

Chemwatch Material Safety Data Sheets

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CAMPBELLS 145E ETCH & PROTECT PRIMER

Chemwatch Material Safety Data Sheet

Issue Date: 20-Nov-2008

NA317TC

CHEMWATCH 5093-95

Version No:4

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Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

PRODUCT NAME

CAMPBELLS 145E ETCH & PROTECT PRIMER

PROPER SHIPPING NAME

PAINT RELATED MATERIAL

PRODUCT USE

» Application is usually by spray atomisation.

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.

Single pack anticorrosive epoxy primer with etching ability. Provides adhesion to mild steel, aluminium and other difficult substrates

For small areas use a brush

SUPPLIER

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AUS

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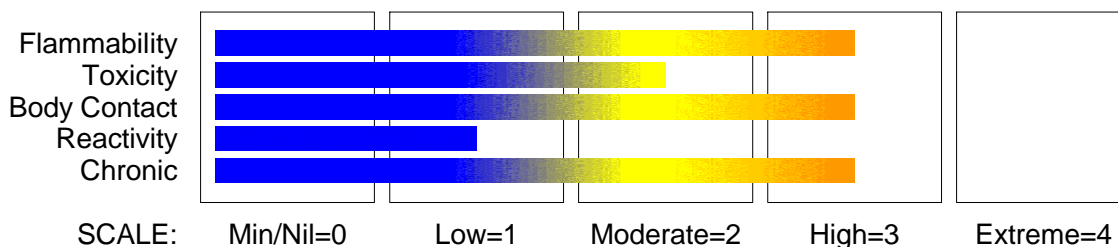
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Section 2 - HAZARDS IDENTIFICATION

STATEMENT OF HAZARDOUS NATURE

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

CHEMWATCH HAZARD RATINGS



continued...

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Section 2 - HAZARDS IDENTIFICATION

POISONS SCHEDULE

S5

- RISK**
- » Highly flammable.
 - » Harmful if swallowed.
 - » Irritating to skin.
 - » Risk of serious damage to eyes.
 - » May cause SENSITISATION by skin contact.

 - » Harmful: danger of serious damage to health by prolonged exposure through inhalation.
 - » Toxic to aquatic organisms may cause long-term adverse effects in the aquatic environment.
 - » Possible risk of harm to the unborn child.

 - » HARMFUL- May cause lung damage if swallowed.

 - » Vapours may cause drowsiness and dizziness.
- SAFETY**
- » Keep away from sources of ignition. No smoking.
 - » 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.
 - » If swallowed IMMEDIATELY contact Doctor or Poisons Information Centre. (show this container or label).
 - » Use appropriate container to avoid environmental contamination.
 - » Avoid release to the environment. Refer to special instructions/Safety data sheets.
 - » This material and its container must be disposed of as hazardous waste.

Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

NAME	CAS RN	%
isopropanol	67-63-0	30-60
toluene	108-88-3	10-30
titanium dioxide	13463-67-7	1-9
methyl ethyl ketone	78-93-3	1-9
bisphenol A/ epichlorohydrin resin	25068-38-6	1-9
xylene	1330-20-7	1-5
isobutanol	78-83-1	1-5
zinc phosphate	7779-90-0	1-9
ethylene glycol monobutyl ether	111-76-2	1-5
phenol/ formaldehyde resin	9003-35-4	1-5
additives		1-9

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.

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Section 4 - FIRST AID MEASURES

- Avoid giving milk or oils.
- Avoid giving alcohol.

EYE

» If this product comes in contact with the eyes:

- Immediately hold eyelids apart and flush the eye continuously with 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.
- Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.
- Transport to hospital or doctor without delay.
- 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

» 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.

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 isopropanol:

- Rapid onset respiratory depression and hypotension indicates serious ingestions that require careful cardiac and respiratory monitoring together with immediate intravenous access.
- Rapid absorption precludes the usefulness of emesis or lavage 2 hours post-ingestion. Activated charcoal and cathartics are not clinically useful. Ipecac is most useful when given 30 mins. post-ingestion.
- There are no antidotes.

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Section 4 - FIRST AID MEASURES

- Management is supportive. Treat hypotension with fluids followed by vasopressors.
- Watch closely, within the first few hours for respiratory depression; follow arterial blood gases and tidal volumes.
- Ice water lavage and serial haemoglobin levels are indicated for those patients with evidence of gastrointestinal bleeding.

Following acute or short term repeated exposures to toluene:

- Toluene is absorbed across the alveolar barrier, the blood/air mixture being 11.2/15.6 (at 37 degrees C.) The concentration of toluene, in expired breath, is of the order of 18 ppm following sustained exposure to 100 ppm. The tissue/blood proportion is 1/3 except in adipose where the proportion is 8/10.
- Metabolism by microsomal mono-oxygenation, results in the production of hippuric acid. This may be detected in the urine in amounts between 0.5 and 2.5 g/24 hr which represents, on average 0.8 gm/gm of creatinine. The biological half-life of hippuric acid is in the order of 1-2 hours.
- Primary threat to life from ingestion and/or inhalation is respiratory failure.
- Patients should be quickly evaluated for signs of respiratory distress (eg cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO₂ <50 mm Hg or pCO₂ > 50 mm Hg) should be intubated.
- Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial damage 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 (adrenaline) 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.

BIOLOGICAL EXPOSURE INDEX - BEI

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

Determinant	Index	Sampling Time	Comments
o- Cresol in urine	0.5 mg/L	End of shift	B
Hippuric acid in urine	1.6 g/g creatinine	End of shift	B, NS
Toluene in blood	0.05 mg/L	Prior to last shift of workweek	

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

B: Background levels occur in specimens collected from subjects NOT exposed.

Section 5 - FIRE FIGHTING MEASURES

EXTINGUISHING MEDIA

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

FIRE FIGHTING

- Alert Fire Brigade and tell them location and nature of hazard.
- May be violently or explosively reactive.
- Wear breathing apparatus plus protective gloves.
- Prevent, by any means available, spillage from entering drains or water course.
- Consider evacuation (or protect in place).
- Fight fire from a safe distance, with adequate cover.

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Section 5 - FIRE FIGHTING MEASURES

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

FIRE/EXPLOSION HAZARD

- Liquid and vapour are highly flammable.
- Severe fire hazard when exposed to heat, flame and/or oxidisers.
- Vapour may travel a considerable distance to source of ignition.
- Heating may cause expansion or decomposition leading to violent rupture of containers.
- On combustion, may emit toxic fumes of carbon monoxide (CO).

Combustion products include:

carbon dioxide (CO₂).

aldehydes.

phosphorus oxides (PO_x).

metal oxides.

other pyrolysis products typical of burning organic material.

Contains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions.

FIRE INCOMPATIBILITY

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

HAZCHEM: None

Personal Protective Equipment

Gas tight chemical resistant suit.

Section 6 - ACCIDENTAL RELEASE MEASURES

EMERGENCY PROCEDURES

MINOR SPILLS

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

MAJOR SPILLS

- 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.

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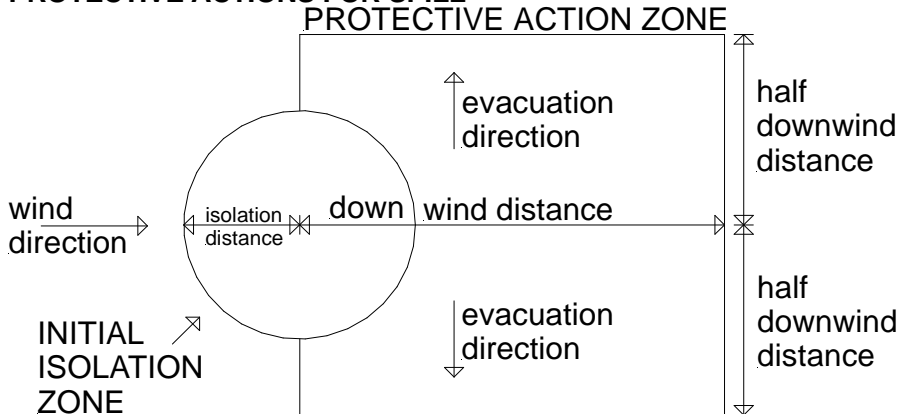
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- Collect recoverable product into labelled containers for recycling.
- Absorb remaining product with sand, earth or vermiculite.
- Collect solid residues and seal in labelled drums for disposal.
- Wash area and prevent runoff into drains.
- If contamination of drains or waterways occurs, advise emergency services.

PROTECTIVE ACTIONS FOR SPILL



From IERG (Canada/Australia)

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

FOOTNOTES

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

EMERGENCY RESPONSE PLANNING GUIDELINES (ERPG)

The maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to one hour WITHOUT experiencing or developing

life-threatening health effects is:
toluene 1000ppm

irreversible or other serious effects or symptoms which could impair an individual's ability to take protective action is:
toluene 300ppm

other than mild, transient adverse effects without perceiving a clearly defined odour is:

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toluene 50ppm

American Industrial Hygiene Association (AIHA)

Ingredients considered according to the following cutoffs

Very Toxic (T+)	>= 0.1%	Toxic (T)	>= 3.0%
R50	>= 0.25%	Corrosive (C)	>= 5.0%
R51	>= 2.5%		
else	>= 10%		

where percentage is percentage of ingredient found in the mixture

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

Section 7 - HANDLING AND STORAGE

PROCEDURE FOR HANDLING

» 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.
- Avoid all personal contact, including inhalation.
- Wear protective clothing when risk of exposure occurs.
- Use in a well-ventilated area.
- Prevent concentration in hollows and sumps.
- DO NOT enter confined spaces until atmosphere has been checked.
- Avoid smoking, naked lights, heat or ignition sources.
- When handling, DO NOT eat, drink or smoke.
- Vapour may ignite on pumping or pouring due to static electricity.
- DO NOT use plastic buckets.
- Earth and secure metal containers when dispensing or pouring product.
- Use spark-free tools when handling.
- Avoid contact with incompatible materials.
- Keep containers securely sealed.
- Avoid physical damage to containers.
- Always wash hands with soap and water after handling.
- Work clothes should be laundered separately.
- Use good occupational work practice.
- Observe manufacturer's storing and handling recommendations.
- Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
- DO NOT allow clothing wet with material to stay in contact with skin.

SUITABLE CONTAINER

- DO NOT use aluminium or galvanised containers.
 - 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.

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Section 7 - HANDLING AND STORAGE

- 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 oxidising agents, acids, acid chlorides, acid anhydrides, chloroformates.

*

STORAGE REQUIREMENTS

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

Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

EXPOSURE CONTROLS

Source	Material	TWA ppm	TWA mg/m ³	STEL ppm	STEL mg/m ³
Australia Exposure Standards	isopropanol (Isopropyl alcohol)	400	983	500	1230
Australia Exposure Standards	toluene (Toluene)	50	191	150	574
Australia Exposure Standards	titanium dioxide (Titanium dioxide (a))		10		
Australia Exposure Standards	methyl ethyl ketone (Methyl ethyl ketone (MEK))	150	445	300	890
Australia Exposure Standards	xylene (Xylene (o-, m-, p-isomers))	80	350	150	655
Australia Exposure Standards	isobutanol (Isobutyl alcohol)	50	152		
Australia Exposure Standards	zinc phosphate (Inspirable dust (not otherwise classified))		10		
Australia Exposure Standards	ethylene glycol monobutyl ether (2-Butoxyethanol)	20	96.9	50	242

The following materials had no OELs on our records

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Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

- bisphenol A/ epichlorohydrin resin: CAS:25068- 38- 6
- phenol/ formaldehyde resin: CAS:9003- 35- 4

EMERGENCY EXPOSURE LIMITS

Material	Revised IDLH Value (mg/m3)	Revised IDLH Value (ppm)
isopropanol		2, 000 [LEL]
toluene		500
titanium dioxide	5, 000	
methyl ethyl ketone		3, 000 [Unch]
xylene		900
isobutanol		1, 600
ethylene glycol monobutyl ether		700 [Unch]

NOTES

Values marked LEL indicate that the IDLH was based on 10% of the lower explosive limit for safety considerations even though the relevant toxicological data indicated that irreversible health effects or impairment of escape existed only at higher concentrations.

MATERIAL DATA

» Not available. Refer to individual constituents.

INGREDIENT DATA

ETHYLENE GLYCOL MONOBUTYL ETHER:

METHYL ETHYL KETONE:

TOLUENE:

XYLENE:

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

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

TLV believed to be adequate to protect reproductive health:

LOD: Limit of detection

Toxic endpoints have also been identified as:

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

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

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

BISPHENOL A/ EPICHLOROHYDRIN RESIN:

ISOPROPANOL:

TITANIUM DIOXIDE:

ZINC PHOSPHATE:

» 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

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- acclimate the worker to the irritant warning properties of these substances thus increasing the risk of overexposure.

BISPHENOL A/ EPICHLOROHYDRIN RESIN:

TITANIUM DIOXIDE:

ZINC PHOSPHATE:

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

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

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

ETHYLENE GLYCOL MONOBUTYL ETHER:

TOLUENE:

XYLENE:

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

TOLUENE:

XYLENE:

- » Exposure limits with "skin" notation indicate that vapour and liquid may be absorbed through intact skin. Absorption by skin may readily exceed vapour inhalation exposure. Symptoms for skin absorption are the same as for inhalation. Contact with eyes and mucous membranes may also contribute to overall exposure and may also invalidate the exposure standard.

ISOPROPANOL:

- » Odour Threshold Value: 3.3 ppm (detection), 7.6 ppm (recognition)

Exposure at or below the recommended isopropanol TLV-TWA and STEL is thought to minimise the potential for inducing narcotic effects or significant irritation of the eyes or upper respiratory tract. It is believed, in the absence of hard evidence, that this limit also provides protection against the development of chronic health effects. The limit is intermediate to that set for ethanol, which is less toxic, and n-propyl alcohol, which is more toxic, than isopropanol.

TOLUENE:

- » For toluene:

Odour Threshold Value: 0.16-6.7 (detection), 1.9-69 (recognition)

NOTE: Detector tubes measuring in excess of 5 ppm, are available.

High concentrations of toluene in the air produce depression of the central nervous system (CNS) in humans. Intentional toluene exposure (glue-sniffing) at maternally-intoxicating concentration has also produced birth defects. Foetotoxicity appears at levels associated with CNS narcosis and probably occurs only in those with chronic toluene-induced kidney failure. Exposure at or below the recommended TLV-TWA is thought to prevent transient headache and irritation, to provide a measure of safety for possible disturbances to human reproduction, the prevention of reductions in cognitive responses reported amongst humans inhaling greater than 40 ppm, and the significant risks of hepatotoxic, behavioural and nervous system effects (including impaired reaction time and incoordination). Although toluene/ethanol interactions are well recognised, the degree of protection afforded by the TLV-TWA among drinkers is not known.

Odour Safety Factor(OSF)

OSF=17 (TOLUENE).

TITANIUM DIOXIDE:

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

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

METHYL ETHYL KETONE:

Odour Threshold Value: Variously reported as 2 ppm and 4.8 ppm

Odour threshold: 2 ppm (detection); 5 ppm (recognition)
25 ppm (easy recognition); 300 ppm IRRITATING

Exposures at or below the recommended TLV-TWA are thought to prevent injurious systemic effects and to minimise objections to odour and irritation. Where synergism or potentiation may occur stringent control of the primary toxin (e.g. n-hexane or methyl butyl ketone) is desirable and additional consideration should be given to lowering MEK exposures.

XYLENE:

» for xylenes:

IDLH Level: 900 ppm

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

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

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

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

Odour Safety Factor(OSF)

OSF=4 (XYLENE).

ISOBUTANOL:

Odour Threshold Value: 0.66-40 ppm (detection), 1.8-53 ppm (recognition)

Although there do not appear to be reports of isobutyl alcohol causing auditory impairment or vestibular damage in humans (as with n-butanol) the recommended TLV-TWA recognises the slightly greater acute toxic potential of isobutanol versus n-butanol. Exposure at or below this limit is thought to significantly reduce the risk of skin irritation.

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).

PHENOL/ FORMALDEHYDE RESIN:

» Odour Threshold Value for phenol: 0.060 ppm (detection)

NOTE: Detector tubes for phenol, measuring in excess of 1 ppm, are commercially available.

Systemic absorption by all routes may induce convulsions with damage to the lungs and central nervous system.

Exposure at or below the recommended TLV-TWA is thought to protect the worker from respiratory, cardiovascular, hepatic, renal and neurological toxicity. Workers or volunteers exposed at or below 5.2 ppm phenol have experienced no ill-effects. Because phenol as a vapour, liquid or solid can penetrate the skin causing systemic effects, a skin notation is considered necessary. Although ACGIH has not recommended a STEL it is felt that ACGIH excursion limits (15 ppm limited to a total duration of 30 minutes with brief excursions limited to no more than 25 ppm) and NIOSH Ceiling values are sufficiently similar

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so as to provide the same margin of safety.

Odour Safety Factor(OSF)

OSF=25 (PHENOL).

Odour Threshold Value for formaldehyde: 0.98 ppm (recognition)

NOTE: Detector tubes for formaldehyde, measuring in excess of 0.2 ppm are available commercially.

Formaldehyde vapour exposure:

Primary irritation is dependent on duration of exposure and individual susceptibility.

The following are typical symptoms encountered at various exposure levels.

0.1 ppm - Lower level of mucous eye, nose and throat irritation

0.8 ppm - Typical threshold of perception

1-2 ppm - Typical threshold of irritation

2-3 ppm - Irritation of eyes, nose and throat

4-5 ppm - Increased irritation, tearing, headache, pungent odour

10-20 ppm - Profuse tearing, severe burning, coughing

50 ppm - Serious bronchial and alveolar damage

100 ppm - Formaldehyde induced chemical pneumonia and death

Despite the intent of the TLV Ceiling recommendation it is believed that 0.3 ppm will not protect that portion of the workforce (up to 20%) reported to be responsive to low ambient concentrations. Because of the dose-related carcinogenic activity for rat and mouse inhalation of formaldehyde, the report of macromolecular adducts in the upper and lower respiratory tracts of nonhuman primates following inhalation of formaldehyde, the human case reports of upper respiratory tract malignant melanoma associated with

formaldehyde inhalation and the suggestive epidemiologic data on human cancer risk, the TLV Committee recommends that workplace formaldehyde air concentrations be reduced to the lowest possible levels that can be achieved using engineering controls.

Odour Safety Factor(OSF)

OSF=0.36 (FORMALDEHYDE).

PERSONAL PROTECTION

EYE

- Safety glasses with side shields.
- Chemical goggles.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lens or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].

HANDS/FEET

- When handling liquid-grade epoxy resins wear chemically protective gloves (e.g nitrile or nitrile-butadiene rubber), boots and aprons.
- DO NOT use cotton or leather (which absorb and concentrate the resin), polyvinyl chloride, rubber or polyethylene gloves (which absorb the resin).
- DO NOT use barrier creams containing emulsified fats and oils as these may absorb the resin; silicone-based barrier creams should be reviewed prior to use.

NOTE:

- The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.

OTHER

- Overalls.
- PVC Apron.
- PVC protective suit may be required if exposure severe.
- Eyewash unit.

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- Ensure there is ready access to a safety shower.

RESPIRATOR

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

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

* - Continuous Flow

** - Continuous-flow or positive pressure demand.

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

ENGINEERING CONTROLS

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

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

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

Within each range the appropriate value depends on:

Lower end of the range

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

Upper end of the range

- 1: Disturbing room air currents
- 2: Contaminants of high toxicity
- 3: High production, heavy use
- 4: Small hood- local control only

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

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air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

APPEARANCE

Coloured flammable liquid with a solvent odour; not miscible with water.

PHYSICAL PROPERTIES

Liquid.

Does not mix with water.

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

Boiling Range (°C): 99- 143
Specific Gravity (water= 1): 0.97- 1.01
pH (as supplied): Not Applicable
Vapour Pressure (kPa): Not Available
Evaporation Rate: Not Available
Flash Point (°C): 0
Upper Explosive Limit (%): Not Available
Decomposition Temp (°C): Not Available
Viscosity: Not Available

Section 10 - CHEMICAL STABILITY AND REACTIVITY INFORMATION

CONDITIONS CONTRIBUTING TO INSTABILITY

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

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

Section 11 - TOXICOLOGICAL INFORMATION

POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

SWALLOWED

» Effects on the nervous system characterise over-exposure to higher aliphatic alcohols. These include headache, muscle weakness, giddiness, ataxia, (loss of muscle coordination), confusion, delirium and coma. Gastrointestinal effects may include nausea, vomiting and diarrhoea. In the absence of effective treatment, respiratory arrest is the most common cause of death in animals acutely poisoned by the higher alcohols. Aspiration of liquid alcohols produces an especially toxic response as they are able to penetrate deeply in the lung where they are absorbed and may produce pulmonary injury. Those possessing lower viscosity elicit a greater response. The result is a high blood level and prompt death at doses otherwise tolerated by ingestion without aspiration. In general the secondary alcohols are less toxic than the corresponding primary isomers. As a general observation, alcohols are more powerful central nervous system depressants than their aliphatic analogues. In sequence of decreasing depressant potential, tertiary alcohols with multiple substituent OH groups are more potent than secondary alcohols, which, in turn, are more potent than primary alcohols. The potential for overall systemic toxicity increases with molecular weight (up to C7), principally because the water solubility is diminished and lipophilicity is increased.

Within the homologous series of aliphatic alcohols, narcotic potency may increase even faster than lethality. Only scanty toxicity information is available about higher homologues of the aliphatic alcohol series (greater than C7) but animal data establish that lethality does not continue to increase with increasing chain length. Aliphatic alcohols with 8 carbons are less toxic than those immediately preceding them in the series. 10 -Carbon n-decyl alcohol has low toxicity as do the solid fatty alcohols (e.g. lauryl, myristyl, cetyl and stearyl). However the rat

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aspiration test suggests that decyl and melted dodecyl (lauryl) alcohols are dangerous if they enter the trachea. In the rat even a small quantity (0.2 ml) of these behaves like a hydrocarbon solvent in causing death from pulmonary oedema.

Primary alcohols are metabolised to corresponding aldehydes and acids; a significant metabolic acidosis may occur. Secondary alcohols are converted to ketones, which are also central nervous system depressants and which, in the case of the higher homologues persist in the blood for many hours. Tertiary alcohols are metabolised slowly and incompletely so their toxic effects are generally persistent.

Considered an unlikely route of entry in commercial/industrial environments. The liquid may produce gastrointestinal discomfort and may be harmful if swallowed. Ingestion may result in nausea, pain and vomiting. Vomit entering the lungs by aspiration may cause potentially lethal chemical pneumonitis.

Central nervous system (CNS) depression may include nonspecific 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.

EYE

» When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.

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

SKIN

» The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either

- produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or
- produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period.

Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.

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

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

INHALED

» Exposure to aliphatic alcohols with more than 3 carbons may produce central nervous system effects such as headache, dizziness, drowsiness, muscle weakness, delirium, CNS depression, coma, seizure, and neurobehavioural changes. Symptoms are more acute with higher alcohols. Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, respiratory depression secondary to CNS depression, pulmonary oedema, chemical pneumonitis and bronchitis. Cardiovascular involvement may result in arrhythmias and hypotension. Gastrointestinal effects may include nausea and vomiting. Kidney and liver damage may result following massive exposures. The alcohols are potential irritants being, generally, stronger irritants than similar organic structures that lack functional groups (e.g. alkanes) but are much less irritating than the corresponding amines, aldehydes or ketones. Alcohols and glycols (diols) rarely represent serious hazards in the workplace, because their vapour concentrations are usually less than the levels which produce significant irritation which, in turn, produce significant central nervous system effects as well.

CHRONIC HEALTH EFFECTS

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

Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis). Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS].

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Chronic toluene habituation occurs following intentional abuse (glue sniffing) or from occupational exposure. Ataxia, incoordination and tremors of the hands and feet (as a consequence of diffuse cerebral atrophy), headache, abnormal speech, transient memory loss, convulsions, coma, drowsiness, reduced colour perception, frank blindness, nystagmus (rapid, involuntary eye-movements), hearing loss leading to deafness and mild dementia have all been associated with chronic abuse. Peripheral nerve damage, encephalopathy, giant axonopathy electrolyte disturbances in the cerebrospinal fluid and abnormal computer tomographic (CT scans) are common amongst toluene addicts. Although toluene abuse has been linked with kidney disease, this does not commonly appear in cases of occupational toluene exposures. Cardiac and haematological toxicity are however associated with chronic toluene exposures. Cardiac arrhythmia, multifocal and premature ventricular contractions and supraventricular tachycardia are present in 20% of patients who abused toluene-containing paints. Previous suggestions that chronic toluene inhalation produced human peripheral neuropathy have been discounted. However central nervous system (CNS) depression is well documented when blood toluene exceeds 2.2 mg%. Toluene abusers can achieve transient circulating concentrations of 6.5 mg%. Amongst workers exposed for a median time of 29 years, to toluene, no subacute effects on neurasthenic complaints and psychometric test results could be established. The prenatal toxicity of very high toluene concentrations has been documented for several animal species and man. Malformations indicative of specific teratogenicity have not generally been found. Neonatal toxicity, described in the literature, takes the form of embryo death or delayed foetal growth and delayed skeletal system development. Permanent damage of children has been seen only when mothers have suffered from chronic intoxication as a result of "sniffing".

One of the constituents of the product has produced skin sensitisation reactions in either experimental animals and/or humans. Such reactions may be manifested as a localised reddening and/or urticaria (a hive-like asthma-like symptoms (shortness of breath, difficult breathing) and/or rhinitis (runny nose). This finding, however, remains speculative as the constituent has not been shown to raise specific antibodies in the blood in the same way as other confirmed allergens. The finding may also be confined to certain hypersensitive (atopic) individuals who show heightened reactions to other allergens such as pollen.

TOXICITY AND IRRITATION

» Not available. Refer to individual constituents.

ISOPROPANOL:

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

TOXICITY

Oral (human) LDLo: 3570 mg/kg

Oral (human) TDLo: 223 mg/kg

Oral (man) TDLo: 14432 mg/kg

Oral (rat) LD50: 5045 mg/kg

Dermal (rabbit) LD50: 12800 mg/kg

» The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

The substance is classified by IARC as Group 3:

NOT classifiable as to its carcinogenicity to humans.

Evidence of carcinogenicity may be inadequate or limited in animal testing.

IRRITATION

Skin (rabbit): 500 mg - Mild

Eye (rabbit): 10 mg - Moderate

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

Eye (rabbit): 100 mg - SEVERE

TOLUENE:

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

TOXICITY

Oral (human) LDLo: 50 mg/kg

Oral (rat) LD50: 636 mg/kg

Inhalation (human) TCLo: 100 ppm

Inhalation (man) TCLo: 200 ppm

Inhalation (rat) LC50: >26700 ppm/1h

Dermal (rabbit) LD50: 12124 mg/kg

» The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

IRRITATION

Skin (rabbit):20 mg/24h- Moderate

Skin (rabbit):500 mg - Moderate

Eye (rabbit):0.87 mg - Mild

Eye (rabbit): 2mg/24h - SEVERE

Eye (rabbit):100 mg/30sec - Mild

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For toluene:

Acute Toxicity

Humans exposed to intermediate to high levels of toluene for short periods of time experience adverse central nervous system effects ranging from headaches to intoxication, convulsions, narcosis, and death. Similar effects are observed in short-term animal studies.

Humans - Toluene ingestion or inhalation can result in severe central nervous system depression, and in large doses, can act as a narcotic. The ingestion of about 60 mL resulted in fatal nervous system depression within 30 minutes in one reported case.

Constriction and necrosis of myocardial fibers, markedly swollen liver, congestion and haemorrhage of the lungs and acute tubular necrosis were found on autopsy.

Central nervous system effects (headaches, dizziness, intoxication) and eye irritation occurred following inhalation exposure to 100 ppm toluene 6 hours/day for 4 days.

Exposure to 600 ppm for 8 hours resulted in the same and more serious symptoms including euphoria, dilated pupils, convulsions, and nausea. Exposure to 10,000-30,000 ppm has been reported to cause narcosis and death

Toluene can also strip the skin of lipids causing dermatitis

Animals - The initial effects are instability and incoordination, lachrymation and sniffles (respiratory exposure), followed by narcosis. Animals die of respiratory failure from severe nervous system depression. Cloudy swelling of the kidneys was reported in rats following inhalation exposure to 1600 ppm, 18-20 hours/day for 3 days

Subchronic/Chronic Effects:

Repeat doses of toluene cause adverse central nervous system effects and can damage the upper respiratory system, the liver, and the kidney. Adverse effects occur as a result from both oral and the inhalation exposures. A reported lowest-observed-effect level in humans for adverse neurobehavioral effects is 88 ppm.

Humans - Chronic occupational exposure and incidences of toluene abuse have resulted in hepatomegaly and liver function changes. It has also resulted in nephrotoxicity and, in one case, was a cardiac sensitiser and fatal cardiotoxin.

Neural and cerebellar dystrophy were reported in several cases of habitual "glue sniffing." An epidemiological study in France on workers chronically exposed to toluene fumes reported leukopenia and neutropenia. Exposure levels were not given in the secondary reference; however, the average urinary excretion of hippuric acid, a metabolite of toluene, was given as 4 g/L compared to a normal level of 0.6 g/L

Animals - The major target organs for the subchronic/chronic toxicity of toluene are the nervous system, liver, and kidney. Depressed immune response has been reported in male mice given doses of 105 mg/kg/day for 28 days. Toluene in corn oil administered to F344 male and female rats by gavage 5 days/week for 13 weeks, induced prostration, hypoactivity, ataxia, piloerection, lachrymation, excess salivation, and body tremors at doses 2500 mg/kg. Liver, kidney, and heart weights were also increased at this dose and histopathologic lesions were seen in the liver, kidneys, brain and urinary bladder. The no-observed-adverse effect level (NOAEL) for the study was 312 mg/kg (223 mg/kg/day) and the lowest-observed-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg/day).

Developmental/Reproductive Toxicity

Exposures to high levels of toluene can result in adverse effects in the developing human foetus. Several studies have indicated that high levels of toluene can also adversely effect the developing offspring in laboratory animals.

Humans - Variable growth, microcephaly, CNS dysfunction, attentional deficits, minor craniofacial and limb abnormalities, and developmental delay were seen in three children exposed to toluene in utero as a result of maternal solvent abuse before and during pregnancy

Animals - Sternebral alterations, extra ribs, and missing tails were reported following treatment of rats with 1500 mg/m³ toluene 24 hours/day during days 9-14 of gestation. Two of the dams died during the exposure. Another group of rats received 1000 mg/m³ 8 hours/day during days 1-21 of gestation. No maternal deaths or toxicity occurred, however, minor skeletal retardation was present in the exposed fetuses. CFLP

Mice were exposed to 500 or 1500 mg/m³ toluene continuously during days 6-13 of pregnancy. All dams died at the high dose during the first 24 hours of exposure, however none died at 500 mg/m³. Decreased foetal weight was reported, but there were no differences in the incidences of skeletal malformations or anomalies between the treated and control offspring.

Absorption - Studies in humans and animals have demonstrated that toluene is readily absorbed via the lungs and the gastrointestinal tract. Absorption through the skin is estimated at about 1% of that absorbed by the lungs when exposed to toluene vapor.

Dermal absorption is expected to be higher upon exposure to the liquid; however, exposure is limited by the

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rapid evaporation of toluene .

Distribution - In studies with mice exposed to radiolabeled toluene by inhalation, high levels of radioactivity were present in body fat, bone marrow, spinal nerves, spinal cord, and brain white matter. Lower levels of radioactivity were present in blood, kidney, and liver. Accumulation of toluene has generally been found in adipose tissue, other tissues with high fat content, and in highly vascularised tissues .

Metabolism - The metabolites of inhaled or ingested toluene include benzyl alcohol resulting from the hydroxylation of the methyl group. Further oxidation results in the formation of benzaldehyde and benzoic acid. The latter is conjugated with glycine to yield hippuric acid or reacted with glucuronic acid to form benzoyl glucuronide. o-cresol and p-cresol formed by ring hydroxylation are considered minor metabolites

Excretion - Toluene is primarily (60-70%) excreted through the urine as hippuric acid. The excretion of benzoyl glucuronide accounts for 10-20%, and excretion of unchanged toluene through the lungs also accounts for 10-20%. Excretion of hippuric acid is usually complete within 24 hours after exposure.

TITANIUM DIOXIDE:

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

TOXICITY

Oral (Rat) LD50: >20000 mg/kg *

Oral (Mouse) LD50: >10000 mg/kg *

» The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

For titanium dioxide:

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

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

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

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

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

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

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

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

Animal carcinogenicity data

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

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

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

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

* IUCLID

METHYL ETHYL KETONE:

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

TOXICITY

Oral (rat) LD50: 2737 mg/kg

Inhalation (human) TClO: 100 ppm/5 m

Inhalation (rat) LD50: 23500 mg/m³/8 hr

Dermal (rabbit) LD50: 6480 mg/kg

Inhalation (man) TClO: 10 mg/m³/6 hr - Mild

Inhalation (rat) LC50: 50100 mg/m³/8 hr

Dermal (rabbit) LD50: 20000 mg/kg

IRRITATION

Eye (human): 350 ppm - Irritant

Eye (rabbit): 80 mg - Irritant

Skin (rabbit): 402 mg/24 hr - Mild

Skin (rabbit): 13.78mg/24 hr Open

BISPHENOL A/ EPICHLOROHYDRIN RESIN:

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

TOXICITY

Oral (rat) LD50: 13600 mg/kg

Oral (rat) LD50: 11400 mg/kg

Intraperitoneal (rat) LD50: 2400 mg/kg

Oral (mouse) LD50: 15600 mg/kg

Intraperitoneal (mouse) LD50: 4000 mg/kg

» Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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

continued...

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dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

for RTECS No: SL 6475000:

(liquid grade)

Equivocal tumourigen by RTECS criteria

Somnolence, dyspnea, peritonitis

XYLENE:

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

TOXICITY

Oral (human) LDLo: 50 mg/kg

Oral (rat) LD50: 4300 mg/kg

Inhalation (human) TClO: 200 ppm

Inhalation (man) LClO: 10000 ppm/6h

Inhalation (rat) LC50: 5000 ppm/4h

Oral (Human) LD: 50 mg/kg

Inhalation (Human) TClO: 200 ppm/4h

Intraperitoneal (Rat) LD50: 2459 mg/kg

Subcutaneous (Rat) LD50: 1700 mg/kg

Oral (Mouse) LD50: 2119 mg/kg

Intraperitoneal (Mouse) LD50: 1548 mg/kg

Intravenous (Rabbit) LD: 129 mg/kg

Inhalation (Guinea) pig: LC 450 ppm/4h

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

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

The substance is classified by IARC as Group 3:

NOT classifiable as to its carcinogenicity to humans.

Evidence of carcinogenicity may be inadequate or limited in animal testing.

Reproductive effector in rats

IRRITATION

Skin (rabbit):500 mg/24h Moderate

Eye (human): 200 ppm Irritant

Eye (rabbit): 87 mg Mild

Eye (rabbit): 5 mg/24h SEVERE

ISOBUTANOL:

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

TOXICITY

Oral (rat) LD50: 2460 mg/kg.

Dermal (rabbit) LD50: 3400 mg/kg.

IRRITATION

Skin (rabbit): mg (open)- SEVERE

Eye (rabbit): 2 20 mg/24h- Moderate

Eye (rabbit): 2 mg/24h - SEVERE

ZINC PHOSPHATE:

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

TOXICITY

Oral (rat) LD50: 15000 mg/kg

IRRITATION

ETHYLENE GLYCOL MONOBUTYL ETHER:

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

TOXICITY

Oral (rat) LD50: 470 mg/kg

Dermal (rabbit) LD50: 220 mg/kg

Inhalation (human) TClO: 100 ppm

Inhalation (human) TClO: 195 ppm/8h * [Union Carbide]

Inhalation (Rat) LC50: 450 ppm *

IRRITATION

Skin (rabbit): 500 mg, open; Mild

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

Eye (rabbit): 100 mg SEVERE

continued...

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

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

For ethylene glycol:

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

Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol

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

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

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

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

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

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

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

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

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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 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

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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 cytogenicity 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 this substance by all routes.

exposed to high concentrations

PHENOL/ FORMALDEHYDE RESIN:

continued...

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

TOXICITY

Oral (rat) LD50: >2500 mg/kg

Dermal (rabbit) LD50: >5000 mg/kg

IRRITATION

Skin (rabbit): 3/8 - Moderate - Draize

Eye(rabbit):40/110 Moderate - Draize

[Manufacturer Mon]

» Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

CARCINOGEN

isopropanol	International Agency for Research on Cancer (IARC) Carcinogens	Group	3
toluene	International Agency for Research on Cancer (IARC) Carcinogens	Group	3
titanium dioxide	International Agency for Research on Cancer (IARC) Carcinogens	Group	2B
xylene	International Agency for Research on Cancer (IARC) Carcinogens	Group	3
ethylene glycol monobutyl ether	International Agency for Research on Cancer (IARC) Carcinogens	Group	3

REPROTOXIN

toluene	ILO Chemicals in the electronics industry that have toxic effects on reproduction	Reduced fertility or sterility
methyl ethyl ketone	ILO Chemicals in the electronics industry that have toxic effects on reproduction	Reduced fertility or sterility
xylene	ILO Chemicals in the electronics industry that have toxic effects on reproduction	Reduced fertility or sterility

SENSITISER

phenol/ formaldehyde resin	Australia Final Report on Hazard Classification of Common Skin Sensitisers	Recommended for Hazard Classification (R43)	No
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SKIN

toluene	Australia Exposure Standards - Skin	Notes	Sk
ethylene glycol monobutyl ether	Australia Exposure Standards - Skin	Notes	Sk

continued...

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Section 12 - ECOLOGICAL INFORMATION

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

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

Avoid release to the environment.

Refer to special instructions/ safety data sheets.

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.

Section 14 - TRANSPORTATION INFORMATION

Labels Required: FLAMMABLE LIQUID

HAZCHEM: 3[Y]E (ADG6)

UNDG:

Dangerous Goods Class: 3

Subrisk: None

UN Number: 1263

Packing Group: II

Shipping Name: PAINT RELATED MATERIAL (including paint thinning or reducing compound)

Air Transport IATA:

ICAO/IATA Class: 3

ICAO/IATA Subrisk: None

UN/ID Number: 1263

Packing Group: II

Special provisions: A3 A72

Shipping name: PAINT RELATED MATERIAL

Maritime Transport IMDG:

IMDG Class: 3

IMDG Subrisk: None

UN Number: 1263

Packing Group: II

EMS Number: F- E, S- E

Special provisions: 163 944

Limited Quantities: 5 L

Marine Pollutant: Not Determined

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

Section 15 - REGULATORY INFORMATION

POISONS SCHEDULE: S5

REGULATIONS

Campbells 145E Etch & Protect Primer (CAS: None):
No regulations applicable

continued...

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Section 15 - REGULATORY INFORMATION

isopropanol (CAS: 67-63-0) 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)
- GESAMP/EHS Composite List of Hazard Profiles - Hazard evaluation of substances transported by ships
- IMO IBC Code Chapter 18: List of products to which the Code does not apply
- IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances
- IMO Provisional Categorization of Liquid Substances - List 1: Pure or technically pure products
- IMO Provisional Categorization of Liquid Substances - List 2: Pollutant only mixtures containing at least 99% by weight of components already assessed by IMO
- International Agency for Research on Cancer (IARC) Carcinogens
- OECD Representative List of High Production Volume (HPV) Chemicals

toluene (CAS: 108-88-3) is found on the following regulatory lists;

- Australia - Australian Capital Territory - Environment Protection Regulation: Ambient environmental standards (Domestic water supply - organic compounds)
- Australia - Australian Capital Territory - Environment Protection Regulation: Pollutants entering waterways taken to cause environmental harm (Aquatic habitat)
- Australia - Australian Capital Territory Environment Protection Regulation Ecosystem maintenance - Organic chemicals - Non-pesticide anthropogenic organics
- Australia - Australian Capital Territory Environment Protection Regulation Pollutants entering waterways - Domestic water quality
- Australia Exposure Standards
- Australia Hazardous Substances
- Australia High Volume Industrial Chemical List (HVICL)
- Australia Illicit Drug Reagents/Essential Chemicals - Category III
- Australia Inventory of Chemical Substances (AICS)
- Australia National Pollutant Inventory
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix E (Part 2)
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix F (Part 3)
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix I
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 5
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 6
- GESAMP/EHS Composite List of Hazard Profiles - Hazard evaluation of substances transported by ships
- IMO IBC Code Chapter 17: Summary of minimum requirements
- IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk
- IMO Provisional Categorization of Liquid Substances - List 1: Pure or technically pure products
- International Agency for Research on Cancer (IARC) Carcinogens
- OECD Representative List of High Production Volume (HPV) Chemicals
- United Nations Convention Against Illicit Traffic in Narcotic Drugs and Psychotropic Substances - Table II
- United Nations List of Precursors and Chemicals Frequently used in the Illicit Manufacture of Narcotic Drugs and Psychotropic Substances Under International Control - Table II
- WHO Guidelines for Drinking-water Quality - Guideline values for chemicals that are of health significance in drinking-water

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

- Australia Exposure Standards
- Australia High Volume Industrial Chemical List (HVICL)
- Australia Inventory of Chemical Substances (AICS)
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 4
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 5
- Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 7
- Australia Therapeutic Goods Administration (TGA) Substances that may be used as active ingredients in Listed medicines
- Australia Therapeutic Goods Administration (TGA) Sunscreening agents permitted as active ingredients in listed products
- CODEX General Standard for Food Additives (GSFA) - Additives Permitted for Use in Food in General, Unless Otherwise Specified, in Accordance with GMP
- IMO IBC Code Chapter 17: Summary of minimum requirements
- International Agency for Research on Cancer (IARC) Carcinogens
- OECD Representative List of High Production Volume (HPV) Chemicals

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

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

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

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

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

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

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

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

methyl ethyl ketone (CAS: 78-93-3) is found on the following regulatory lists;

- Australia Exposure Standards
- Australia Hazardous Substances
- Australia High Volume Industrial Chemical List (HVICL)
- Australia Illicit Drug Reagents/Essential Chemicals - Category III
- Australia Inventory of Chemical Substances (AICS)

continued...

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Section 15 - REGULATORY INFORMATION

Australia National Pollutant Inventory

Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix E (Part 2)

Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix F (Part 3)

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

GESAMP/EHS Composite List of Hazard Profiles - Hazard evaluation of substances transported by ships

IMO IBC Code Chapter 17: Summary of minimum requirements

IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk

OECD Representative List of High Production Volume (HPV) Chemicals

United Nations Convention Against Illicit Traffic in Narcotic Drugs and Psychotropic Substances - Table II

United Nations List of Precursors and Chemicals Frequently used in the Illicit Manufacture of Narcotic Drugs and

Psychotropic Substances Under International Control - Table II

bisphenol A/ epichlorohydrin resin (CAS: 25068-38-6) is found on the following regulatory lists;

Australia - Victoria Occupational Health and Safety Regulations - Schedule 9: Materials at Major Hazard

Facilities (And Their Threshold Quantity) Table 2

Australia Hazardous Substances

Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix E (Part 2)

Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix F (Part 3)

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

OECD Representative List of High Production Volume (HPV) Chemicals

xylene (CAS: 1330-20-7) is found on the following regulatory lists;

Australia - Australian Capital Territory - Environment Protection Regulation: Ambient environmental standards

(Domestic water supply - organic compounds)

Australia - Australian Capital Territory Environment Protection Regulation Pollutants entering waterways -

Domestic water quality

Australia Exposure Standards

Australia Hazardous Substances

Australia High Volume Industrial Chemical List (HVICL)

Australia Inventory of Chemical Substances (AICS)

Australia National Pollutant Inventory

Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix E (Part 2)

Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix F (Part 3)

Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix I

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

Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 6

GESAMP/EHS Composite List of Hazard Profiles - Hazard evaluation of substances transported by ships

IMO IBC Code Chapter 17: Summary of minimum requirements

IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk

IMO Provisional Categorization of Liquid Substances - List 1: Pure or technically pure products

International Agency for Research on Cancer (IARC) Carcinogens

International Council of Chemical Associations (ICCA) - High Production Volume List

OECD Representative List of High Production Volume (HPV) Chemicals

WHO Guidelines for Drinking-water Quality - Guideline values for chemicals that are of health significance in

drinking-water

isobutanol (CAS: 78-83-1) is found on the following regulatory lists;

Australia Exposure Standards

Australia Hazardous Substances

Australia Inventory of Chemical Substances (AICS)

GESAMP/EHS Composite List of Hazard Profiles - Hazard evaluation of substances transported by ships

IMO IBC Code Chapter 17: Summary of minimum requirements

IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances

International Council of Chemical Associations (ICCA) - High Production Volume List

OECD Representative List of High Production Volume (HPV) Chemicals

zinc phosphate (CAS: 7779-90-0) is found on the following regulatory lists;

Australia Exposure Standards

Australia Inventory of Chemical Substances (AICS)

Australia National Pollutant Inventory

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

OECD Representative List of High Production Volume (HPV) Chemicals

zinc phosphate (CAS: 7543-51-3) is found on the following regulatory lists;

Australia Exposure Standards

Australia National Pollutant Inventory

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

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)

Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix E (Part 2)

Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix F (Part 3)

Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix I

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

GESAMP/EHS Composite List of Hazard Profiles - Hazard evaluation of substances transported by ships

IMO IBC Code Chapter 17: Summary of minimum requirements

IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances

International Agency for Research on Cancer (IARC) Carcinogens

OECD Representative List of High Production Volume (HPV) Chemicals

phenol/ formaldehyde resin (CAS: 9003-35-4) is found on the following regulatory lists;

Australia Final Report on Hazard Classification of Common Skin Sensitisers

Australia Inventory of Chemical Substances (AICS)

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

Section 16 - OTHER INFORMATION

INGREDIENTS WITH MULTIPLE CAS NUMBERS

Ingredient Name	CAS
titanium dioxide	13463- 67- 7, 1317- 70- 0, 1317- 80- 2, 12188- 41- 9, 1309- 63- 3, 100292- 32- 8, 101239- 53- 6, 116788- 85- 3, 12000- 59- 8, 12701- 76- 7, 12767- 65- 6, 12789- 63- 8, 1344- 29- 2, 185323- 71- 1, 185828- 91- 5, 188357- 76- 8, 188357- 79- 1, 195740- 11- 5, 221548- 98- 7, 224963- 00- 2, 246178- 32- 5, 252962- 41- 7, 37230- 92- 5, 37230- 94- 7, 37230- 95- 8, 37230- 96- 9, 39320- 58- 6, 39360- 64- 0, 39379- 02- 7, 416845- 43- 7, 494848- 07- 6, 494848- 23- 6, 494851- 77- 3, 494851- 98- 8, 55068- 84- 3, 55068- 85- 4, 552316- 51- 5, 62338- 64- 1, 767341- 00- 4, 97929- 50- 5, 98084- 96- 9
zinc phosphate	7779- 90- 0, 7543- 51- 3

REPRODUCTIVE HEALTH GUIDELINES

Ingredient	ORG	UF	Endpoint	CR	Adeq TLV
toluene	9.6 mg/m3	10	D	NA	-
methyl ethyl ketone	590 mg/m3	NA	NA	NA	Yes
xylene	1.5 mg/m3	10	D	NA	-
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:

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).

MSDS SECTION CHANGES

The following table displays the version number of and date on which each section was last changed.

Section Name	Version	Date	Section Name	Version	Date	Section Name	Version
Date							
First Aid	4	20- Nov- 2008	Storage (storage	4	20- Nov- 2008	Acute Health	4
20- Nov- 2008			requirement)			(swallowed)	
(inhaled)			Exposure Standard	4	20- Nov- 2008	Chronic Health	4
Fire Fighter (fire	4	20- Nov- 2008					
20- Nov- 2008			Acute Health	4	20- Nov- 2008	Environmental	4
fighting)			(inhaled)				
Fire Fighter	4	20- Nov- 2008					
20- Nov- 2008			Acute Health	4	20- Nov- 2008	Disposal	4
(fire/explosion			(skin)				
hazard)							
Spills (major)	4	20- Nov- 2008					
20- Nov- 2008							
Storage (storage	4	20- Nov- 2008					
incompatibility)							

» Classification of the preparation and its individual components has drawn on official and authoritative sources as

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Section 16 - OTHER INFORMATION

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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