DY-MARK INK T400 LEAD FREE COLOURS

Hazard Alert Code: HIGH

Chemwatch Material Safety Data Sheet | Revision No: 4 | Chemwatch 15840

Issue Date: 21-Nov-2006 | CD 2009/1

Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

PRODUCT NAME
DY-MARK INK T400 LEAD FREE COLOURS

SYNONYMS
"T-400 Marking Pen Ink", "Ball Marker Ink", "11010111 white", "11010101 black", "11010102 red", "11010103 blue", "11010104 green", "11010106 orange", "11010108 violet", "11010105 Yellow"

PROPER SHIPPING NAME
PRINTING INK

PRODUCT USE
Ink.

SUPPLIER
Company: Dy-Mark Pty Ltd
Address:
89 Formation Street
Wacol
QLD, 4076
AUS
Telephone: +61 7 3271 2222
Fax: +61 7 3271 2751

Section 2 - HAZARDS IDENTIFICATION

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

POISONS SCHEDULE
None

RISK
» Highly flammable.
» Harmful by inhalation in contact with skin and if swallowed.
» Irritating to eyes and skin.
» Cumulative effects may result following exposure*.
» May produce discomfort of the respiratory system*.
» Limited evidence of a carcinogenic effect*.
» May be harmful to the foetus/ embryo*.
» May possibly affect fertility*.
» Vapours potentially cause drowsiness and dizziness*.

* (limited evidence).

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.
» Do not empty into drains.
» To clean the floor and all objects contaminated by this material use water.
» Keep container tightly closed.
» Keep away from food drink and animal feeding stuffs.
» In case of contact with eyes rinse with plenty of water and contact Doctor or Poisons Information Centre.
» If swallowed IMMEDIATELY contact Doctor or Poisons Information Centre (show this container or label).
» This material and its container must be disposed of as hazardous waste.

Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

NAME	CAS RN	%
ethanol	64-17-5	30-70
ethylene glycol monobutyl ether	111-76-2	10-30

pigment (lead-free)	<30
resin	<15
NOTE: Manufacturer has supplied full ingredient information to allow CHEMWATCH assessment.	

Section 4 - FIRST AID MEASURES

SWALLOWED

» For advice, contact a Poisons Information Centre or a doctor.

- 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.

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.

SKIN

» If skin contact occurs:

- Immediately remove all contaminated clothing, including footwear.
- Flush skin and hair with running water (and soap if available).

INHALED

»

- If fumes or combustion products are inhaled remove from contaminated area.
- Lay patient down. Keep warm and rested.

NOTES TO PHYSICIAN

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

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

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

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

[Ellenhorn and Barceloux: Medical Toxicology] It has been suggested that there is a need for establishing a new biological exposure limit before a workshift that is clearly below 100 mmol ethoxy-acetic acids per mole creatinine in morning urine of people occupationally exposed to ethylene glycol ethers. This arises from the finding that an increase in urinary stones may be associated with such exposures.
Laitinen J., et al: Occupational & Environmental Medicine 1996; 53, 595-600.

Section 5 - FIRE FIGHTING MEASURES

EXTINGUISHING MEDIA

»

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

FIRE FIGHTING

»

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

FIRE/EXPLOSION HAZARD

»
- Liquid and vapour are highly flammable.
- Severe fire hazard when exposed to heat, flame and/or oxidisers.
- Vapour forms an explosive mixture with air.
- Severe explosion hazard, in the form of vapour, when exposed to flame or spark.
- Vapour may travel a considerable distance to source of ignition.
- Heating may cause expansion / decomposition with violent rupture of containers.
- On combustion, may emit toxic fumes of carbon monoxide (CO)

Other combustion products include: carbon dioxide (CO_2).

FIRE INCOMPATIBILITY

» Avoid contamination with strong oxidising agents as ignition may result.

HAZCHEM

None

Section 6 - ACCIDENTAL RELEASE MEASURES

EMERGENCY PROCEDURES

MINOR SPILLS

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

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.
- 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.

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

Section 7 - HANDLING AND STORAGE

PROCEDURE FOR HANDLING

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

SUITABLE CONTAINER

»
- Packing as supplied by manufacturer.

- Plastic containers may only be used if approved for flammable liquid.
- Check that containers are clearly labelled and free from leaks.

STORAGE INCOMPATIBILITY

Avoid storage with oxidising agents, acids and alkalis.

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/m³	STEL ppm	STEL mg/m³	Peak ppm	Peak mg/m³	TWA F/CC
Australia Exposure Standards	ethanol (Ethyl alcohol)	1000	1880					
Australia Exposure Standards	ethylene glycol monobutyl ether (2-Butoxyethanol)	20	96.9	50	242			

PERSONAL PROTECTION

RESPIRATOR

Type ANO Filter of sufficient capacity

EYE

» No special equipment for minor exposure i.e. when handling small quantities.

- OTHERWISE:
- Safety glasses with side shields.
- 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

»

- Neoprene gloves
- PVC gloves
- Rubber boots

OTHER

» No special equipment needed when handling small quantities.

OTHERWISE:

- Overalls.
- Barrier cream.
- Eyewash unit.

ENGINEERING CONTROLS

» General exhaust is adequate under normal operating conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	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)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)

Within each range the appropriate value depends on:

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

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the

extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

APPEARANCE
Coloured high viscosity ink with sweet solvent odour; mixes with water.

PHYSICAL PROPERTIES

Liquid.
Mixes with water.

Molecular Weight: Not applicable.	Boiling Range (°C): 80
Melting Range (°C): Not applicable.	Specific Gravity (water=1): 0.85-1.00
Solubility in water (g/L): Miscible	pH (as supplied): Not available
pH (1% solution): Not available.	Vapour Pressure (kPa): Not available
Volatile Component (%vol): Not available.	Evaporation Rate: Not available
Relative Vapour Density (air=1): Not available.	Flash Point (°C): 13
Lower Explosive Limit (%): Not available	Upper Explosive Limit (%): Not available
Autoignition Temp (°C): Not available.	Decomposition Temp (°C): Not available
State: Liquid	Viscosity: Not available

Section 10 - CHEMICAL STABILITY AND REACTIVITY INFORMATION

CONDITIONS CONTRIBUTING TO INSTABILITY
»
- Presence of incompatible materials.
- Product is considered stable.
- Hazardous polymerisation will not occur.

Section 11 - TOXICOLOGICAL INFORMATION

POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS	CHRONIC HEALTH EFFECTS
» Harmful by inhalation, in contact with skin and if swallowed.	» Limited evidence of a carcinogenic effect*.
» Irritating to eyes and skin.	» May be harmful to the foetus/embryo*.
» Can be absorbed through skin.	» May possibly affect fertility*.
» Vapours may cause dizziness or suffocation.	» Cumulative effects may result following exposure*.
» May produce discomfort of the respiratory system*.	» * (limited evidence).
» Vapours potentially cause drowsiness and dizziness*.	
» * (limited evidence).	

TOXICITY AND IRRITATION
» Not available. Refer to individual constituents.
ETHANOL:
» unless otherwise specified data extracted from RTECS - Register of Toxic Effects of Chemical Substances.

TOXICITY	IRRITATION
Oral (rat) LD50: 7060 mg/kg	Skin (rabbit):20 mg/24hr-Moderate
Oral (human) LDLo: 1400 mg/kg	Skin (rabbit):400 mg (open)-Mild
Oral (man) TDLo: 50 mg/kg	Eye (rabbit):100mg/24hr-Moderate
Oral (man) TDLo: 1.40 mg/kg	Eye (rabbit): 500 mg SEVERE
Oral (woman) TDLo: 256 mg/kg/12 wks	
Inhalation (rat) LC50: 20,000 ppm/10h	
Inhalation (rat) LC50: 64000 ppm/4h	

» The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.

ETHYLENE GLYCOL MONOBUTYL ETHER:
» unless otherwise specified data extracted from RTECS - Register of Toxic Effects of Chemical Substances.

TOXICITY	IRRITATION
Oral (rat) LD50: 470 mg/kg	Skin (rabbit): 500 mg, open; Mild
Dermal (rabbit) LD50: 220 mg/kg	Eye (rabbit): 100 mg/24h-Moderate
Inhalation (human) TCLo: 100 ppm	Eye (rabbit): 100 mg SEVERE
Inhalation (human) TCLo: 195 ppm/8h	* [Union Carbide]

Inhalation (Rat) LC50: 450 ppm *

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

The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.

For ethylene glycol:

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

dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO_2, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO_2, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

Respiratory Effects. Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning	The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases).
Cardiovascular Effects. Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12- 24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop	and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol . As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol.

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

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

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

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

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

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

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

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

Mutagenicity: In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in S. typhimurium strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. In vitro 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 foetoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species.

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

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

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

forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration -dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma.

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

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

CARCINOGEN

ethanol	International Agency for Research on Cancer (IARC) Carcinogens	Group 1
ethylene glycol monobutyl ether	International Agency for Research on Cancer (IARC) Carcinogens	Group 3

SKIN

ethylene glycol monobutyl ether	Australia Exposure Standards - Skin	Notes Sk

Section 12 - ECOLOGICAL INFORMATION

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

Section 13 - DISPOSAL CONSIDERATIONS

»
- Consult manufacturer for recycling options and recycle where possible .
- Consult State Land Waste Management Authority for disposal.
- Incinerate residue at an approved site.
- Recycle containers if possible, or dispose of in an authorised landfill.

Section 14 - TRANSPORTATION INFORMATION

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

Class or division:	3	Subsidiary risk:	None
UN No.:	1210	UN packing group:	II

Shipping Name:PRINTING INK flammable

Air Transport IATA:

ICAO/IATA Class:	3	ICAO/IATA Subrisk:	None
UN/ID Number:	1210	Packing Group:	II
Special provisions:	A3 A72		

Shipping Name: PRINTING INK FLAMMABLE

Maritime Transport IMDG:

IMDG Class:	3	IMDG Subrisk:	None
UN Number:	1210	Packing Group:	II
EMS Number:	F-E,S-D	Special provisions:	163
Limited Quantities:	5 L		

Shipping Name: PRINTING INK flammable or PRINTING INK RELATED MATERIAL (including printing ink thinning or reducing compound), flammable

Section 15 - REGULATORY INFORMATION

POISONS SCHEDULE
None

REGULATIONS
Regulations for ingredients
Dy- Mark Ink T400 Lead Free Colours (CAS: None):
No regulations applicable
ethanol (CAS: 64- 17- 5) 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)
Australia National Pollutant Inventory
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 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
International Air Transport Association (IATA) Dangerous Goods Regulations
International Council of Chemical Associations (ICCA) - High Production Volume List
OECD Representative List of High Production Volume (HPV) Chemicals
ethylene glycol monobutyl ether (CAS: 111- 76- 2) is found on the following regulatory lists;
Australia Exposure Standards
Australia Hazardous Substances
Australia High Volume Industrial Chemical List (HVICL)
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix E (Part 2)
Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix F (Part 3)
Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Appendix I
Australia Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) - Schedule 6
GESAMP/EHS Composite List of Hazard Profiles - Hazard evaluation of substances transported by ships
IMO IBC Code Chapter 17: Summary of minimum requirements
IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances
International Agency for Research on Cancer (IARC) Carcinogens
OECD Representative List of High Production Volume (HPV) Chemicals

Section 16 - OTHER INFORMATION

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

Issue Date: 21-Nov-2006

Print Date:16-Jun-2009