects to the unborn baby.

Based on experience with similar materials, there is a possibility that exposure to the material may reduce fertility in humans at levels which do not cause other toxic effects.

Constant or exposure over long periods to mixed hydrocarbons may produce stupor with dizziness, weakness and visual disturbance, weight loss and anaemia, and reduced liver and kidney function. Skin exposure may result in drying and cracking and redness of the skin. Chronic exposure to lighter hydrocarbons can cause nerve damage, peripheral neuropathy, bone marrow dysfunction and psychiatric disorders as well as damage the liver and kidneys.

Repeated oral doses of 3 g/kg produced minor changes in the liver and kidneys in rats. Repeated doses on the skin over a 90-day period resulted in absorption and anaesthetic death at 7-10 ml/kg/day. Mild narcosis was observed after topical application of 2-4 ml/kg/day.

Administration of 2% PGME in drinking water ad libitum to males for 25 days did not elicit significant changes in testes or seminal vesicle and coagulating gland weights or in peripheral leukocyte counts. No significant testicular toxicity was found in rats or rabbits that were exposed at up to 3000 PGME, 6 hours/day, 5 days/week for 13 weeks. Oral and parenteral administration to pregnant rabbits, mice and rats did not induce congenital malformations at concentrations up to 1800 mg/kg/day.

In a study on the teratogenic potential of the acetate of the beta-isomer (2-methoxy-1-propyl acetate), a significant increase in the number of litters with abnormal rats and rabbits was found after inhalation exposure by the mothers to 2700 ppm or 550 ppm, respectively, on days 6 to 15, or 6 to 18 of gestation. The rabbit inhalation no-observed-adverse effect concentration was 145 ppm. A similar embryotoxicity profile was seen after inhalation of 2-methoxy-1-propanol (beta-PGMA). In contrast to the alpha-isomer, beta-PGMA is oxidised in rats to 2-methoxypropionic acid.

Male dogs exposed to the beta-isomer, developed numerous spermiphages in epididymi. Administration of high doses of the beta-isomer to rats, by gavage, caused delayed ossification of the skull of rat foetus.

Whilst alpha-PGMA undergoes hepatic O-demethylation as the principal pathway, the beta-isomer is detoxified by alcohol/aldehyde dehydrogenase. Commercial PGME contains low concentrations of the beta-isomer.

There has been concern that this material can cause cancer or mutations, but there is not enough data to make an assessment.

TOXICITY AND IRRITATION

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

■ **for propylene glycol ethers (PGEs):**

Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene

127 UV WASH

Hazard Alert Code:
MODERATE

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

Issue Date: 13-Nov-2009

CD 2009/3

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/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 sensitisers.

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.

Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.

Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetotoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species.

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

Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility.

Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of

127 UV WASH

Hazard Alert Code:
MODERATE

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

Issue Date: 13-Nov-2009

CD 2009/3

neoplasms and nonneoplastic lesions (1). The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of erythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration - dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma.

1: NTP Toxicology Program Technical report Series 484, March 2000.

SOLVENT NAPHTHA PETROLEUM, HEAVY AROMATIC:

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

TOXICITY

Oral (rat) LD50: 3200 mg/kg

Dermal (rabbit) LD50: 3160 mg/kg

IRRITATION

Eye (rabbit): Irritating

[PETROFIN]

- Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.

ETHYLENE GLYCOL MONOBUTYL ETHER:

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

Oral (rat) LD50: 470 mg/kg

Dermal (rabbit) LD50: 220 mg/kg

Inhalation (human) TClO: 100 ppm

Inhalation (human) TClO: 195 ppm/8h

Inhalation (Rat) LC50: 450 ppm *

Skin (rabbit): 500 mg, open; Mild

Eye (rabbit): 100 mg/24h-Moderate

Eye (rabbit): 100 mg SEVERE

* [Union Carbide]

- 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 on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.

For ethylene glycol:

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

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

Respiratory Effects. Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning

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

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

and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of

127 UV WASH

Hazard Alert Code:
MODERATE

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

Issue Date: 13-Nov-2009

CD 2009/3

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 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 ethylene glycol monoalkyl ethers and their acetates (EGMAEs):

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

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

Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m³) for EGHE, LC50 > 400ppm (2620 mg/m³) for EGBEA to LC50 > 2132 ppm (9061 mg/m³) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members

127 UV WASH

Hazard Alert Code:
MODERATE

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

Issue Date: 13-Nov-2009

CD 2009/3

cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitizers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE in vitro than those of rats.

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

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

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

Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes).

Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on

EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m³ and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m³), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m³), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m³) indicate that the members of the category are not teratogenic.

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

Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetotoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species.

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

Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility.

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

1: NTP Toxicology Program Technical report Series 484, March 2000.

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

PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER:

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

Oral (rat) LD50: 3739 mg/kg	Skin (rabbit) 500 mg Open - Mild
Inhalation (human) TClO: 3000 ppm	Eye (rabbit) 230 mg Mild
Inhalation (rat) LC50: 10000 ppm/5 h.	Eye (rabbit) 500 mg/24 h. - Mild
Dermal (rabbit) LD50: 13000 mg/kg	Eye (rabbit): 100 mg SEVERE

■ 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).

127 UV WASH

Hazard Alert Code:
MODERATE

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

Issue Date: 13-Nov-2009

CD 2009/3

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/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.

For PGE - mixed isomers: Exposure of pregnant rats and rabbits to the substance did not give rise to teratogenic effects at concentrations up to 3000 ppm. Foetotoxic effects were seen in rats but not in rabbits at this concentration; maternal toxicity was noted in both species.

NOTE:

CARCINOGEN

Gasoline (NB: Overall evaluation upgraded from 3 to 2B with supporting evidence from other relevant data)

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

Group 2B

127 UV WASH

Hazard Alert Code:
MODERATE

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

Issue Date: 13-Nov-2009

CD 2009/3

Petroleum solvents

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

Group 3

2-Butoxyethanol

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

Group 3

SKIN

ethylene glycol monobutyl ether

Australia Exposure Standards - Skin

Notes

Sk

Section 12 - ECOLOGICAL INFORMATION

Refer to data for ingredients, which follows:

127 UV WASH:

PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER:

ETHYLENE GLYCOL MONOBUTYL ETHER:

■ For glycol ethers:

Environmental fate:

Ether groups are generally stable to hydrolysis in water under neutral conditions and ambient temperatures. OECD guideline studies indicate ready biodegradability for several glycol ethers although higher molecular weight species seem to biodegrade at a slower rate. No glycol ethers that have been tested demonstrate marked resistance to biodegradative processes. Upon release to the atmosphere by evaporation, high boiling glycol ethers are estimated to undergo photodegradation (atmospheric half lives = 2.4-2.5 hr). When released to water, glycol ethers undergo biodegradation (typically 47-92% after 8-21 days) and have a low potential for bioaccumulation (log Kow ranges from -1.73 to +0.51).

Ecotoxicity:

Aquatic toxicity data indicate that the tri- and tetra ethylene glycol ethers are "practically non-toxic" to aquatic species. No major differences are observed in the order of toxicity going from the methyl- to the butyl ethers.

Glycols exert a high oxygen demand for decomposition and once released to th environments cause the death of aquatic organisms if dissolved oxygen is depleted.

127 UV WASH:

PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER:

■ for propylene glycol ethers:

Environmental fate:

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 5.7×10^{-9} atm-m³/mole for TPM to 2.7×10^{-9} atm-m³/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.

127 UV WASH:

PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER:

ETHYLENE GLYCOL MONOBUTYL ETHER:

SOLVENT NAPHTHA PETROLEUM, HEAVY AROMATIC:

■ DO NOT discharge into sewer or waterways.

127 UV WASH:

ETHYLENE GLYCOL MONOBUTYL ETHER:

■ For ethylene glycol monoalkyl ethers and their acetates:

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

Environmental fate:

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

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

Ecotoxicity:

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

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

127 UV WASH

Hazard Alert Code:
MODERATE

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

Issue Date: 13-Nov-2009

CD 2009/3

reproduction in *Ceriodaphnia dubia*.

127 UV WASH:

SOLVENT NAPHTHA PETROLEUM, HEAVY AROMATIC:

- Toxic to aquatic organisms.

127 UV WASH:

SOLVENT NAPHTHA PETROLEUM, HEAVY AROMATIC:

- Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

127 UV WASH:

Marine Pollutant:

Not Determined

/53#90alk#90hc

SOLVENT NAPHTHA PETROLEUM, HEAVY AROMATIC:

- May cause long-term adverse effects in the aquatic environment.
- For petroleum derivatives:

Chemical analysis for all individual compounds in a petroleum bulk product released to the environment is generally unrealistic due to the complexity of these mixtures and the laboratory expense. Determining the chemical composition of a petroleum release is further complicated by hydrodynamic, abiotic, and biotic processes that act on the release to change the chemical character.

The longer the release is exposed to the environment, the greater the change in chemical character and the harder it is to obtain accurate analytical results reflecting the identity of the release. After extensive weathering, detailed knowledge of the original bulk product is often less valuable than current site-specific information on a more focused set of hydrocarbon components. Health assessment efforts are frequently frustrated by three primary problems: (1) the inability to identify and quantify the individual compounds released to the environment as a consequence of a petroleum spill; (2) the lack of information characterizing the fate of the individual compounds in petroleum mixtures; and (3) the lack of specific health guidance values for the majority of chemicals present in petroleum products. To define the public health implications associated with exposure to petroleum hydrocarbons, it is necessary to have a basic understanding of petroleum properties, compositions, and the physical, chemical, biological, and toxicological properties of the compounds most often identified as the key chemicals of concern.

Environmental fate:

Petroleum products released to the environment migrate through soil via two general pathways: (1) as bulk oil flow infiltrating the soil under the forces of gravity and capillary action, and (2) as individual compounds separating from the bulk petroleum mixture and dissolving in air or water. When bulk oil flow occurs, it results in little or no separation of the individual compounds from the product mixture and the infiltration rate is usually fast relative to the dissolution rate. Many compounds that are insoluble and immobile in water are soluble in bulk oil and will migrate along with the bulk oil flow. Factors affecting the rate of bulk oil infiltration include soil moisture content, vegetation, terrain, climate, rate of release (e.g., catastrophic versus slow leakage), soil particle size (e.g., sand versus clay), and oil viscosity (e.g., gasoline versus motor oil).

As bulk oil migrates through the soil column, a small amount of the product mass is retained by soil particles. The bulk product retained by the soil particles is known as "residual saturation".

Depending upon the persistence of the bulk oil, residual saturation can potentially reside in the soil for years. Residual saturation is important as it determines the degree of soil contamination and can act as a continuing source of contamination for individual compounds to separate from the bulk product and migrate independently in air or groundwater. Residual saturation is important as it determines the degree of soil contamination and can act as a continuing source of contamination for individual compounds to separate from the bulk product and migrate independently in air or groundwater. When the amount of product released to the environment is small relative to the volume of available soil, all of the product is converted to residual saturation and downward migration of the bulk product usually ceases prior to affecting groundwater resources. Adverse impacts to groundwater may still occur if rain water infiltrates through soil containing residual saturation and initiates the downward migration of individual compounds. When the amount of product released is large relative to the volume of available soil, the downward migration of bulk product ceases as water-saturated pore spaces are encountered. If the density of the bulk product is less than that of water, the product tends to "float" along the interface between the water saturated and unsaturated zones and spread horizontally in a pancake-like layer, usually in the direction of groundwater flow. Almost all motor and heating oils are less dense than water. If the density of the bulk product is greater than that of water, the product will continue to migrate downward through the water table aquifer under the continued influence of gravity. Downward migration ceases when the product is converted to residual saturation or when an impermeable surface is encountered.

As the bulk product migrates through the soil column, individual compounds may separate from the mixture and migrate independently. Chemical transport properties such as volatility, solubility, and sorption potential are often used to evaluate and predict which compounds will likely separate from the mixture. Since petroleum products are complex mixtures of hundreds of compounds, the compounds characterized by relatively high vapor pressures tend to volatilise and enter the vapor phase. The exact composition of these vapors depends on the composition of the original product. Using gasoline as an example, compounds such as butane, propane, benzene, toluene, ethylbenzene and xylene are preferentially volatilised. Because volatility represents transfer of the compound from the product or liquid phase to the air phase, it is expected that the concentration of that compound in the product or liquid phase will decrease as the concentration in the air phase increases.

In general, compounds having a vapor pressure in excess of 10-2 mm Hg are more likely to be present in the air phase than in the liquid phase. Compounds characterized by vapor pressures less than 10-7 mm Hg are more likely to be associated with the liquid phase. Compounds possessing vapor pressures that are less than 10-2 mm Hg, but greater than 10-7 mm Hg, will have a tendency to exist in both the air and the liquid phases.

Lighter petroleum products such as gasoline contain constituents with higher water solubility and volatility and lower sorption potential than heavier petroleum products such as fuel oil.

Data compiled from gasoline spills and laboratory studies indicate that these light-fraction hydrocarbons tend to migrate readily through soil, potentially threatening or affecting groundwater supplies. In contrast, petroleum products with heavier molecular weight constituents, such as fuel oil, are generally more persistent in soils, due to their relatively low water solubility and volatility and high sorption capacity. Solubility generally decreases with increasing molecular weight of the hydrocarbon compounds. For compounds having similar molecular weights, the aromatic hydrocarbons are more water soluble and mobile in water than the aliphatic hydrocarbons and branched aliphatics are less water-soluble than straight-chained aliphatics. Aromatic compounds in petroleum fuels may comprise as much as 50% by weight; aromatic compounds in the C6-C13, range made up approximately 95% of the compounds dissolved in water.

Indigenous microbes found in many natural settings (e.g., soils, groundwater, ponds) have been shown to be capable of degrading organic compounds. Unlike other fate processes that disperse contaminants in the environment, biodegradation can eliminate the contaminants without transferring them across media.

127 UV WASH

Hazard Alert Code:
MODERATE

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

Issue Date: 13-Nov-2009

CD 2009/3

The final products of microbial degradation are carbon dioxide, water, and microbial biomass. The rate of hydrocarbon degradation depends on the chemical composition of the product released to the environment as well as site-specific environmental factors. Generally the straight chain hydrocarbons and the aromatics are degraded more readily than the highly branched aliphatic compounds. The n-alkanes, n-alkyl aromatics, and the aromatics in the C10-C22 range are the most readily biodegradable; n-alkanes, n-alkyl aromatics, and aromatics in the C5-C9 range are biodegradable at low concentrations by some microorganisms, but are generally preferentially removed by volatilisation and thus are unavailable in most environments; n-alkanes in the C1-C4 ranges are biodegradable only by a narrow range of specialized hydrocarbon degraders; and n-alkanes, n-alkyl aromatics, and aromatics above C22 are generally not available to degrading microorganisms. Hydrocarbons with condensed ring structures, such as PAHs with four or more rings, have been shown to be relatively resistant to biodegradation. PAHs with only 2 or 3 rings (e.g., naphthalene, anthracene) are more easily biodegraded. PAHs with only 2 or 3 rings (e.g., naphthalene, anthracene) are more easily biodegraded. A large proportion of the water-soluble fraction of the petroleum product may be degraded as the compounds go into solution. As a result, the remaining product may become enriched in the alicyclics, the highly branched aliphatics, and PAHs with many fused rings.

In almost all cases, the presence of oxygen is essential for effective biodegradation of oil. Anaerobic decomposition of petroleum hydrocarbons leads to extremely low rates of degradation. The ideal pH range to promote biodegradation is close to neutral (6-8). For most species, the optimal pH is slightly alkaline, that is, greater than 7. The moisture content of the contaminated soil will affect biodegradation of oils due to dissolution of the residual compounds, dispersive actions, and the need for microbial metabolism to sustain high activity. The moisture content in soil affects microbial locomotion, solute diffusion, substrate supply, and the removal of metabolic by-products. Biodegradation rates in soils are also affected by the volume of product released to the environment. At concentrations of 0.5% of oil by volume, the degradation rate in soil is fairly independent of oil concentrations. However, as oil concentration rises, the first order degradation rate decreases and the oil degradation half-life increases. Ultimately, when the oil reaches saturation conditions in the soil (i.e., 30-50% oil), biodegradation virtually ceases.

Excessive moisture will limit the gaseous supply of oxygen for enhanced decomposition of petroleum hydrocarbons. Most studies indicate that optimum moisture content is within 50-70% of the water holding capacity.

All biological transformations are affected by temperature. Generally, as the temperature increases, biological activity tends to increase up to a temperature where enzyme denaturation occurs. The presence of oil should increase soil temperature, particularly at the surface. The darker color increases the heat capacity by adsorbing more radiation. The optimal temperature for biodegradation to occur ranges from 18 C to 30 C. Minimum rates would be expected at 5 C or lower.

ETHYLENE GLYCOL MONOBUTYL ETHER:

■ Fish LC50 (96hr.) (mg/l):	1490
■ BCF<100:	0.4
■ log Kow (Prager 1995):	0.83
■ log Kow (Sangster 1997):	0.8
■ Half- life Soil - High (hours):	672
■ Half- life Soil - Low (hours):	168
■ Half- life Air - High (hours):	32.8
■ Half- life Air - Low (hours):	3.28
■ Half- life Surface water - High (hours):	672
■ Half- life Surface water - Low (hours):	168
■ Half- life Ground water - High (hours):	1344
■ Half- life Ground water - Low (hours):	336
■ Aqueous biodegradation - Aerobic - High (hours):	672
■ Aqueous biodegradation - Aerobic - Low (hours):	168
■ Aqueous biodegradation - Anaerobic - High (hours):	2688
■ Aqueous biodegradation - Anaerobic - Low (hours):	672
■ Photooxidation half- life air - High (hours):	32.8
■ Photooxidation half- life air - Low (hours):	3.28
■ Fish LC50 (96hr.) (mg/l):	1250- 1650
■ Daphnia magna EC50 (48hr.) (mg/l):	600- 1000

log Kow: 0.76-0.83

Koc: 67

Half-life (hr) air: 17

Henry's atm m³ /mol: 2.08E-08

BOD 5 if unstated: 0.71

COD: 2.2

Log BCF: 0.4

Fish LC50 (24 h): 983-1650 mg/L

Fish LC50 (96 h): fathead minnow 1700 mg/L **

Invertebrate toxicity:

cell mult. inhib.91-900mg/L

(Daphnia) 48h LC50: >1000 mg/L **

Bioaccumulation: not sig

Effects on algae and plankton: cell mult. inhib.35-900mg/L

Degradation Biological: rapid

processes Abiotic: no hydrol&photol,RxnOH* ** [Union Carbide]

PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER:

log Kow: -0.53

127 UV WASH

Hazard Alert Code:
MODERATE

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

Issue Date: 13-Nov-2009

CD 2009/3

Dissolves rapidly in water; the material is volatile and will partition to the air compartment.

Readily biodegradable.

Degradation by activated sludge in 29 days=90%

Not expected to bioaccumulate

Fish LC50 (96 h): fathead minnow >2000 mg/l

Ecotoxicity

Ingredient	Persistence: Water/Soil	Persistence: Air	Bioaccumulation	Mobility
127 UV Wash solvent naphtha petroleum, heavy aromatic ethylene glycol monobutyl ether propylene glycol monomethyl ether - alpha isomer	LOW	No data	LOW	HIGH
	LOW	No data	LOW	HIGH

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: 3Y (ADG7)

ADG7:

Class or division:

3

Subsidiary risk:

None

127 UV WASH

Hazard Alert Code:
MODERATE

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

Issue Date: 13-Nov-2009

CD 2009/3

UN No.:	3295	UN packing group:	III
Special provisions:	223	Packing Instructions:	None
Notes:	None	Limited quantities:	5 L
Portable tanks and bulk containers - Instructions:	T4	Portable tanks and bulk containers - Special provisions:	TP1, TP29
Packagings and IBCs - Packing instruction:	P001, IBC03, LP01	Packagings and IBCs - Special packing provisions:	None

Shipping Name:HYDROCARBONS, LIQUID, N.O.S. (contains solvent naphtha petroleum, heavy aromatic)
Land Transport UNDG:

Class or division:	3	Subsidiary risk:	None
UN No.:	3295	UN packing group:	III

Shipping Name:HYDROCARBONS, LIQUID, N.O.S. (contains solvent naphtha petroleum, heavy aromatic)

Air Transport IATA:

ICAO/IATA Class:	3	ICAO/IATA Subrisk:	☐
UN/ID Number:	3295	Packing Group:	III
Special provisions:	A3		

Shipping Name: HYDROCARBONS, LIQUID, N.O.S.(CONTAINS SOLVENT NAPHTHA PETROLEUM, HEAVY AROMATIC)

Maritime Transport IMDG:

IMDG Class:	3	IMDG Subrisk:	None
UN Number:	3295	Packing Group:	III
EMS Number:	F-E,S-D	Special provisions:	223 944
Limited Quantities:	5 L	Marine Pollutant:	Not Determined

Shipping Name: HYDROCARBONS, LIQUID, N.O.S.(contains solvent naphtha petroleum, heavy aromatic)

Section 15 - REGULATORY INFORMATION

POISONS SCHEDULE

S5

REGULATIONS

Regulations for ingredients

solvent naphtha petroleum, heavy aromatic (CAS: 64742-94-5) is found on the following regulatory lists;

"Australia Hazardous Substances","Australia High Volume Industrial Chemical List (HVICL)","Australia Inventory of Chemical Substances (AICS)","International Council of Chemical Associations (ICCA) - High Production Volume List","OECD Representative List of High Production Volume (HPV) Chemicals"

ethylene glycol monobutyl ether (CAS: 111-76-2) is found on the following regulatory lists;

"Australia Exposure Standards","Australia Hazardous Substances","Australia High Volume Industrial Chemical List (HVICL)","Australia Inventory of Chemical Substances (AICS)","IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances","International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs","OECD Representative List of High Production Volume (HPV) Chemicals"

propylene glycol monomethyl ether - alpha isomer (CAS: 107-98-2) is found on the following regulatory lists;

"Australia Exposure Standards","Australia Hazardous Substances","Australia Inventory of Chemical Substances (AICS)","IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk","International Council of Chemical Associations (ICCA) - High Production Volume List","OECD Representative List of High Production Volume (HPV) Chemicals"

No data for 127 UV Wash (CW: 21-9294)

Section 16 - OTHER INFORMATION

REPRODUCTIVE HEALTH GUIDELINES

Ingredient	ORG	UF	Endpoint	CR	Adeq TLV
ethylene glycol monobutyl ether	3.6 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:

127 UV WASH

Hazard Alert Code:
MODERATE

Chemwatch Material Safety Data Sheet (REVIEW)

Revision No: 2.0

Chemwatch 21-9294

Issue Date: 13-Nov-2009

CD 2009/3

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

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Issue Date: 13-Nov-2009

Print Date:18-Feb-2010