Initial Preparation Date:7/8/2004Last Revision Date:4/30/2012Effective Date:6/4/2012

MATERIAL SAFETY DATA SHEET

PRODUCT IDENTITY: PEAK® LONG LIFE CONCENTRATE ANTIFREEZE & COOLANT

1. CHEMICAL PRODUCT & COMPANY INFORMATION

OLD WORLD INDUSTRIES, LLC 4065 COMMERCIAL AVENUE NORTHBROOK, ILLINOIS 60062 PHONE: 847-559-2000 EMERGENCY PHONE: 1-800-424-9300 (CHEMTREC)

2. COMPOSITION/INFORMATION ON INGREDIENTS

<u>Material</u>	CAS#	<u>% by Wt</u>	PEL (OSHA)	TLV (ACGIH)
Ethylene Glycol	107-21-1	90 - 97	50 ppm	50 ppm
Diethylene Glycol	111-46-6	< 5	None	None
Organic acid salts	proprietary	< 5	10 mg/m^3	1 mg/m^3
Denatonium Benzoate	3734-33-6	30-50 ppm	None	None
Water	7732-18-5	< 4	None	None

3. HAZARDS IDENTIFICATION

EMERGENCY OVERVIEW

Slight odor.	May be fatal if swallowed.	Vapors can cause eye irritation.
Lowest Known LD50 (Oral Lowest Known LD50 (Skin	/	5840 mg/kg (Rats) 9530 mg/kg (Rabbits)
	HAZARD RATING SY	STEM
NFPA: HEALTH: 1 HMIS: HEALTH: 2	FLAMMABILITY: 1 FLAMMABILITY: 1	REACTIVITY: 0 REACTIVITY: 0
KEY: (0 – Minimal 1 – Slight 2 - I	Moderate 3 - Serious 4 - Severe

POTENTIAL HEALTH EFFECTS

Routes of Exposure: Inhalation, Ingestion, Skin Contact/Absorption, Eye Contact

Eye: May cause slight transient (temporary) eye irritation. Corneal injury is unlikely. Vapors or mists may cause eye irritation.

Skin: Prolonged or repeated exposure not likely to cause significant skin irritation. A single prolonged exposure is not likely to result in the material being absorbed through skin in harmful amounts. Repeated skin exposure may result in absorption of harmful amounts. Massive contact with damaged skin or of material sufficiently hot to burn skin may result in absorption of potential lethal amounts.

Ingestion: Single dose oral toxicity is considered to be moderate. Excessive exposure may cause central nervous system effects, cardiopulmonary effects (metabolic acidosis), and kidney failure. Small amounts swallowed incidental to normal handling operations are not likely to cause injury; however, swallowing amounts larger than that may cause serious injury, even death.

Inhalation: At room temperature, exposures to vapors are minimal due to physical properties; higher temperatures may generate vapor levels sufficient to cause adverse effects.

Systemic (Other Target Organ) Effects: Repeated excessive exposures may cause severe kidney and also liver and gastrointestinal effects. Signs and symptoms of excessive exposure may be central nervous system effects. Signs and symptoms of excessive exposure may be nausea and/or vomiting. Signs and symptoms of excessive exposure may be anesthetic or narcotic effects. Observations in animals include formation of bladder stones after repeated oral doses of ethylene glycol. Reports of kidney failure and death in burn patients suggest the ethylene glycol may have been a factor. The use of topical applications containing this material may not be appropriate in severely burned patients or individuals with impaired renal function.

Cancer Information: Based on data from long-term animal studies, ethylene glycol is not believed to pose a carcinogenic risk to man.

Teratology (Birth Defects): Exposure to ethylene glycol has caused birth defects in laboratory animals only at doses toxic to the mother.

Reproductive Effects: Ethylene glycol has not interfered with reproduction in animal studies except at very high doses.

CHRONIC, PROLONGED OR REPEATED OVEREXPOSURE

Effects of Repeated Overexposure: Repeated inhalation of ethylene glycol mist may produce signs of central nervous system involvement, particularly dizziness and nystagmus.

Other Effects of Overexposure: repeated skin contact with ethylene glycol may, in a very small proportion of cases, cause sensitization with the development of allergic contact dermatitis. The incidence is significantly less than 1% with the undiluted material.

4. FIRST AID MEASURES Ensure physician has access to this MSDS.

TREATMENT

Eyes: Immediately flush eyes with large amounts of water for 15 minutes, lifting lower and upper lids. Get medical attention as soon as possible. Contact lenses should never be worn when working with this chemical.

Skin: Flush area of skin contact immediately with large amounts of water for at least 15 minutes while removing contaminated clothing. If irritation persists after flushing, get medical attention promptly. Wash clothing before reuse.

Inhalation: If inhaled, immediately remove victim to fresh air and call *emergency medical care*. If not breathing, give artificial respiration. If breathing is difficult, give oxygen.

Ingestion: Obtain medical attention immediately. If patient is fully conscious, give two glasses of water. Do not induce vomiting. If medical advice is delayed, and if the person has swallowed a moderate volume of material (a few ounces), then give three to four ounces of hard liquor, such as whisky. For children, give proportionally less liquor, according to weight.

Notes to Physician: It is estimated that the lethal oral dose to adults is of the order of 1.0 ml/kg. Ethylene glycol is metabolized by alcohol dehydrogenate to various metabolites including glyceraldehydes, glycolic acid and oxalic acid which cause an elevated anion-gap metabolic acidosis and renal tubular injury. The signs and symptoms in ethylene glycol poisoning are those of metabolic acidosis, CNS depression, and kidney injury. Urinalysis may show albuminuria, hematuria and oxaluria. Clinical chemistry may reveal anion-gap metabolic acidosis and uremia. The currently recommended medical management of ethylene glycol poisoning includes elimination of ethylene glycol and metabolites, correction of metabolic acidosis and prevention of kidney injury. It is essential to have immediate and follow up urinalysis and clinical chemistry. There should be particular emphasis on acid-base balance and renal function tests. A continuous infusion of 5% sodium bicarbonate with frequent monitoring of electrolytes and fluid balance is used to achieve correction of metabolic acidosis and forced diuresis. As a competitive substrate for alcohol dehydrogenase, ethanol is antidotal. Given in the early stages of intoxication, it blocks the formulation of nephrotoxic metabolites. A therapeutically effective blood concentration of ethanol is in the range 100-150 mg/dl, and should be achieved by a rapid loading dose and maintained by intravenous infusion. For severe and/or deteriorating cases, hemodialysis may be required. Dialysis should be considered for patients who are symptomatic, have severe metabolic acidosis, a blood ethylene glycol concentration greater than 25 md/dl, or compromise of renal functions. A more effective intravenous antidote for physician use is 4-methylpyrazole, a potent inhibitor of alcohol dehydrogenases, which effectively blocks the formation of toxic metabolites of ethylene glycol. It has been used to decrease the metabolic consequences of ethylene glycol poisoning before metabolic acidosis coma, seizures, and renal failure have occurred. A generally recommended protocol is a loading dose of 15 mg/kg followed by 10 mg/kg every 12 hours for 4 doses and then 15 mg/kg every 12 hours until ethylene glycol concentrations are below 20 mg/100 ml. Slow intravenous infusion is required. Since 4-methyplyrozole is dialyzable, increased dosage may be necessary during hemodialysis. Additional therapeutic measures may include the administration of cofactors involved in the metabolism of ethylene glycol. Thiamine (100 mg) and pyridoxine (50 mg) should be given every six hours.

Pulmonary edema with hypoxemia has been described in a number of patients following poisoning with ethylene glycol. The mechanism of production has not been elucidated, but it appears to be non-cardiogenic in origin in several cases. Respiratory support with mechanical ventilation and positive end expiratory pressure may be required. There may be cranial nerve involvement in the late stages of toxicity from swallowed ethylene glycol. In particular, effects have been reported involving the seventh, eighth and ninth cranial nerves, presenting with bilateral facial paralysis, diminished hearing and dysphasia.

5. FIRE FIGHTING MEASURES

Flammable Properties:

Flash Point: 119°C (247°F) Method Used: Setaflash Auto-ignition Temperature: Auto-ignition temperature for ethylene glycol is 398°C (748°F).

Flammability Limits: Percentage of vapor concentration at which product can ignite in presence of spark: Lower Flammability Limit: 3.2% Upper Flammability Limit: 15.3%

Hazardous Combustion Products: Hazardous combustion products may include and are not limited to carbon monoxide, carbon dioxide and trace amounts of aldehydes and organic acids. When available oxygen is limited, as in a fire or when heated to very high temperatures by a hot wire or plate, carbon monoxide and other hazardous compounds such as aldehydes might be generated.

Extinguishing Media: Water fog or fine spray. Alcohol resistant foams (ATC type) are preferred if available. General purpose synthetic foams (including AFFF) or protein foams may function, but much less effectively. Carbon dioxide. Dry chemical. Do not use direct water stream. May spread fire.

Fire Fighting Instructions: No fire and explosion hazards expected under normal storage and handling conditions (i.e. ambient temperatures). However, ethylene glycol or solutions of ethylene glycol and water can form flammable vapors with air if heated sufficiently. Keep people away. Isolate fire area and deny unnecessary entry.

Protective Equipment for Fire Fighters: Wear positive-pressure, self-contained breathing apparatus (SCBA) and protective fire fighting clothing (includes fire-fighting helmet, coat, pants, boots and gloves).

6. ACCIDENTAL RELEASE MEASURES

Protect People: Material is moderately toxic when ingested. Take adequate precautions to keep people, especially children away from spill site. PVC-coated rubber gloves and monogoggles or face shield can be used during cleanup of spill site. Product on surfaces can cause slippery conditions. Practice reasonable care and cleanliness. Avoid breathing spray mists if generated. Keep out of reach of children. Product may become a solid at temperatures below -18°C (0°F). Do not store near food, foodstuffs, drugs or potable water supplies.

Protect the Environment: Do not dump used product or diluted material into sewers, on the ground, or into any body of water.

Cleanup: Small spills: Soak up with absorbent material. Large spills: Dike and pump into suitable containers for disposal. Ensure compliance with all applicable statues that require notification of appropriate government officials.

7. HANDLING AND STORAGE

Steps to be Taken in Case Material is Released or Spilled: Eliminate all sources of ignition in vicinity of the spilled or released fluid.

Other Precautions: Use normal precautions in handling any combustible liquid. Keep container closed when not in use. Store away from heat or open flame. Product on surfaces can cause slippery conditions. Practice reasonable

care and cleanliness. Avoid breathing spray mists if generated. Keep out of reach of children. Product may become a solid at temperatures below $-18^{\circ}C(0^{\circ}F)$. Do not store near food, foodstuffs, drugs or potable water supplies.

8. EXPOSURE CONTROLS / PERSONAL PROTECTION

Respiratory Protection: Respiratory protection is required if airborne concentration exceeds TLV. At any detectable concentration any self-contained breathing apparatus with a full face piece and operated in a pressure-demand or other positive pressure mode or any supplied-air respirator with a full face piece and operated in a pressure-demand or other positive pressure mode in combination with an auxiliary self-contained breathing apparatus operated in pressure-demand or other positive pressure mode.

Escape: Any air-purifying full face piece respirator (gas mask) with a chin-style or front- or back-mounted organic vapor canister or any appropriate escape-type self-contained breathing apparatus.

Skin Protection: Protective gloves recommended when prolonged skin contact cannot be avoided. Polyethylene; Neoprene; Nitrile; Polyvinyl alcohol; Natural Rubber, Butyl Rubber. Safety shower should be available.

Eye Protection: Safety goggles and face shield. Emergency eyewash should be available. Contact lenses should not be worn when working with this chemical.

Engineering Controls: Use general or local exhaust ventilation to meet TLV requirements.

EXPOSURE LIMITS

<u>Component</u>	Exposure Limits	<u>Skin Form</u>
Ethylene glycol	100 mg/m3 CEILING ACGIH	Aerosol
Ethylene glycol	125 mg/m3 CEILING OSHA-vacated	
	50 ppm CEILING OSHA – vacated	
	100 mg/m3 CEILING UCC	Aerosol and Vapor
Diethylene glycol	50 ppm TWA8 AIHA WEEL	Aerosol and Vapor
Diethylene glycol	10 mg/m3 TWA8 AIHA WEEL	Aerosol

In the Exposure Limits Chart above, if there is no specific qualifier (i.e., Aerosol) listed in the Form Column for a particular limit, the listed limit includes all airborne forms of the substance that can be inhaled. A "blank" in the Skin column indicates that exposure by the cutaneous (skin) route is not a potential significant contributor to overall exposure.

9. PHYSICAL / CHEMICAL PROPERTIES

Boiling Point:	176°C (349°F)
Freeze Point:	-15°C (5°F)
Specific Gravity (Water =1):	1.12
Pounds/gallon	9.3
Vapor Pressure (mm of Hg) @ 20C:	< 0.1
Vapor Density (air=1):	Not established
Water Solubility:	Complete
Evaporation Rate (BuAc = 1):	Nil
% Volatile By Volume:	90-97
Appearance:	Yellow/Green
Odor:	Mild
pH (50% Water Solution):	8.0

PEAK Long Life Antifreeze/Coolant

10. STABILITY & REACTIVITY DATA

Stability: Conditions to Avoid: Incompatibility (Materials to Avoid): Hazardous Decomposition Products: Hazardous Polymerization: Stable Keep away from flame Strong acid or oxidizing agents Incomplete combustion may produce CO gas Will not occur

11. TOXICOLOGICAL INFORMATION

Skin: The dermal LD50 has not been determined.

Ingestion: The lethal dose in humans is estimated to be 100 ml (3 oz.). The oral LD50 for rats is in the 6000-13,000-mg/kg range.

Hydrated Inorganic Acid Sodium Salt: The lowest dose of a similar compound reported to produce death in humans was estimated to be 709 mg/kg body weight for a person weighing 150 pounds, this would be equivalent to swallowing about one-tenth (.1) of a pound of the dry material in a short period of time.

Acute oral LD50s for a similar compound = 2,650 mg/kg (rats) 2,000 mg/kg (mice)

Mutagenicity (The Effects on Genetic Material): In vitro mutagenicity studies were negative. Animal mutagenicity studies were negative.

Significant Data with Possible Relevance to Humans: Ethylene glycol has been shown to produce dose-related teratogenic effects in rats and mice when given by gavage or in drinking water at high concentrations or doses. The no-effect doses for developmental toxicity for ethylene glycol given by gavage over the period of organogenesis has been shown to be 150 mg/kg/day for the mouse and 500 mg/kg/day for the rat. Also, in a preliminary study to asses the effects of exposure of pregnant rats and made to aerosis at concentrations of 150, 1000 and 25000 mg/m3 for 6 hours a day throughout the period of organogenesis, teratogenic effects were produced at the highest concentration, but only in mice. The conditions of these latter experiments did not allow a conclusion as to whether the developmental toxicity was mediated by inhalation of aerosol percutaneous absorption of ethylene glycol from contaminated skin, or swallowing ethylene glycol as a result of grooming the wetted coat. In a further study, comparing effects from high aerosol concentration by whole-body or nose-only exposure, it was shown that noseonly exposure resulted in maternal toxicity (1000 and 25000 mg/m3) and developmental toxicity with minimal evidence of teratogenicity (2500 mg/m3). The no-effects concentration (based on maternal toxicity) was 500 mg/m3. In a further study in mice, no teratogenic effects could be produced when ethylene glycol was applied to skin of pregnant mice over the period of organogenesis. The above observations suggest that ethylene glycol is to be regarded as an animal teratogen. There is currently no available information to suggest that ethylene glycol has caused birth defects in humans. Cutaneous application of ethylene glycol is ineffective in producing developmental toxicity. Exposure to high aerosol concentrations is only minimally effective in producing developmental toxicity. The major route for producing developmental toxicity is perorally. Two chronic feeding studies, using rats and mice, have not produced any evidence that ethylene glycol causes dose-related increases in tumor incidence or a different pattern of tumors compared with untreated controls. The absence of carcinogenic potential for ethylene glycol has been supported by numerous in vitro genotoxicity studies showing that it does not produce mutagenic or clastogenic effects.

A chronic dietary feeding study of diethylene glycol with rats showed mild kidney injury at 1%, while concentrations of 2% and 4% caused more marked kidney injury. In addition, at 2% and 4% of diethylene glycol in the diet, some rats developed benign papillary tumors in the urinary bladder. These have been attributed to the presence of urinary bladder calcium oxalate stones. No evidence for carcinogenicity was found with a chronic skin-painting study with diethylene glycol in mice. The absence of a direct chemical carcinogenic effect addords with the results in vitro PEAK Long Life Antifreeze/Coolant 6

genotoxicity studies that show that it does not produce mutagenic or clastogenic effects. A feeding study employing up to 5.0% diethylene glycol in the diet failed to produce any teratogenic effects. In a mouse continuous breeding study with large doses of diethylene glycol in drinking water, there was evidence for reproductive toxicity at 3.5% (equivalent to 6.1 g/kg/day) as reduced number of litter, live pups per litter and live pup weight. No such effects were seen at 1.75% (approximately 3.05 g/kg/day). The relevance of these very high dosages to human health is uncertain. Pregnant rats receiving undiluted diethylene glycol by gavage over the period of organogenesis had toxic effects at 4.0 and 8.0 ml/kg/day as mortality, decreased body weight, decreased food consumption increased water consumption and increased liver and kidney weights. Fetotoxicity was seen only at these maternally toxic dosages. Decreased fetal body weight occurred at 8.0 ml/kg/day, and increased skeletal variants at 4.0 and 8.0 ml/kg/day. No embryotixic or teratogenic effects were seen. Neither maternal toxicity nor fetotoxicity occurred at 1.0 ml/kg/day. In a study with mice also receiving undiluted diethylene glycol over the period of organogenesis, maternal toxicity occurred at 2.5 and 10.0 ml/kg/day, but not at 0.5 ml/kg/day. Definitive developmental toxicity was not seen in this species.

ACUTE TOXICITY

Peroral:	The lethal dose in humans is estimated to be 3 oz. or 100 ml. Rat: LD50 (6000 – 13000) mg/kg
Percutaneous:	Rabbit: $LD50 = >22270 \text{ mg/kg}$; 24 h occluded
Inhalation:	Rat: 8-hour exposure, substantially saturated vapor studies, dynamic generation method Mortality: 0/6
Inhalation:	Mist/vapor study, rat, at 170°C, 8-hour exposure = 2.2 mg/l Mortality: 0/6
Inhalation:	Rat: 8-hour exposure, fog = 10000 ppm; $65^{\circ} - 70^{\circ}$ C Mortality: 0/6
	IRRITATION
Skin:	Rabbit: 24-hour occluded contact, 0.5 ml Results: Minor erythema and edema
Skin:	Human: Primary irritation patch test, 48-hour occluded, 0.2 ml Results: Evidence of irritation
Eye:	Rabbit: 0.1 ml Results: Minor transient iritis, conjunctival irritation with discharge

REPEATED EXPOSURE

In a 7-day dietary study with rats, a significant increase in kidney weights in females was observed at 5.0 gm/kg. The NOEL was 2.5 gm/kg.

In a 24-month dietary study with rats, increased mortality in males was observed at the highest dose, 1.0 gm/kg/day. There were multiple signs: mineralization of several organs, including the cardiac vessels, cardiac muscle, vas deferens, stomach and pulmonary vessels; cellular hyperplasia of the parathyroids, hemosiderosis of the spleen, myocardial fibrosis, portal fibrosis of the liver, bile duct hyperplasia and hydronephrosis and oxylate nephrosis of the kidneys. Ethylene glycol was not oncogenic.

In a 90-day dietary study with dogs, repeated exposures to 2.5 gm/kg resulted in acute renal failure and deaths. The NOAEL was 1.0 gm/kg.

SENSITIZATION (ANIMAL AND HUMAN STUDIES)

Repeated skin contact with ethylene glycol may, in a very small proportion of cases, cause sensitization with the development of allergic contact dermatitis. The incidence is significantly less than 1% with the undiluted material.

REPRODUCTIVE TOXICITY

A three-generation study indicated that ethylene glycol did not affect reproductive parameters at dietary concentrations up to 1.0 gm/kg/day in any generation.

CHRONIC TOXICITY AND CARCINOGENICITY

Two chronic feeding studies, using rats and mice, have not produced any evidence that ethylene glycol causes doserelated increases in tumor incidence or a different pattern of tumors compared with untreated controls. The absence of a carcinogenic potential for ethylene glycol has been supported by numerous in vitro genotoxicity studies showing that it does not produce mutagenic or clastogenic effects.

GENETIC TOXICOLOGY

In Vitro: Ethylene glycol was devoid of genotoxic activity in an Ames test, forward gene mutation and sister chromatid exchange (SCE) studies in Chinese Hamster Ovary (CHO) cells and an in vitro cytogenetics study.

In Vivo: Ethylene glycol by three different routes (intravenous, peroral and percutaneous) demonstrates apparent first-order pharmacokinetic behavior for the disposition in and the elimination from the plasma. Dose-dependent changes occur for the elimination of metabolites in the urine and as 14CO² after single doses for the intravenous and peroral, but not the percutaneous route. The hypothesis from literature sources exists that developmental toxicity is caused by a metabolite of ethylene glycol, called glycolic acid, and not parent ethylene glycol. Under most conditions of ethylene glycol exposure, the glycolic acid metabolite is present in the blood in very low levels. However, it can become the major metabolite following large doses of ethylene glycol due to saturation of glycolic acid oxidation and/or elimination. When levels of this acidic metabolite exceed the capacity of maternal blood buffers to neutralize it, a maternal metabolic acidosis ensues, which has been hypothesized to be the true agent responsible for ethylene glycol induced developmental toxicity. Research suggests that ethylene glycol developmental toxicity is due to a dose-rate dependent toxicokinetic shift leading to glycolate accumulation and metabolic acidosis.

ADDITIONAL STUDIES

Ethylene glycol has been shown to produce dose-related teratogenic effects in rats and mice when given by gavage or in drinking water at high concentrations or doses. The no-effect doses for developmental toxicity for ethylene glycol given by gavage over the period of organogenesis has been shown to be 150 mg/kg/day for the mouse and 500 mg/kg/day for the rat. Also, in a preliminary study to assess the effects of exposure of pregnant rats and mice to aerosols at concentrations of 150, 1000 and 2500 mg/m³ for 6 hours a day throughout the period of organogenesis, teratogenic effects were produced at the highest concentration, but only in mice. The conditions of these latter experiments did not allow a conclusion as to whether the developmental toxicity was mediated by inhalation of aerosol, percutaneous absorption of ethylene glycol from contaminated skin, or swallowing of ethylene glycol as a result of grooming the wetted coat. In a further study, comparing effects from high aerosol concentration by wholebody or nose-only exposure, it was shown that nose-only exposure resulted in maternal toxicity (1000 and 2500 mg/m^3) and developmental toxicity with minimal evidence of teratogenicity (2500 mg/m³). The no-effects concentration (based on maternal toxicity) was 500 mg/m³. In a further study in mice, no teratogenic effects could be produced when ethylene glycol was applied to the skin of pregnant mice over the period of organogenesis. The PEAK Long Life Antifreeze/Coolant 8

above observations suggest that ethylene glycol is to be regarded as an animal teratogen. There is currently no available information to suggest that ethylene glycol has caused birth defects in humans. Cutaneous application of ethylene glycol is ineffective in producing developmental toxicity. Exposure to high aerosol concentrations is only minimally effective in producing developmental toxicity.

12. ECOLOGICAL INFORMATION

ENVIRONMENTAL FATE

Movement & Partitioning: Bioconcentration potential is low (BCF less than 100 or Log Kow less than 3). Log octanol/water partition coefficient (log Kow) is -1.36. Henry's Law Constant (H) is 6.0E-08 atm-m3/mol. Bioconcentration factor (BCF) is 10 in golden orfe.

Degradation & Transformation: Biodegradation under aerobic static laboratory conditions is high (BOD20 or BOD28/ThOD greater than 40%). 5-Day biochemical oxygen demand (BOD5) is 0.78 p/p. 10-Day biochemical oxygen demand (BOD10) is 1.06 p/p. 20-Day biochemical oxygen demand (BOD20) is 1.15 p/p. Theoretical oxygen demand (THOD) is calculated to be 1.29 p/p. Biodegradation may occur under both aerobic and anaerobic conditions (in either the presence or absence of oxygen). Inhibitory concentration (IC50) in OECD "Activated Sludge, Respiration Inhibition Test" (Guideline # 209) is < 1000 mg/L. Degradation is expected in the atmospheric environment within days to weeks.

Ecotoxicology: Material is practically non-toxic to aquatic organisms on an acute basis (LC50 greater than 100 mg/L in most sensitive species). Acute LC50 for fathead minnow (Pimephales promelas) is 51000 mg/L. Acute LC50 for bluegill (Lepomis macrochirus) is 27549 mg/L. Acute LC50 for rainbow trout (Oncorhynchus mykiss) is about 18000-46000 mg/L. Acute LC50 for guppy (Poecilia reticulata) is 49300 mg/L. Acute LC50 for water flea (Daphnia magna) is 46300-51100 mg/L. Acute LC50 for the cladoceran Ceriodaphnia dubia is 10000-25800 mg/L. Acute LC50 for crayfish is 91430 mg/L. Acute LC50 for brine shrimp (Artemia salina) is 20000 mg/L. Acute LC50 for golden orfe (Leuciscus idus) is greater than 10000 mg/L. Acute LC50 for goldfish (Carassius auratus) is greater than 5000 mg/L. Growth inhibition EC50 for green alga Selenastrum capricornutum is 9500-13000 mg/L.

BOD (% Oxygen Consumption):

Day	y 5	Day 10	Day 15	Day 20	Day 30
51	%	80%		97%	

ECOTOXICITY

Toxicity to Micro-organisms:

Bacterial / NA: 16 h; IC50 Result Value: >10000 mg/l

Toxicity to Aquatic Invertebrates:

Daphnia: 48 h; LC50 Result Value: >100000 mg/l

Toxicity to Fish

Fathead Minnow: 94 h; LC50 Result Value: 70000 mg/l

FURTHER INFORMATION

Chemical Oxygen Demand (COD) – Measured: 1.29 mg/mg Theoretical Oxygen Demand (THOD) – Calculated: 1.30 mg/mg Octanol/Water Partition Coefficient – Measured: -1.36

13. DISPOSAL CONSIDERATIONS

DO NOT discharge to sewer. Wear appropriate personal protection. Take up with sand, vermiculite, or similar inert material. Dispose in accordance with federal, state and local regulations.

14. TRANSPORT INFORMATION

U.S. DEPARTMENT OF TRANSPORTATION

Non-Bulk: Not regulated by the US D.O.T. (in quantities under 5,000 lbs in any one inner package)

Bul	k:	
Dee		CL

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Proper Shipping Name:	Environmentally Hazardous Substance, LIQUID N.O.S. (ETHYLENE GLYCOL)
Technical Name:	ETHYLENE GLYCOL
ID Number:	UN 3082
Hazard Class:	9
Packing Group:	PG III
Reportable Quantity:	5,000 lb.

IATA

Non-Bulk: Not Regulated by IATA

IMDG

Non-Bulk: Not regulated by IMDG (in quantities under 5,000 lbs in any one inner package)

15. REGULATORY INFORMATION

THIS PRODUCT CONTAINS COMPONENT(S) CITED ON THE FOLLOWING REGULATIONS.

	<u>Chemical Name</u> Ethylene Glycol	<u>Cas Number</u> 107-21-1
United States - TSCA		
Inventory:	Listed	
Water Standards:	No data available	
Atmospheric Standards:	Clean Air Act (1990) - List of H	Hazardous Air Contaminants: listed
CERCLA:	Reportable Quantity (RQ): 5,00	0 pounds (532 gallons)

OSHA Hazard Communication Standard: This product is a "hazardous chemical" as defined by the OSHA Hazard Communication Standard, 29 CFR 1910.1200.

SARA Title III:	Section 311/312 - Categories: Acute hazard; chronic hazard	
	<u>Section 312 - Inventory Reporting</u> : Ethylene glycol is subject to Tier I and/or Tier II annual inventory reporting.	
	<u>Section 313</u> - <u>Emission Reporting</u> : Ethylene glycol is subject to Form R reporting requirements.	
	Section 302 - Extremely Hazardous Substances: Ethylene glycol is not listed.	

State Right-To-Know:

California - Exposure Limits - Ceilings:	vapor-50 ppm ceiling; 125 mg/m3 ceiling
Director's List of Hazardous Substances:	listed
Florida - Hazardous Substances List:	listed
Massachusetts - Right-to-Know List:	listed
Minnesota - Haz. Subs. List:	listed (particulate and vapor)
New Jersey - Right-to-Know List (Total):	Present greater than 1.0%
Pennsylvania Right-to-Know List:	environmental hazard

Canadian Regulations: This product has been classified in accordance with the hazard criteria of the Controlled Products Regulations (CPR) and the MSDS contains all the information required.

WHMIS Information: D2A - material has potential toxic effects. Refer elsewhere in the MSDS for specific warnings and safe handling information. Refer to the employer's workplace education program.

California Proposition 65 (Safe Drinking Water and Toxic Enforcement Act of 1986): The normal consumer use of this product does not result in exposure to chemicals known to the state of California to cause Cancer and/or reproductive harm above the significant risk level for carcinogens or the maximum allowable dose levels for reproductive toxins. Warnings are not required for consumer packaging. However, industrial or other occupational use of this product at higher frequency and using larger quantities of this product may result in exposures exceeding these levels and are labeled accordingly.

California SCAQMD Rule 443.1 (South Coast Air Quality Management District Rule 443.1, Labeling of Materials Containing Organic Solvents): VOC: Vapor pressure 0.06 mmHg at 20°C 1113.38 g/l

16. OTHER INFORMATION

Contact: Thomas Cholke

Phone: (847) 559-2225

Old World Industries, LLC makes no warranty, representation or guarantee as to the accuracy, sufficiency or completeness of the material set forth herein. It is the user's responsibility to determine the safety, toxicity and suitability of his own use, handling and disposal of this product. Since actual use by others is beyond our control, no warranty, expressed or implied, is made by Old World Industries, LLC as to the effects of such use, the results to be obtained or the safety and toxicity of this product, nor does Old World Industries, LLC assume liability arising out of the use by others of this product referred to herein. The data in this MSDS relates only to the specific material designated herein and does not relate to use in combination with any other material or in any process.

PEAK Long Life Antifreeze/Coolant